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A

TEXT-BOOK

OF

PRACTICAL MEDICINE

DESIGNED FOR THE USE OF

STUDENTS AND PRACTITIONERS OF MEDICINE

BY

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EIGHTH EDITION

REVISED AND ENLARGED

WITH TWO HUNDRED AND FIFTEEN ILLUSTRATIONS

NEW YORK
WILLIAM WOOD AND COMPANY
1893

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L86
1892

PREFACE TO THE EIGHTH EDITION.

In the progress of medical sciences during the four years since the third edition of this treatise was published, many unsettled questions have received definite answers, and numerous theories have crystallized into definite forms of fact or fancy. The author has always sought to maintain a conservative yet liberal attitude in his consideration of any statements emanating from recognized authorities. The contributions of intelligent observers, both in this country and Europe, have been carefully studied, and it is believed that the present edition, much of which has been rewritten, and all of which has undergone careful revision, will be found abreast with the present position of conservative scientific medicine ; that its dogmatic affirmations will be found reliable, and its statements of still debatable questions will prove unbiassed and just to both sides, although intended to be conservative rather than radical.

Such changes as are at variance with former editions have been made as the result of more exact knowledge of the etiology and pathology of disease, and from a more extended experience in its treatment. The important additions include brief descriptions of the more frequent pathological processes, and a detailed statement of the methods employed in bacteriological study, with an enumeration of the distinguishing characteristics of those micro-organisms which at the present time are regarded as pathogenic. Several additions also have been made to the list of diseases considered. In the revision, Phthisis has been classed as an infectious disease, although retained among pulmonary diseases for description, and its pathology and etiology rewritten in conformity with its bacillary nature, adopting the name *pulmonary tuberculosis.*

The pathological term *nephritis* has been substituted for the indefinite one *Bright's disease,* with a view of making a more exact classification of renal diseases.

The methods advised to be pursued in bacteriological studies are those now employed in the "Loomis' Laboratory," the details of which have been furnished by J. D. Byron, M.D., Instructor in the Bacteriological Department of the Laboratory.

The changes and additions which have been made in the plates have been furnished by H. P. Loomis, M.D., Director of the Pathological Department of the Loomis Laboratory. C. E. Quimby, M.D., Assistant Professor of Practice in the Medical Department of the University of the City of New York, has rendered me valuable assistance in the preparation of this edition.

A. L. L.

19 West 34th Street,
New York, *August*, 1889.

32038

PREFACE TO FIRST EDITION.

In the preparation of a Text-book of Practical Medicine, my experience as a medical teacher has led me to employ, quite extensively, plates illustrating the morbid changes and objective symptoms of disease.

The present work, both in text and illustration, is practically a revision and an elaboration of lectures given during the past eighteen years in the Medical Department of the University of the City of New York.

I have avoided, as far as possible, the discussion of unsettled questions, and in order to economize space have made reference to many of these only in brief foot-notes.

The Classification adopted is that which it has been my custom to follow in teaching, and is based on our present knowledge of the etiology of disease.

It is well known that many diseases present very different types in different countries, and I have selected for description those types commonly observed by the American physician.

I have considered only those diseases which come strictly within the province of Practical Medicine, and have endeavored to indicate the treatment usually followed in this country.

The illustrations, with but few exceptions, have been made by my assistant, Dr. Maurice N. Miller (Instructor in the Laboratory of Normal and Pathological Histology, University Medical College). The microscopical drawings were, in most instances, made from sections prepared in the Laboratory by Dr. Miller, especially for this work, and they will, I believe, aid in the appreciation of the actual morbid processes and conditions.

In the consultation of Authorities, particularly the German and French, in the reading of the proof, and in the preparation of the Index, I have been assisted by Dr. Leigh Hunt, Assistant Instructor in the Pathological Laboratory of the University.

If I have failed to give credit—either in the text or in foot-notes—to those from whom many of the facts stated have been drawn, it has been an unintentional omission.

19 West 34th Street, New York City,
July, 1884.

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A TEXT-BOOK

OF

PRACTICAL MEDICINE.

INFLAMMATION.

According to the older writers the cardinal symptoms of inflammation are pain, heat, redness and swelling, features which are more striking in those forms of inflammation which come under the care of the surgeon than in those which the physician is called upon to treat. As the knowledge of tissues and processes became more detailed and complete, and as the hidden changes underlying these grosser ones were brought to light, pathologists sought to discover the essence of the inflammatory process, to find its cause, and to determine in which tissues or organs the primary change occurred. The history of the theories and definitions of inflammation is a record of the varying importance that has been attached to one or another of the changes observed. Into these theories, and the arguments by which they have been in turn supported and assailed, it is not desirable here to enter. It will be sufficient to describe the changes observed in the tissues, and to define the associated terms of which use will hereafter be made. The pathological results of inflammation are the product of three factors, combined in varying proportions, the vascular, the exudative and the parenchymatous changes. According as one or another is prominent it determines the character of the inflammation.

Vascular Changes.—Except in the non-vascular tissues, as the cornea and cartilage, the earliest change observed is in the circulation, and this change is manifested by a change in the color of the affected part, which becomes red and congested. This redness is due to an increase of the quantity blood in the part ; at first the hyperæmia is active, that is, blood is brough to the part and passed through the capillaries in larger quantities tha before ; but it may become passive, a condition in which, while the quan tity of blood present in the part is greater than usual, the current is muc slower, the amount which actually passes through the capillaries in a give time being less than normal ; finally, this retardation of the flow may en in actual arrest : *stasis*. That hyperæmia is only an accompaniment, an

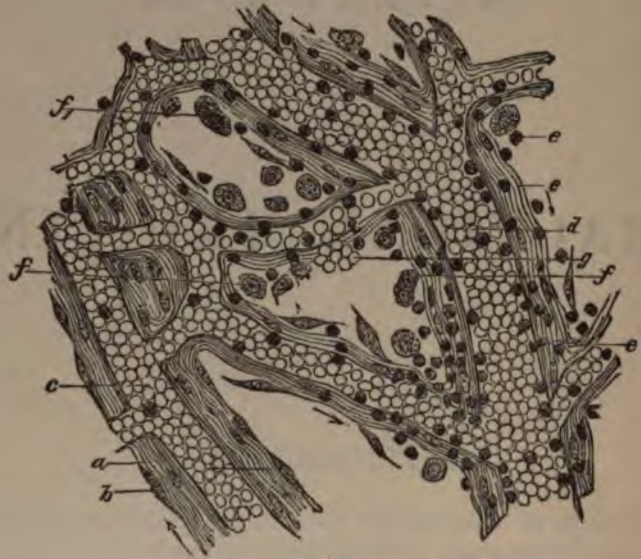

FIG. 1.

Inflamed Omentum from Human Subject.

a, *Normal fibrous trabecula ; b, normal endothelium ; c, small artery ; d, vein with white blood-corpuscl peripherally disposed ; e, white blood-cells migrated or migrating ; f, desquamated endothelium ;* f_1*, mult nuclear cell ; g, migrated red blood.—Zeigler.*

not the essence, of inflammation, is shown by the fact that the hyperæmi which is caused by section of the sympathetic nerves is not accompanie by the other symptoms and changes observed in inflammation.

Exudative Changes.—The swelling which has been mentioned as one the four cardinal symptoms is due mainly to the presence of a liqui infiltrated through the tissues. This liquid comes from the blood b exudation through the walls of the capillaries ; but it is not simply th plasma or serum of the blood ; it contains large numbers of emigrate white blood-cells, which, with the fixed cells of the part, furnish thos peculiar constituents of the exudation which distinguish this liquid fro the normal juices of the part, or from that of œdema, and which mak it an inflammatory exudation. The character of the exudation, whethe it be serum, fibrin, or pus, is determined also by the cellular elements and more especially by the emigrated white cells.

Examination of fresh normal tissues shows, scattered through them, fre cells which closely resemble the colorless corpuscles of the blood and lymph. Like them, they possess the power of amœboid movement, of rapidly changing their shape by throwing out processes, of moving from place to place by means of this change of form, and of multiplying by division. They are called "wandering cells," or leucocytes, because of their supposed identity with the colorless corpuscles of the blood. Under normal conditions the leucocytes contained within the blood-vessels may occasionally be seen to pass through the unbroken wall of a capillary by means of this power of amœboid movement which they possess; when the tissue adjoining the capillary is inflamed, the number of cells "migrating" through its wall is notably increased. The emigrated white cells form the larger portion of the cellular part of inflammatory exudations, and play a most prominent part in both necrotic and reparative processes. The number of free cells is increased by the fixed corpuscles of the omnipresent connective-tissue, which swell, as has been described, and give rise by proliferation to other cells, which cannot be distinguished morphologically from the normal wandering cells or leucocytes.

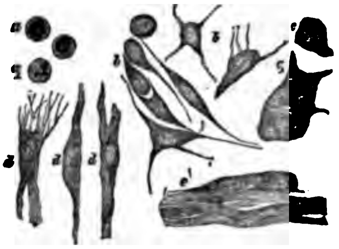

FIG. 2.

Granulation-cells in Various Stages.

a, uninuclear; a_1, multinuclear migrated white blood-corpuscles; b, various forms of uninuclear formative cells; c, binuclear; c_1, multinuclear formative cells; d, formative cells developing into connective-tissue; e, developed connective-tissue. × 250.—*Zeigler.*

Parenchymatous Changes. — The cellular elements of the tissues undergo change in form and nutrition. The chemical interchanges which constitute normal nutrition, and which are carried on between the cells of the tissues and the liquid furnished to the cells by the blood, are modified in character or extent, and the cells themselves are correspondingly modified in form. On the one hand the cells may show a tendency to return to their earlier embryonal form, to become swollen, globular, pale and succulent, perhaps to divide, to form new cells, by proliferation; on the other hand, the exaggerated activity of the cell may prove too great a strain upon it, and it dies or becomes disabled by passage into the condition known as fatty degeneration. The former of these two results is the one seen most commonly in the connective-tissue framework and envelopes of the various parts and organs, and the latter in the specific cells that constitute their parenchyma. These modifications of nutrition vary with the nature of the irritant, and the extent of the tissue compression from the exudation.

Terminations of Inflammation.—The inflammatory process, as thus described, may be arrested at any point. If the irritation is slight or of short duration, if the change has not progressed to the point of tissue destruction and formation of pus, the withdrawal of the primary cause is followed by a diminution of the swelling and congestion, and by return to the normal state;—this is called ***resolution.*** When, however, the vitality of the tissues is impaired, by prolonged irritation, by compression or by the action of specific poisons, parenchymatous degeneration or necrosis of the tissues may occur. This necrosis may be preceded or followed by a localized or diffuse infiltration of the tissues by leucocytes. When these cells accumulate in great numbers (whether by transformation of connective-tissue cells or by migration from the vessels), those which are in the necrotic area lose their vitality and constitute the cellular portion of pus, while those which fill the immediately adjacent parts pass on to the formation of connective-tissue, producing the limiting wall of fibrin about the necrotic area. When this necrosis is gradual and attended by molecular disintegration of the tissues, it is called ulceration if upon a free surface, and abscess when the pus and necrotic tissue are retained in the substance of an organ. When necrosis affects palpable masses of tissue it is called sloughing. When necrotic changes have taken place, the simple arrest of the inflammation is not sufficient; the losses must be made good, the destruction repaired. This is *cicatrization*, and it is accomplished largely by cells of the connective-tissue. As the conditions become more favorable, the cells newly formed by proliferation no longer remain stationary, lose their vitality, and become pus, but they progress in the direction of a normal development and form new tissue. The irregular pink granulations seen within a wound or upon an ulcerated surface are formed of masses of young cells crowded with capillary loops of new formation. The cells, at first large, soft, finely granular and juicy, become smaller and firmer, and the intercellular substance increases and becomes fibrous. If the inflammatory process has taken place in the interior of an organ, involving only a small portion of tissue, and has stopped short of the formation of a distinct collection of pus, the result of the reparative process is a mass of fibrous connective-tissue, a *cicatrix;* and if a collection of pus has actually formed, but is only of small size, the pus may disappear by liquefaction of its cellular elements and absorption. If, on the other hand, a larger abscess has formed and has been opened, its cavity becoming filled by the granulations, the same change into fibrous tissue follows, and a cicatrix is again the result. The same is true of ulceration of a free surface, with the addition that the surface of the cicatrix is covered by a layer of epithelium resembling more or less closely the original layer which has been destroyed by the ulceration. When the irritation has been less active but more prolonged, and has perhaps involved an entire organ, although the cellular development is abundant, the elements retain their vitality, and neither suppurative nor necrotic changes occur, but the effect again appears in an increase of the connective-tissue of the part involved; the consequences of this increase are most serious. The original "fixed cells" of the connective-tissue multiply as in the other case mentioned, and develop into fully formed tissue, and the amount of this tissue becomes in consequence much greater than normal. In its natural evolution it retracts, and by its quantity and its retraction it presses upon, and interferes with the nutrition of the specific cellular elements of the organ, so that they become less fit to perform their functions. This change is called *induration* or *cirrhosis;* common examples are cirrhosis of the liver, and "contracted kidney" or interstitial nephritis.

INFLAMMATION OF FREE SURFACES.

First : Serous Surfaces.—The most common form of inflammation of serous membranes is that which results in the production of serum, fibrin, or pus, in variable proportions ; these products may infiltrate the substance of the inflamed membrane, be poured out upon its free or attached surface, or collect in cavities lined by it. The first change in this inflammatory process is in the blood-vessels, which contain more than their normal quantity of blood, and it is from the blood circulating in the vessels that most of the characteristic inflammatory products are derived. After the initial hyperæmia, the fibrinogen of the exudation comes in contact with the fibrino-plastic material of the cellular elements (there being a ferment present), coagulation takes place, and layers of fibrin containing few or many cells are formed on the free surface. These layers are called pseudo-membranes, or coagulable lymph. If the inflammation occurs in a membrane whose normal conformation makes a free effusion possible (as the pleura and peritoneum) some serum is always present. It may be only infiltrated through the meshes of the tissues, or it may accumulate at some point as a serofibrinous collection. When leucocytes are present in great numbers, the exudation is fibrino-purulent. The greater the intensity of the inflammation, and the more enfeebled the patient, the greater is the liability to pus formation. In certain serous inflammations the exudation may be hemorrhagic ; the blood may come from a ruptured capillary vessel, or the coloring matter of the blood-corpuscle may be set free and color the exudation without vascular lesion. Such inflammation of serous membranes may end in necrosis or in resolution. If the inflammation is intense, and stasis occurs throughout a wide area of tissue, it will result in necrosis. Stasis is the expression of a higher degree of injury than that which exists in simple inflammation.[1] The intensity of the inflammation deter-

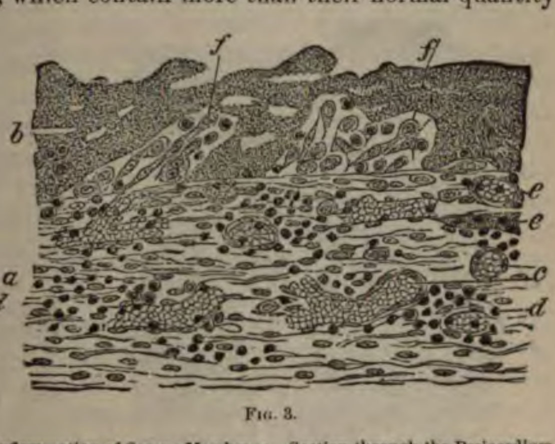

FIG. 3.

Inflammation of Serous Membrane. Section through the Pericardium and Overlying False Membrane. × 150.

a, Visceral pericardium ; b, fibrinous false membrane ; c, distended blood-vessel ; d, leucocytes infiltrating tissues ; e, lymphatics filled with cells ; f, formative cells within the false membrane.—After Zeigler.

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[1] If inflammation is an arrest of function, and *not* diversion of agents of nutrition into new channels of activity, restoration of a part to the natural state must be as simple as its departure from it, and resolution of inflammation means, either that the temporarily arrested process goes on again, or, if the process has proceeded to its ultimate issue (death of the affected part), that the destroyed part has to be repaired, not by a continuation of the morbid process, but simply by the restitution of the normal condition.

mines whether the result shall be a return to the normal condition or a destruction of tissue. An uncomplicated serous inflammation is neither reproductive nor infective. It has no tendency except to stop as soon as its primary cause ceases to act. When resolution occurs, the emigration of leucocytes ceases, the serous fluid disappears, and the fibrin and the cell elements, after they have undergone molecular change, are absorbed.

A second variety of inflammation of serous membranes is characterized by the production of new connective-tissue cells either with or without a sero-purulent exudation. It may be an acute or chronic process. The inflamed membrane becomes thickened, and there is abundant cell development in its substance and on its surface. If the inflammatory process is prolonged, or if the membrane becomes very much thickened, elevations are formed on the surface of the membrane, and thus adhesion takes place between opposing serous surfaces, or the membrane becomes thickened and indurated. If bands of adhesion form, they have the appearance of delicate membranes. This new tissue at first is exceedingly rich in capillary vessels, which are distinguished from the normal capillaries of the membrane by their large calibre and thin walls. As the new tissue contracts, it may shut off its own blood supply, and then undergo fatty change and be absorbed, leaving no trace of its existence.

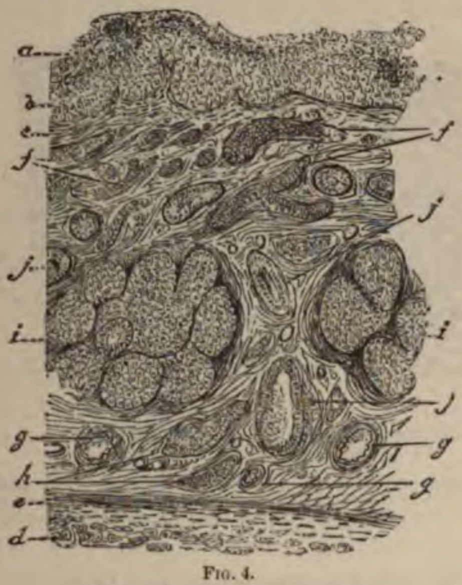

FIG. 4.

Inflammation of Mucous Membrane.

Vertical section of nasal septum.

a. Pus corpuscles and degenerating epithelium on the free surface.
b. Superficial layers of epithelium.
c. Sub-epithelial tissue.
In the submucous tissue beneath the last will be seen—
f f, g g. Longitudinal and transversely divided arteries, increased in number and size.
h h. Veins.
i i. Portions of enlarged mucous glands.
j j. Gland ducts.
A portion of the cartilage of nasal septum is seen at (d), with its perichrondrium (e). × 200. *After Thierfelder.*

Second: Mucous Surfaces.—Inflammation affecting mucous membranes may be either *catarrhal, croupous,* or *diphtheritic.* Catarrhal mucous inflammations are either acute, sub-acute, or chronic. In the acute variety the affected mucous membrane, at the very beginning of the process, is congested and dryer than normal, the functional activity of the mucous glands being diminished. After a time an abnormal quantity of mucus is poured out on its surface, the result of an increase in the functional activity of the glands. This mucus may be thicker or thinner than normal, and may have an acrid or irritating quality. Mucous exudations do not coagulate, but adhere somewhat closely to the surface of the inflamed membrane; these changes are accompanied by desquamation of the superficial epithelial cells. If the catarrh assumes a purulent character in addition to the above changes, the mucous surface assumes a darker and livid hue, and pus cells are developed both in the mucous membrane and in the deeper substance. The amount of pus will indicate the intensity and character of the inflammation. In some cases there are very few pus cells, in others the quantity of pus is very large, and the tissues are extensively infiltrated. In chronic catarrh the blood-vessels of the inflamed membrane are either increased in size and number, or they are less numerous and more swollen than normal, giving to the membrane a grayish appearance. The production of mucus will be increased or diminished, according as the functional activity of the mucous glands is increased or diminished. When it is diminished, the membrane assumes a dry and shining appearance. The stroma of the affected membrane may be hypertrophied or atrophied. The mucous glands may also undergo hypertrophy or atrophy. If their ducts become obstructed they may suffer cystic change; superficial erosions sometimes occur from a rapid epithelial desquamation.

Croupous Inflammation of Mucous Membranes.—In croupous inflammation, the hyperæmia is more intense than in catarrhal, so that the mucous surface usually assumes a dark livid color and becomes swollen; soon its free surface is covered with a fibrinous exudation, which takes the place of the epithelium, and lies upon the sub-epithelial structures in the form of a network or in irregular masses. Enclosed in its meshes are epithelial and pus cells; it varies in thickness from an exceedingly thin semi-transparent membrane to one that may be an eighth of an inch in thickness. This membranous exudation may be limited to small patches, or extend over a large surface. At first it is firm in consistency, and adheres closely to the tissues which it covers; afterward it becomes soft, and is easily separated from the subjacent membrane; when fully formed it may be cast off in patches or shreds. Its separation is accomplished by the returning secretion of the follicles which have been obstructed, as well as by the serous effusion from the inflamed surface. It may sometimes undergo fatty, and more rarely a mucous degeneration, and so become a fluid resembling mucus. Generally in simple croupous inflammation the submucous tissue is but slightly involved, and its meshes are rarely infiltrated.

Diphtheritic Inflammation of Mucous Surfaces.—By some this is regarded as identical in character with croupous. It differs from it in a more intense hyperæmia, and a more extensive infiltration of the affected tissue. The fibrinous exudation is more abundant and granular, and there is a greater metamorphosis of the epithelial and tissue cells. The membranous exudation seems to be a part of the mucous and sub-mucous tissues, and cannot be removed without the loss of their substance. In the surface exudation, and in the infiltrated tissues underlying it, are found multitudes of bacteria, especially the micrococci. When the mucous and sub-mucous tissues are so infiltrated as to cause undue pressure, and to cut off their nutritive supply, the affected tissue dies and sloughs away. Between simple croupous and diphtheritic exudation there is every possible gradation. Some claim that the fibrinous degeneration of the epithelial cells is the source of the diphtheritic exudation; nearly all agree that the primary changes are epithelial. This form of inflammation must be regarded as the local expression of a constitutional affection.

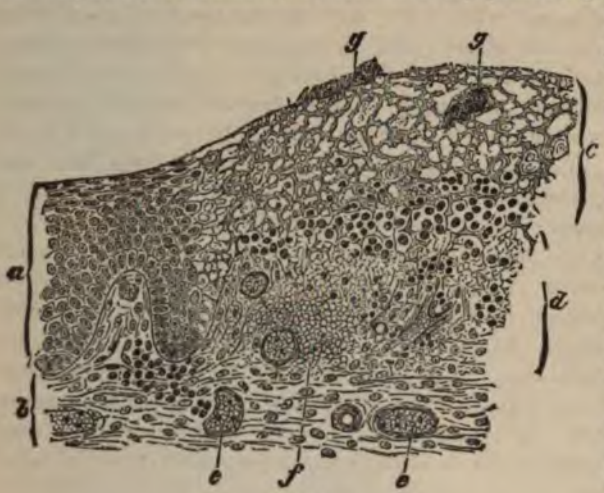

FIG. 5.

Diphtheritic Inflammation of Mucous Membrane.

Section through the uvula.

a, Normal epithelium; b, normal areolar tissue; c, necrosed epithelium transformed into a coarse mesh-work; d, areolar tissue infiltrated with fibrin and leucocytes; e, blood-vessels; f, hemorrhage; g, heaps of micrococci. × 75.—*After Zeigler.*

Ulceration of Mucous Surfaces.—Necrotic processes may be the result of intense purulent catarrhs or diphtheritic inflammations of mucous surfaces. Superficial loss of substance from rapid epithelial degeneration, and ulcers formed by the bursting of small abscesses, are the chief varieties of necrosis, except in those catarrhal inflammations where the blood supply is so suddenly and completely shut off that the mucous membrane dies in bulk and sloughs away (as in acute dysentery). Most of the little abscesses that produce ulceration of mucous surfaces are due to obstruction of the follicles and lymph structures that lie in the substance of the membrane; in consequence of their obstruction their contents degenerate, an abscess is formed, and an ulcer is the result.

Parenchymatous Inflammation.—In parenchymatous inflammation, the cells which perform the functions of the organ, the blood-vessels, and the stroma are in a greater or less degree involved in the inflammatory process; and this may pursue an *acute* or *chronic* course. In a mild type of parenchymatous inflammation the cells are enlarged, granular, and opaque, and their functional activity is increased, the blood-vessels contain more than their normal quantity of blood, and the stroma is infiltrated with serum. The affected organ is slightly increased in bulk, but returns to its normal size if the inflammation terminates by resolution. If the inflammatory process is very intense and prolonged, the cells are destroyed, the circulation is checked or arrested, and the stroma is extensively infiltrated with serum and pus. The inflamed organ is greatly increased in size, assumes a livid or purple hue, and its functional activity is arrested. In chronic parenchymatous inflammation the cells undergo fatty degeneration and disintegration. The walls of the vessels undergo extensive thickening, their calibre is diminished, and they may be obliterated. The stroma is increased by the development of new tissue. The function of the affected organ is impaired, and never returns to its normal condition, as the resulting changes are permanent.

In interstitial inflammation the connective-tissue or stroma of the organ in the part involved is affected. If the inflammation is acute, it is usually suppurative. The pus formation may be limited to small areas, or it may be diffused. When the pus cells are few, resolution is possible, but if they are numerous and infiltrate a large area of the organ, abscesses are formed with dense, firm walls. If the inflammation is chronic, it ends in induration and cirrhosis, by the formation of new connective-tissue, but does not form pus. The new tissue corresponds in kind to the original stroma of the organ, and is permanent. The affected organ never returns to its normal condition.

Fate of Pus.—Pus may undergo absorption, be evacuated, become inspissated, or undergo caseous transformation. To be absorbed it must undergo fatty degeneration and become converted into granular matter; its absorption is accomplished by the lymphatics. Its evacuation is accomplished by an ulcerative process, established in the tissues which contain it. When it has been converted partly into fatty and partly into granular matter, it may become inspissated by the absorption of its liquid portion and remain unchanged for a long period. If it becomes incapsulated, it changes into caseous matter and remains as a cheesy mass.

HYPERTROPHY.

Hypertrophy is an enlargement of an organ or tissue, due either to an increase in the size of its elements or to an increase in their number. The former is called *simple hypertrophy*, and the latter *numerical hypertrophy* or *hyperplasia*.

In most cases of hypertrophy these two forms are found associated.

Examples of physiological hypertrophy are the enlargement of the uterus during pregnancy; the increase in the size of the mammary gland in certain uterine changes; the hypertrophy of the prostate in old men, and the increase in size of muscles following persistent exercise.

Causes of Hypertrophy.—1. Increased functional activity is a prominent cause of hypertrophy, as is shown in the heart when called upon to perform extra work in overcoming obstructions at its orifices, or an impeded circulation from obliterating changes in the arteries.

Also in long-continued obstruction to the outflow of urine from the bladder, requiring an increase of force to overcome the obstruction, the muscular coat of the bladder becomes hypertrophied, and in the same way the coats of the stomach and intestines become hypertrophied above old obstructions.

Hypertrophy of one kidney after removal or destruction of the other is the result of enforced functional activity thrown upon the remaining organ to enable it to carry on the normal elimination. All these are examples of what may be called conservative hypertrophy.

2. Increased supply of nutrition caused by augmented blood supply is a less frequent cause of hypertrophy. The formation of the corpus luteum of pregnancy, in contradistinction to what is known as the "false corpus luteum," is possibly the best example of this. The hypertrophy of the cheeks and nose, as seen in acne rosacea, is another illustration.

On account of the excessive amount of blood which surrounds areas of long-continued inflammation, there is often an excessive outgrowth of tissue. This is seen in the well-marked hypertrophy of bone which follows inflammation of the periosteum. Another example is the rapid outgrowth of hair in the neighborhood of ulcers and diseased joints.

3. A partial arrest or delay of the normal retrograde changes in tissues may equally result in hypertrophy when the nutritive changes are not lessened. Many cases of obesity are thus induced.

ATROPHY.

Atrophy is either a diminution in the size of the histological elements of a part, or a decrease in their number. The former is called *simple* and the latter *numerical* atrophy.

Simple atrophy occurs in ordinary emaciation, which affects first the subcutaneous adipose tissue, then the muscles, omentum, intestines, heart, and last of all the brain and skeleton. The fat is removed from the cells more or less completely, and they diminish in size, their walls and nuclei becoming more distinct. This process is a physiological one in certain tissues and organs, especially in those which have only a temporary function, as the supra-renal capsules, mammæ, female genital organs, and thymus gland.

In all pathological forms the simple atrophic tissue elements remain unchanged except in size. Degenerative changes are sometimes associated with this form of atrophy.

Numerical Atrophy.—In numerical atrophy the elements of an organ or tissue are diminished, not only in size, but in number. Gradually, isolated elements or groups of elements of a part become involved. In many respects this change is quite as important as necrosis, and very much more so than simple atrophy, for it involves absolute loss of tissue, a loss which cannot be restored except by the production of new elements, which is very seldom, if ever, possible.

Atrophied organs are firmer and more anæmic than normal.

The causes of atrophy may be either general or local.

General atrophy may be due—

First. To deficient supply of nutritive material.

Second. To excessive waste.

Third. To impaired nutritive activity.

Deficient supply of nutritive material is present in cases of gradual starvation, in obstruction in the alimentary canal (as occurs in cancer of the pylorus), in mal-assimilation due to diseases of the alimentary tissues, obstruction of the thoracic duct or lacteals, or disease of the mesenteric glands.

Excessive waste is the exciting cause when it is due to prolonged hemorrhages, suppurations, diarrhœas, and the excessive loss of albumen or sugar.

Impaired nutritive activity is an accompaniment of senile atrophy, where the vitality of the tissues gradually diminishes, and they become less able to assimilate the nutritive material brought to them by the blood.

Sometimes general atrophy results from a combination of these causes, as occurs in chronic phthisis.

The causes of *partial atrophy* are—

First. Insufficient supply of blood.

Second. Diminished functional activity.

Third. Excessive functional activity.

Fourth. Nervous influence.

Insufficient supply of blood may be due to partial obstruction of an artery by pressure of a tumor, by the contraction of new connective-tissue on the smaller vessels, the result of an inflammatory process. Pressure upon the skull in hydrocephalus, upon bone by an aneurism, are also examples of local atrophy from pressure.

Local or partial atrophy from *diminished functional activity* may be physiological or pathological. Examples of physiological atrophy are furnished by those parts which have a use only at certain periods of life, such as the ductus arteriosus, umbilical vessels, thymus gland, the mammæ, the female sexual organs, and the jaw after the falling of the teeth. Pathological examples of atrophy depending upon diminished functional activity are seen in the wasting of muscles after paralysis, dislocations, anchylosis, or chronic disease of bone or joints, and in the rectum after establishment of an artificial anus.

Examples of local atrophy from *excessive functional activity* are seen in the class of hyperkinetic diseases, of which writer's cramp, acute cardiac dilatation from violent exercise, and atrophy of the testicles from sexual excesses are examples.

Nervous Influence.—Muscles which have been cut off from their trophic nerves, as in hemiplegia after injury of the anterior cornua, show atrophy depending upon this cause. The atrophy of the muscles which occurs after degeneration of the nerve-cells in the anterior horn of the spinal cord is also an example. It has also been proven that nerve fibres which are cut off from their ganglia all soon atrophy.

DEGENERATIONS.

The tissues of the body may undergo certain morbid processes characterized by alteration in their *quality.* They degenerate, and, as it were, take a step backward in their development.

These changes may lead to an impairment of their function, and often to a complete destruction of their elements.

The term *degeneration*, in its strict sense, is synonymous with *metamorphosis*, and implies a direct transformation of the albuminous elements of the tissues into a new material.

It is often used, in a broad sense, to include *infiltration;* but, strictly speaking, infiltrations are characterized by the introduction into the tissues of a new material brought from without.

There are certain prominent types of degenerative changes whose histological characteristics are much the same wherever they occur. All structures of the body are more or less liable to them; so that when one is familiar with one of these types of degeneration in one tissue, he is practically familiar with it in every tissue of the body, and has only to recall the histological elements of a part, to understand the changes in any particular degeneration.

TABLE OF DEGENERATIVE PROCESSES AND INFILTRATIONS.

NAME.	NATURE OF PROCESS.	PARTS AFFECTED.	CAUSE.
Parenchymatous..	Degeneration.....	Epithelium, muscle.....	Fever, poisons, etc.
Fatty............	Infiltration	Gland cells, connective tissue.	Imperfect oxidation.
Fatty	Degeneration	Gland cells, coats of small arteries, muscle.	Impairment of quality or quantity of blood, etc.
Colloid	Degeneration	Epithelial cells.........	Unknown.
Mucoid..........	Degeneration ...	Connective-tissue.......	Unknown.
Calcareous.......	Deposit..........	Connective-tissue, epithelium.............	1. Excess of salts in the blood. 2. Anæmic and atrophic tissues.
Pigmentary	Deposit..........	Normal situations, seats of hemorrhage.	Hemorrhage, stasis, cachexias.
Amyloid	Infiltration	Blood-vessels...........	Syphilis, prolonged suppuration.

PARENCHYMATOUS DEGENERATION.

(*Cloudy Swelling.*)

Parenchymatous degeneration consists in an alteration of the anatomical elements of the body, especially the epithelial cells and muscle fibres, so that they appear swollen, cloudy, and filled with minute granules which look like fat granules, but differ from them in being soluble in acetic acid.

This condition has also been called albuminous, serous, and granular degeneration. The name cloudy swelling is the one by which it is now generally designated.

The parenchymatous cells of the glandular organs—as the liver and kidneys—are especially liable to be affected with this change. The organ becomes enlarged and of a pale color. If a microscopic examination is made, the cells appear swollen and opaque; the body of the cell is filled with albuminous granules, which give the appearance as if the cell was sprinkled with fine sand. The normal markings of the cell are lost, and the nucleus is obscured. Slight grades of this form of degeneration are very difficult to appreciate, for many of the cells are normally granular, and we must not forget that all cells after death become somewhat granular. A cell which has undergone this form of degeneration is not necessarily destroyed; if the change is only transitory, the cell may return to its normal condition. As a result of this form of degeneration the cardiac muscle and the muscles of the skeleton undergo exactly the same change as the epithelial cells.

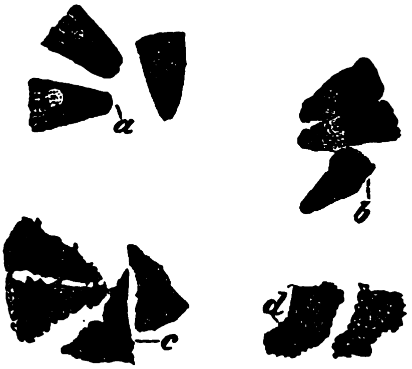

FIG. 6.

Cloudy Swelling of Cells.

a, Normal epithelium.
b, Commencing cloudy swelling.
c, d, Cells in extreme degeneration.

Parenchymatous degeneration occurs in most febrile diseases, especially the specific fevers, as pyæmia, diphtheria, scarlet fever, typhus, and typhoid fevers.

It is also seen after poisoning by arsenic, phosphorus, and the mineral acids.

High temperature was at one time supposed to cause it, but this has lately been abundantly disproven. This form of degeneration was at first supposed to represent the first stage of inflammation, and hence was called parenchymatous degeneration. While in a certain number of cases it may be considered as the result of the action on epithelial cells of the same influence which, acting upon the blood-vessels and connective-tissue, produces hyperæmia, exudation, and cell proliferation, still, in the majority of cases it cannot be considered a true inflammatory process.

FATTY INFILTRATION.

In fatty infiltration, fat is deposited within the cells of a tissue in the form of distinct drops.

In the earlier stages the drops are small, but as they increase in number they run together, and larger drops are formed, which gradually displace the nucleus of the cell and obscure more or less of its protoplasm. The vitality and function of the cell is but little impaired by the accumulation of fat; and the protoplasm, although compressed by the fat globule, is still unaltered, and quickly assumes its normal condition when the fat is removed.

Fatty infiltration is a normal physiological process under certain conditions, as during the growth of adipose tissue, or in the liver cells during the process of digestion. More or less fatty infiltration takes place in the liver of many healthy persons after a hearty meal, but in a few hours the fat is "burnt up," or oxidized, and the liver cells return to their normal condition.

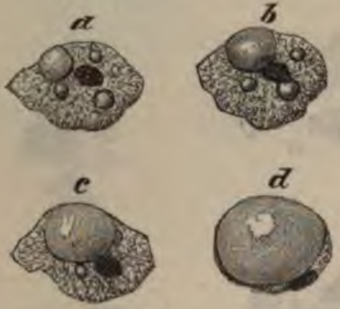

FIG. 7.

Fatty Infiltration of Liver Cells.

a, b, Early stages.
c, Nucleus of cell displaced by fat globule.
d, Cells completely filled by fat globule.

When for any reason the fat is not completely oxidized, then it collects in the tissues. This occurs under two opposite conditions: the one associated with general obesity, where an excess of fat, or substances capable of being converted into fat, are taken into the system, while the amount of oxygen received is insufficient to oxidize the excess, and it consequently accumulates in the cells; the other, in which there is general emaciation and a consequent impairment of the oxygenating power of the blood. As a result of this imperfect oxidation the fat contained in the food is incompletely oxidized, and so accumulated. This is well illustrated by the fatty infiltration often present in chronic phthisis. Fatty infiltration usually begins and is most marked in those cellular elements which are adjacent to the radicles of the nutrient vessels.

FATTY DEGENERATION.

This differs from fatty infiltration in that the fat is derived from the albuminous constituents of the tissues themselves.

It is a far more serious process, as it ends in the destruction of the cells; and, there being no provision for the production of new cells, the loss is a permanent one.

The fat makes its appearance as minute globules or granules in the protoplasm of the cell, the cell itself furnishing the fat from its own substance, and in time the cell is converted into a mass of granular fat, and is destroyed. In fatty infiltration the protoplasm is displaced by the fat which is deposited in the cell, but it does not suffer materially in its integrity; remove the fat, and the cell returns to its normal condition.

As, in fatty infiltration, the well-known physiological deposit of fat in the cells of the liver during digestion furnished a type of what might become a pathological process, so in fatty degeneration the secretion of milk illustrates a fatty degeneration which is perfectly normal. The young cells in the acini of the mammary gland become converted into fat, they break up, and the fatty matter constitutes the milk corpuscles.

Fatty degeneration depends upon all those conditions which interfere with the quality of the blood, so that the tissues are imperfectly nourished; as a result there is an interference with the oxidizing process in the cell, which loses its vitality and undergoes this form of degeneration. The diseases in which this change is apt to occur are, acute yellow atrophy of the liver, chronic alcoholism, progressive pernicious anæmia, and pulmonary phthisis.

It is also seen in poisoning by phosphorus, arsenic, and antimony.

The tissues which are most commonly the seat of fatty degeneration when it depends upon a general condition are the liver, kidneys, glands, heart, walls of the arteries, and voluntary muscles.

When the cause of this degeneration is local, it is due generally to an insufficient supply of blood to a part, as would result from narrowing of the nutrient blood-vessels. This is well seen in the heart, as the result of atheromatous changes in the coronary arteries. Nerves which have been separated from their nerve-centres undergo this degeneration. Organs and tissues which have been long disused, and in which, consequently, the quantity of circulating blood is diminished, also undergo fatty degeneration.

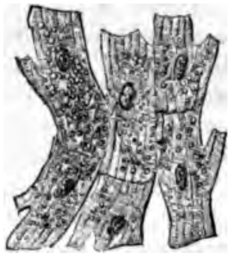

FIG. 8.

Fatty Degeneration of Cardiac Muscle.

This change is often seen in the tissues of old persons, especially in the cartilages, the walls of the arteries, the edge of the cornea (arcus senilis) and the lens (cataract). Cerebral softening is only fatty degeneration of brain tissue, caused by the cutting off of the normal blood-supply to the softened part.

COLLOID DEGENERATION.

Colloid degeneration is closely allied to mucoid, but differs from it in affecting principally the epithelial cells. The cells become filled with colloid material, which is derived from their substance. This material appears at first as minute spherules in the body of the cells, but eventually it escapes or is set free by the breaking down of the cells, and then the globules coalesce and form colloid masses.

Colloid matter resembles mucine, but differs from it. It is more dense, contains sulphur, and is not precipitated by acetic acid.

As a physiological process, colloid material is deposited in the thyroid gland, especially in the aged. When there is a large accumulation of this material in the follicles of the gland, then a goître is formed.

Colloid change is met with in lymphatic glands, in the tubules of altered kidneys, in the choroid plexus, and in certain new growths, as compound ovarian cysts and colloid cancers.

The cause of this change is unknown.

MUCOID DEGENERATION.

Mucoid degeneration consists in a transformation of the albuminous elements of the tissues into a transparent, homogeneous, colorless, jelly-like material called mucine.

Mucine is closely allied to albumen. Like albumen it is coagulated by acetic acid, but differs from it in not being redissolved by an excess of the acid, and in not containing sulphur. Mucoid change has its physiological prototype in the secretion of mucus by the goblet-cells of mucous membranes and the epithelium of certain mucous glands. The material is formed by metabolism from protoplasm of the cells.

Mucoid degeneration affects both cells and intercellular substance.

In epithelial structures mucoid degeneration can hardly be called a pathological process. In any catarrh of a mucous surface there will be found in the secretion cells which have undergone this degeneration.

Fibrous tissue is especially liable to undergo mucoid degeneration, and the albuminous intercellular substance is replaced by mucoid material. This is a return of the tissues to their fœtal elements, for mucine is found distributed in fœtal structures.

Mucoid degeneration is frequently met with in the intervertebral and costal cartilages of old people, in certain tumors, in bone, and in the disease known as myxœdema.

The causes of this degeneration are unknown.

CALCAREOUS DEGENERATION.

(*Calcification.*)

This form of degeneration is characterized by an infiltration in the tissues of calcareous particles composed mainly of lime and magnesia salts. It occurs physiologically in the formation of the bones of the skeleton, preceding actual ossification, and also in the formation of a deer's-horn. It is often confounded with ossification, but must be distinguished from it, for it never results in the formation of true bone.

The degeneration is common in old age in the coats of the arteries.

Tissues which have undergone calcareous degeneration are rendered hard and gritty. When the process has not advanced very far, the tissues feel as if they had been sprinkled with sand, as the deposit occurs in the form of irregular granules. At a later stage large masses are formed of a gray or whitish color, the result of an increase in the number and size of these granules. Under the microscope the affected tissue appears as an opaque mass in which no trace of structure can be distinguished, or at an early period the tissue and cells can be recognized, filled with small dark particles. If the tissue be treated with dilute hydrochloric acid, the salts dissolve, bubbles of carbonic acid gas appear, and the original structure of the part may be recognized.

The deposit occurs first and most abundantly in the intercellular tissue; afterwards the epithelial cells are involved.

The effect of this degeneration is to destroy the life of the part, which remains as an inert mass; but the histological structure of the part is not destroyed; the calcified cells remain, but they are dead; no further change occurs, and there is no softening. In these respects it differs from fatty degeneration, and is in fact a favorable termination of the latter.

The results of this change are very serious when it occurs in the arteries, for it destroys their elasticity and leads to rupture and aneurism; so also when it takes place in the valves of the heart.

It occurs as a conservative process when it checks the growth of tumors and renders pathological products inert, as is often seen in the lungs in a case of arrested phthisis.

Calcareous degeneration may occur under two conditions.

1. When there is an excess of salts in the blood. 2. When there is no such excess, and the deposit takes place when certain alterations have occurred in the blood or tissues.

The *first* is by far the least frequent; it occurs in certain forms of softening of bone, osteomalacia, and extensive caries. Apparently as the bone breaks down, its salts are taken up by the blood and deposited in other places. In such cases it is usually quite general, involving the kidneys, lungs, dura mater, liver, stomach, and intestines.

In the *second* it is local and due apparently to a diminution of vitality in the tissues by which the normal relation between the tissues and the small amount of calcareous salts in the blood is destroyed.

Atrophic and retrogressive processes are apt to be accompanied by it.

The favorite seats of calcific deposits are tumors, in and about parasites, old blood clots in veins, cheesy deposits, false membranes, and other pathological products.

PIGMENTATION.

Pigmentation is characterized by the presence in the tissues of a colored substance, amorphous or crystalline, which is derived from the coloring matter of the blood.

Normal pigmentation occurs in the choroid, rete mucosum, and in some motor cells of the cerebral peduncles.

An absence of this normal pigment is seen in albinos; an increase in Addison's disease, freckles, melanotic tumors, and brown atrophy of muscle.

Pathological pigmentation follows the extravasation of red blood-corpuscles, and the escape of their coloring matter, which may also be liberated from the corpuscles while in the vessels.

This coloring matter (hæmoglobin) infiltrates the tissues and stains both the cells and intercellular substance. It becomes converted into hæmatoidin, and appears as minute yellow and reddish-brown granules, or as orange-colored needles and rhombic prisms.

Pigmentation occurs in most cachectic conditions; especially in chronic malarial poisoning by a destruction of the corpuscles in the blood. Local causes are rupture of small vessels, and those which cause obstruction to the flow of the blood and favor diapedesis of the red blood-corpuscles, as in a nutmeg liver and brown induration of the lungs, dependent upon heart disease.

The staining of the tissues yellow with the coloring matter of the bile, as occurs in "jaundice," is not true pigmentation.

Pigmentation may follow the introduction of extraneous substances into the body, as occurs in the lungs by the inhalation of coal dust, soot, and particles of iron, and in the skin and lymphatic glands by tattooing.

AMYLOID DEGENERATION.

This form of degeneration is characterized by the presence in the tissues of a homogeneous, structureless, translucent substance, differing but little from albumen, and giving peculiar reactions with certain staining reagents.

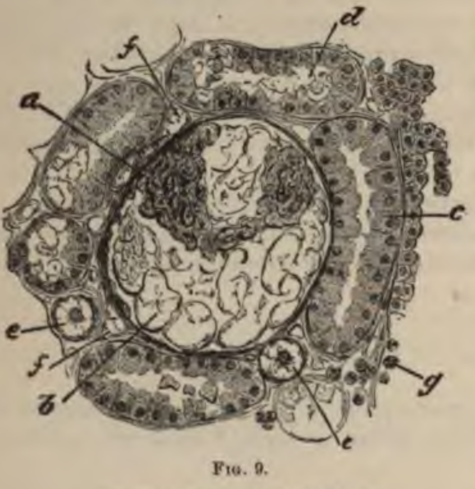

FIG. 9.

Amyloid Degeneration of Kidney.

a, *Normal capillary loop.*
b, *Amyloid capillary loop.*
c, *Normal epithelium in tube.*
d, *Hyaline tube cast.*
e, *Amyloid arteriole.*
f, *Amyloid capillary.*
g, *Loosened fatty epithelium.* × 300.

Iodine in solution gives it a mahogany brown color; iodine and sulphuric acid a blue color; methyl blue, and gentian violet a bright red or pink color.

The nature of this material is still in dispute. Virchow held it was starchlike, and gave it the name "amyloid;" other writers, from its resemblance to wax or lard, have called this change "waxy" or "lardaceous" degeneration.

Amyloid material is an albuminoid rich in nitrogen, and is in all probability derived from the fibrin of the blood by direct metamorphosis (amyloid reaction having been obtained in the fibrin of a hæmatocele). As this material is found in its earliest stage in close proximity to the blood-vessels, it in all probability soaks out from the blood into the tissues and is in reality an infiltration.

The change is a pathological one. There is no physiological analogue unless we consider senile changes physiological, for amyloid degeneration is an almost constant accompaniment of senility, in the cartilages of joints, especially the sterno-clavicular and vertebral.

Under other more strictly pathological circumstances, amyloid degeneration may involve almost every kind of tissue ; but the change shows itself at first always in the walls of the capillaries and the small arteries.

They become thickened, have a homogeneous look, and their lumen is diminished. Not every vessel is affected, nor is the change regularly distributed over the same vessel. In time, as the process advances, the connective-tissue may become involved, and last of all, and this but seldom, the epithelial cells may show this change.

Amyloid degeneration may be local, but usually a number of organs are affected at the same time.

The affected organ is enlarged, and heavier (denser) than normal, its capsule is tense, its borders rounded, its surface paler and dryer than normal, and of a grayish appearance.

The organs which are most frequently affected with this change, in the order of their frequency, are the spleen (sago-spleen), kidneys, liver, lymphatic glands, mucous membrane of the stomach and intestines, and occasionally the pancreas, thyroid, lungs, ovaries, and supra-renal capsules.

Although this degeneration is a constant accompaniment of old age, it occurs pathologically principally between the ages of twenty and forty. It depends upon some nutritive disturbance, and in a large proportion of cases is associated with prolonged suppuration of bone (out of 96 cases examined by Wilks, bone suppuration existed in 68 and had been present in 17 others).

It is common in persons suffering from certain cachexias. Pulmonary phthisis is one of its most common antecedents. Syphilis and chronic malaria are apt to lead to it.

The prognosis of the disease is very unfavorable, and, considering the resistance of this substance to all reagents, it seems improbable that we shall ever be able to combat it successfully.

Corpora amylacea are concretions which have been supposed to be identical with amyloid material ; they give the same color-reaction with iodine, and with iodine and sulphuric acid. It is now known that they have no relation to amyloid degeneration, and are of no special significance. They are hard, stratified concretions found in the prostate, seminal vesicles, and in the central nervous system, and are very common in the aged.

NECROSIS.

Necrosis is a term applied to the death of a portion of the body. In the soft tissues the process is mortification, and the dead tissue a slough.

As soon as a tissue dies, it becomes subject to the conditions of inorganic bodies. Its materials are held together only by cohesion, and that cohesion usually yields promptly to the forces of decomposition due to the action of a ferment generated by certain bacteria. When the entire body dies, its component parts are no longer capable of taking up new material and changing it into living tissue ; as a result the body is unable to resist decomposition, and this inability is made manifest when the first symptoms of dissolution appear. All the elements of the body do not lose their vitality at the same time; as a rule the epithelial cells die sooner than the connective-tissue framework. When a part of a living body dies (necrosis), it undergoes similar changes to those occurring after death of the whole body, being modified only by the locality in which it occurs.

The causes of necrosis are:

1. *Arrest of blood supply,* caused by obstruction in the arteries, veins, or capillaries,—in the arteries by ligature, compression, embolism, thrombosis; in the veins principally by pressure or thrombosis; but the anastomoses are so free in the veins that death of the part seldom occurs from this cause alone. Obstruction in the capillaries is much more serious; for when complete capillary stasis occurs,—as from pressure or diminished heart power,—the vitality of the capillary walls is destroyed, and necrosis results.

2. *Direct Injuries to the Cells.*—Such injuries are:

a. Mechanical, as chemical corrosives, crushing, and animal poisons.

b. Bacteria, setting up septic processes, as from putrid pus, dead tissue, decomposing urine, etc.

3. *Abnormal Temperatures.*—All changes which cause slight increase or decrease of temperature produce inflammation, but when the variations of temperature are excessive, necrosis is the result: this is seen after frostbites or a burn.

The above causes of necrosis do not always act singly, but are often combined; for example, in a part which is imperfectly supplied with blood, only a slight injury to the cells may cause necrosis; or, again, let the vitality of the tissues be weakened by an extreme cachexia, and a slight interference with the blood supply will cause necrosis. An illustration of the first is seen in senile gangrene, and of the second in gangrene in children (noma). Certain general pathological conditions may so lower the vitality of the whole body that slight causes will produce necrosis. This is illustrated by the sloughing bed-sores which form in adynamic fevers and exhausting diseases on parts of the body exposed to pressure, and in the liability to senile gangrene, which is increased by the diminished heart power and arterial changes that accompany old age.

The rapidly forming bed-sores which result from injury to the spinal cord are regarded by some as due to special trophic changes, but it seems more probable that they are the result of a general vaso-motor disturbance, which produces paralytic hyperæmia, and so necrosis.

Varieties of Necrosis.—Restricting the term "necrosis" to a local death of tissue, the following varieties are met with:

1. *Dry Gangrene or Mummification.*—If tissues that are the seat of this change contain little water, and are composed largely of earthy matters, they may preserve their outline and appearance for a long time. If the water contained in them evaporates rapidly, they shrivel and become hard.

2. *Moist Gangrene.*—Tissues that are the seat of this variety of necrosis undergo putrefaction, and, as the evaporation of the water contained in them is prevented, they are moist, soft, and doughy, and in time gases are formed, and their color becomes dark. Such changes are similar to those that occur in the body after death. Moist gangrene always has its seat in parts which are exposed to the air.

3. *Hospital Gangrene.*—This is a progressive necrosis which affects wounds, and is due to a special micro-organism (septic).

4. *Senile Gangrene.*—It affects the aged, and is due to an impeded circulation from atheromatous, or calcareous changes in the arteries.

5. *Coagulation Necrosis.*—In this variety of necrosis the cells of a tissue undergo a change analogous to the coagulation of the fibrin of the blood. Their protoplasm becomes rigid and converted into a homogeneous hyaline substance; their nuclei disappear, and finally the cell breaks up into a granular-looking débris. Coagulation necrosis may affect entire organs, or portions of organs or cells. It occurs in infarctions, tubercular nodules, tumors, waxy degeneration of muscles, and in diphtheria, typhoid and relapsing fevers.

The terminations of necrosis, when putrefaction is absent, are:

1. *Liquefaction.*—In tissues where there is an abundance of liquid, and its removal is prevented by non-evaporation, or the action of the lymphatics is imperfect, the softened tissue becomes converted into a thick creamy fluid. This occurs especially in brain necrosis, and in the softening of infarctions and thrombi.

2. *Caseation.*—This is a change which results in the formation of a more or less dry, whitish-yellow mass resembling cheese. It is met with most frequently in encapsulated collections of pus, in tubercular masses, and in inflammatory products of low vitality. As the cell elements of such masses die, their watery portions are absorbed, they become granular and dry up. The fat contained in them becomes partly saponified, and crystals of cholestrine are deposited. If there are any cells which have not been entirely destroyed, they shrivel and atrophy. This cheesy change is not always a final one, for the cheesy mass may become calcified, calcareous matter (lime salts) being deposited in and about it. The physical organism behaves towards all necrotic tissues the same as it does towards foreign bodies. If possible, it removes them; when unable to do this, it surrounds them with a fibrous capsule and so renders them inert.

TUBERCLE.

To-day experimental research teaches that tubercle is the result of an infectious inflammatory process, and that the infection which excites the inflammation is a specific vegetable parasite, the "tubercle bacillus," which gains entrance into the body through the respiratory and digestive tract, rarely through the genito-urinary passages or wounds of the skin.

Wherever the bacillus lodges and finds conditions suitable for its develop-

ment and multiplication, there are found in a short time small, gray, translucent nodules, looking like particles of coarse sand, which are called miliary tubercles. If a tubercle is examined microscopically before any degenerative changes have taken place, it will be found to be composed of a reticulated basement substance, lymphoid, epithelioid and giant cells arranged in the following manner: near its centre are one or more large branching cells, the processes of which blend with the surrounding growth. These are the giant cells, which have a homogeneous cell body and a number of nuclei arranged around the periphery like a belt or ring; surrounding these giant cells are a large number of epithelioid cells with single nuclei, packed so closely together that it is almost impossible to distinguish individual cells. Beyond these cells, as one looks towards the periphery of the nodule, is seen a large number of lymphoid cells. These three varieties of cells are enclosed and supported by a fine reticular stroma, similar to that of a lymphatic gland. The explanation of the formation of a tubercle is as follows: The tubercle bacillus, acting as a specific poison, sets up an inflammatory process, which leads first to an accumulation of lymphoid cells with some proliferated fixed cells of the part; some of the lymphoid cells change into epithelioid cells, and a few of the latter become giant cells by the enlargement of the cell body and a repeated division of their nucleus. Tubercles are non-vascular structures, no vessels have ever been found in them; sometimes they start from the wall of a vessel, and often, in early growth, the remains of a partially obliterated vessel are seen in them, which has given rise to a dispute whether they were vascular or non-vascular growths. The changes which take place in tubercles are as follows:

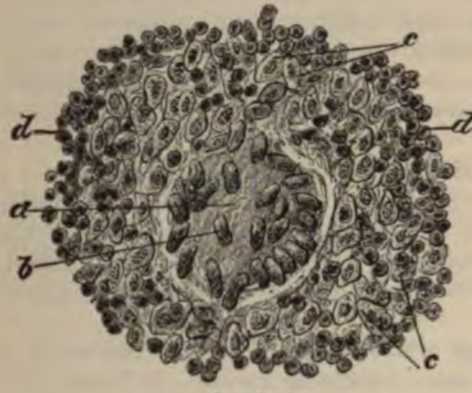

FIG. 10.

Miliary Tubercle.

a, Giant cell.
b, Nuclei of giant cell.
c, Epithelioid cells.
d, Lymphoid cells. × 300.

1st. They undergo caseous or cheesy degeneration. As they are non-vascular, their centres are shut off from nutrition, and as they are subjected to the necrotic action of the tubercle bacilli, in a short time they die and undergo what is called "coagulation necrosis," so that in old tubercles no cells can be found in the centre of the tubercular nodule. Just outside of the caseous centre will be found giant cells blended with a few epithelioid cells, while still further from the centre, constituting the peripheral zone of the nodule, are a large number of lymphoid cells. By special methods of staining the tubercle, bacilli may be found in the

caseous centre and often in the giant cells. The ultimate change in a tubercle which has undergone caseous degeneration is disintegration with the formation of an irregular abscess cavity. If a number of miliary tubercles are united to form a tubercular mass, as is often seen in the lungs, the union of several of these small abscesses forms cavities of considerable size, in the walls of which are generally found a large number of tubercle bacilli.

These cheesy masses may become encapsulated, calcified, and remain as inert masses for an indefinite period.

2d. Tubercle may undergo fibroid change. The tissues which surround the tubercle taking on a chronic fibroid inflammation, the newly formed connective-tissue contracts, and, compressing the tubercle, converts it into a fibrous nodule. This change is most likely to occur in the aged, and in those of a strong fibrous diathesis.

The characteristics of a typical tubercle may be briefly summarized as follows: First, a non-vascular nodular growth; second, a growth composed of reticular basement substance and giant epithelioid and lymphoid cells; third, this growth has a tendency to undergo coagulation necrosis due to its non-vascularity and the direct action of the tubercle bacillus; fourth, the special and distinguishing characteristic of this growth is the tubercle bacillus.

TUBERCULOSIS.

We now understand, by the term *tuberculosis,* a diseased condition caused by the introduction into the body of tubercle bacilli. Its anatomical characteristic is the development of specific nodules. Its clinical characteristic is the consecutive invasion of one organ or of the entire system.

Tuberculosis may be local or general: *local,* when it is limited, and gradually destroys the organ or tissue primarily involved, as occurs in tuberculosis of the lungs, kidney, liver, or peritoneum; *general,* when large numbers of tubercle bacilli gain entrance into the general blood current, and in a few weeks develop in the various organs of the body a multitude of miliary tubercles, called acute miliary tuberculosis.

It has been shown by experiments on animals that the tubercle bacilli and their spores are alone the infectious agent, and that they travel in the blood current; and wherever they lodge, there the characteristic cellular changes take place, and a tubercle is developed. We consequently reach the conclusion that tuberculosis is an infectious disease, and that its cause is the tubercle bacillus.

The danger of local tuberculosis is that it may become general; for the first irruption of tubercles in the neighborhood of an existing tubercular focus is usually followed, sooner or later, by the appearance of nodules in the lymphatic system. It is in the glands that these tubercular irruptions are most intense; generally the process makes a kind of halt in these gland stations, but in time spreads onwards, and at length reaches the thoracic duct, and through it the general blood current.

We therefore conclude that tubercular infection may take place in the

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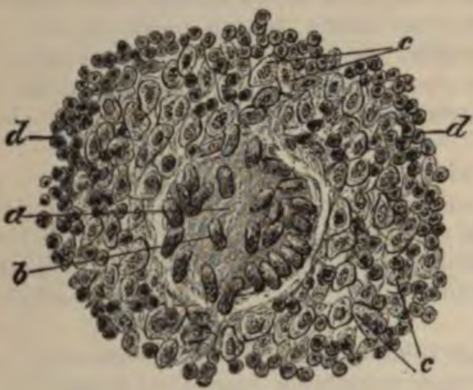

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TUBERCULOSIS.

We now understand, by the term *tuberculosis,* a diseased condition caused by the introduction into the body of tubercle bacilli. Its anatomical characteristic is the development of specific nodules. Its clinical characteristic is the consecutive invasion of one organ or of the entire system.

Tuberculosis may be local or general: *local,* when it is limited, and gradually destroys the organ or tissue primarily involved, as occurs in tuberculosis of the lungs, kidney, liver, or peritoneum; *general,* when large numbers of tubercle bacilli gain entrance into the general blood current, and in a few weeks develop in the various organs of the body a multitude of miliary tubercles, called acute miliary tuberculosis.

It has been shown by experiments on animals that the tubercle bacilli and their spores are alone the infectious agent, and that they travel in the blood current; and wherever they lodge, there the characteristic cellular changes take place, and a tubercle is developed. We consequently reach the conclusion that tuberculosis is an infectious disease, and that its cause is the tubercle bacillus.

The danger of local tuberculosis is that it may become general; for the first irruption of tubercles in the neighborhood of an existing tubercular focus is usually followed, sooner or later, by the appearance of nodules in the lymphatic system. It is in the glands that these tubercular irruptions are most intense; generally the process makes a kind of halt in these gland stations, but in time spreads onwards, and at length reaches the thoracic duct, and through it the general blood current.

We therefore conclude that tubercular infection may take place in the

ment and multiplication, there are found in a short time small, gray, translucent nodules, looking like particles of coarse sand, which are called miliary tubercles. If a tubercle is examined microscopically before any degenerative changes have taken place, it will be found to be composed of a reticulated basement substance, lymphoid, epithelioid and giant cells arranged in the following manner: near its centre are one or more large branching cells, the processes of which blend with the surrounding growth. These are the giant cells, which have a homogeneous cell body and a number of nuclei arranged around the periphery like a belt or ring; surrounding these giant cells are a large number of epithelioid cells with single nuclei, packed so closely together that it is almost impossible to distinguish individual cells. Beyond these cells, as one looks towards the periphery of the nodule, is seen a large number of lymphoid cells. These three varieties of cells are enclosed and supported by a fine reticular stroma, similar to that of a lymphatic gland. The explanation of the formation of a tubercle is as follows: The tubercle bacillus, acting as a specific poison, sets up an inflammatory process, which leads first to an accumulation of lymphoid cells with some proliferated fixed cells of the part; some of the lymphoid cells change into epithelioid cells, and a few of the latter become giant cells by the enlargement of the cell body and a repeated division of their nucleus. Tubercles are non-vascular structures, no vessels have ever been found in them; sometimes they start from the wall of a vessel, and often, in early growth, the remains of a partially obliterated vessel are seen in them, which has given rise to a dispute whether they were vascular or non-vascular growths. The changes which take place in tubercles are as follows:

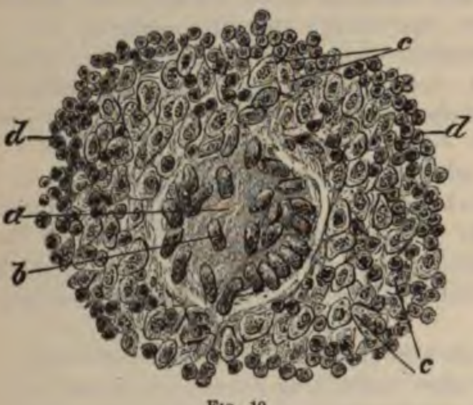

FIG. 10.

Miliary Tubercle.

a, *Giant cell.*
b, *Nuclei of giant cell.*
c, *Epithelioid cells.*
d, *Lymphoid cells.* × 300.

1st. They undergo caseous or cheesy degeneration. As they are non-vascular, their centres are shut off from nutrition, and as they are subjected to the necrotic action of the tubercle bacilli, in a short time they die and undergo what is called "coagulation necrosis," so that in old tubercles no cells can be found in the centre of the tubercular nodule. Just outside of the caseous centre will be found giant cells blended with a few epithelioid cells, while still further from the centre, constituting the peripheral zone of the nodule, are a large number of lymphoid cells. By special methods of staining the tubercle, bacilli may be found in the

caseous centre and often in the giant cells. The ultimate change in a tubercle which has undergone caseous degeneration is disintegration with the formation of an irregular abscess cavity. If a number of miliary tubercles are united to form a tubercular mass, as is often seen in the lungs, the union of several of these small abscesses forms cavities of considerable size, in the walls of which are generally found a large number of tubercle bacilli.

These cheesy masses may become encapsulated, calcified, and remain as inert masses for an indefinite period.

2d. Tubercle may undergo fibroid change. The tissues which surround the tubercle taking on a chronic fibroid inflammation, the newly formed connective-tissue contracts, and, compressing the tubercle, converts it into a fibrous nodule. This change is most likely to occur in the aged, and in those of a strong fibrous diathesis.

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We therefore conclude that tubercular infection may take place in the

following way: First, by the blood, as when tubercular masses grow into the pulmonary veins. Second, by the lymphatic system. Third, by continuity, as on the surface of mucous or serous membranes. Fourth, by the direct application of the bacilli to a part, as when a tubercular mass is coughed up, and, before it can be expectorated, falls back into another bronchus.

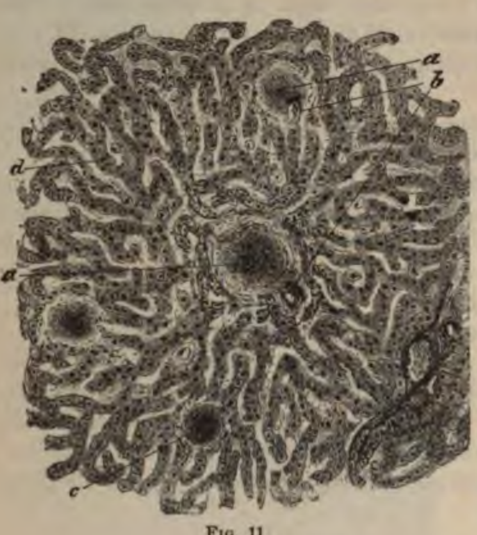

Fig. 11.

Miliary Tubercles in the Liver.

× 250 *drawn by camera lucida.*

a, Miliary tubercle.
b, Giant cells.
c, Tubercle with cheesy centre.
d, Normal liver cells.

At the present time the following questions in the pathology of tuberculosis are being discussed by scientists:

1. Can man become infected with tubercle by eating tubercular meat, or by drinking the milk of tubercular cows?

2. May not the tubercle bacilli contained in the sputum and other discharges from tubercular subjects be infecting agents, and thus render tuberculosis a contagious disease?

3. As the tubercle bacilli preserve their vitality for a long time outside the body, may not those contained in the sputum of tubercular subjects, after the sputum is dried and becomes pulverized, be inhaled with the air as dust particles, and set up tubercular processes in the respiratory organs of non-tubercular subjects?

4. As a preventive measure for the spread of phthisis, should not the sputum and other discharges be burned or disinfected in their fresh state?

Accepting the doctrine that the tubercle bacillus is the only cause of tuberculosis, we are compelled to answer these questions affirmatively, and to say that the doctrine of heredity of tuberculosis must be abandoned, the tubercular taint being nothing more than a hereditary enfeeblement, which furnishes a better soil for the lodgment and development of the tubercle bacilli, or a physical condition which is less able to resist their invasion.

BACTERIOLOGY.

Bacteria are microscopic organisms belonging to the vegetable kingdom, whose characteristics are the absence of chlorophyl and their peculiar method of reproduction by transverse scission, or by means of small spherules called spores. The science of bacteria is termed *Bacteriology.*

To facilitate bacterial studies I shall divide the subject into four sections:

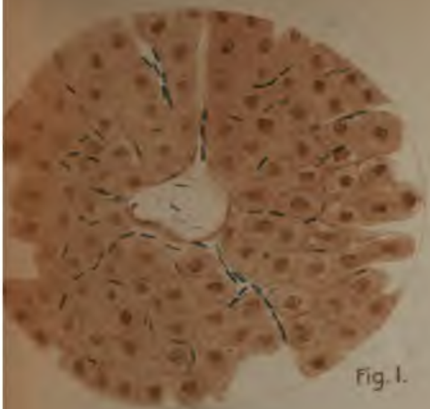

Fig. I.

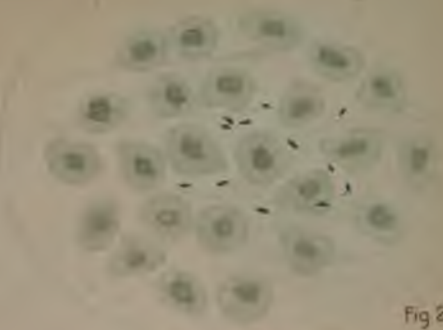

Fig 2.

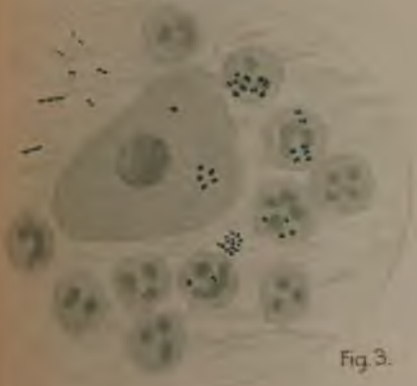

Fig. 3.

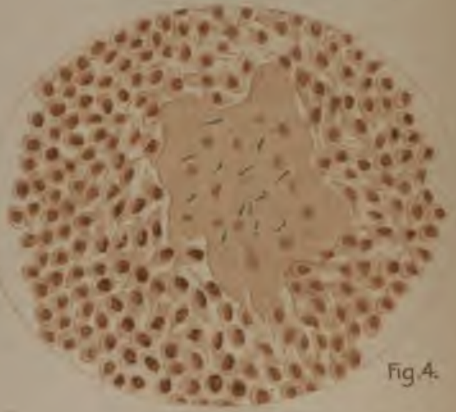

Fig. 4.

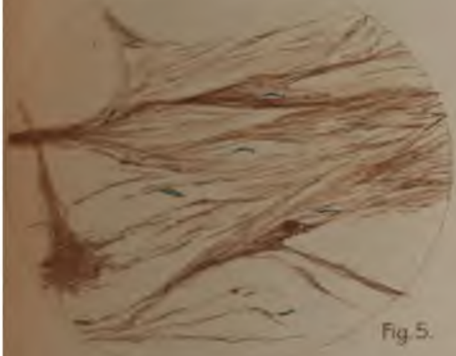

Fig. 5.

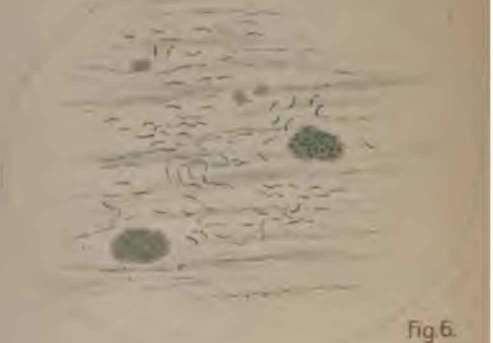

Fig. 6.

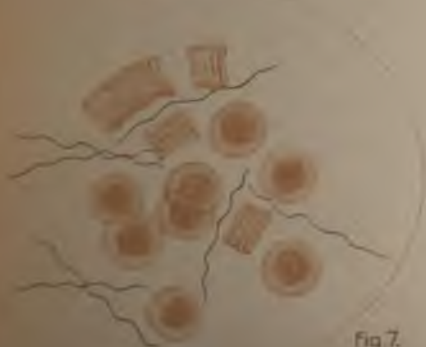

Fig. 7.

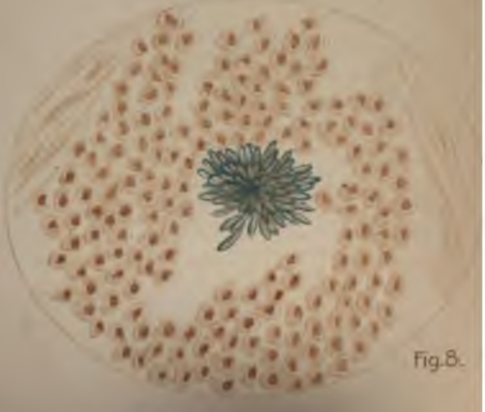

Fig. 8.

PATHOGENIC BACTERIA

BY J. M. BYRON, M.D.

First. Their technology, or the consideration of the principal facts relating to cultures, staining, etc.

Second. A consideration of their forms, dimensions, classification, etc.

Third. Their biology, including their origin, nutrition, constitution, reproduction, and special action.

Fourth. A description of those whose pathogenic action on man has been established.

TECHNOLOGY.

In order to study the life history of the different known organisms, pure cultures must be obtained. A pure culture is an artificial growth on nutrient media, of a single species of micro-organisms.

Media.—Nutrient media are either *artificial* or *natural.* Natural media are obtained directly from man or animals, as blood serum, pleuritic fluid, hydrocele fluid, etc.

Artificial media are prepared from different substances and in different ways. Media may be liquid or solid.

All media must be sterilized before using for cultures; that is, they must be made free of all germs that by contamination may be contained in them. This is accomplished by subjecting the media several times to a temperature ranging from 50° C. to 120° C.

All vessels and instruments used must be sterilized by heating in dry air to a temperature of 150° C. for an hour.

Natural Media.—Blood serum is obtained by leaving in a cool place, for twenty-four to forty-eight hours, the blood of an animal in a sterilized jar. The blood-corpuscles precipitate with the clot, and the clear serum is then transferred to test tubes plugged with cotton-wool, which have been previously submitted to a dry temperature of 150° C. for an hour. The tubes and serum are then submitted during eight days—one hour every day—to a temperature of 50° C. If solid blood serum is desired, after the eighth day the tubes are transferred to the serum inspirator, and the temperature slowly raised to 70 or 80° C., when the blood serum coagulates. Pleuritic and hydrocele fluids are treated in the same way.

Potatoes and other vegetables, as well as eggs, can be used for cultures with advantage in some cases. They are sterilized by boiling them, or keeping them fifteen minutes a day, during three days, at a temperature of 100° C.

Artificial Media.—Pasteur's, Cohn's, and other fluids classed as mineral media, are made by mixing certain salts and water.

Bouillon is the principal ingredient used, either as a medium itself or in the preparation of the artificial media used in bacteriological culture. It is prepared by boiling one part of lean chopped beef and two parts water. Filter, and add peptone, 1%; salt, ½%: filter again, and sterilize. To make gelatine: To 100 parts of bouillon add 7 to 10 parts good gelatine which has been previously soaked in water, heat in water bath, and pour in sterilized test tubes. If agar is needed, take 1 part agar and soak for a night in salt water; rinse and add it to 100 parts bouillon, heat till completely dissolved, filter, and pour in tubes and sterilize. All media should be made slightly alkaline in re-action; sodium carbonate is best suited for this purpose.

Cultures.—To obtain pure cultures, inoculate, by means of a recently sterilized platinum needle, with the substance suspected to contain micro-organisms, gelatine and agar tubes; the former are kept at a constant temperature of about 22°; the latter at a temperature of 37° to 40° C.

If a pure culture is obtained from the beginning, which is seldom the case, the micro-organism can be directly studied; but if an impure culture is obtained, which is the most frequent case, the cultures are *plated;* that is, very dilute cultures are spread out on sterilized glass plates, in order to allow every microbe to grow separately. This operation is done as follows: Three tubes of gelatine or agar are heated to the melting-point, then with a platinum needle inoculate from the impure culture one of the tubes; sterilize needle, and from this first tube inoculate a second tube; and from this in the same manner a third tube; thus very few micro-organisms are contained in the last tube, perhaps six or eight only. Now spread the contents of those tubes on pieces of sterilized glass 5 × 6 inches. After the gelatine or agar has hardened, the glass plates are allowed to remain at an even temperature; then the colonies of bacteria begin to grow. Each colony is inoculated with the platinum needle in a separate tube, and cultivated.

Most bacteria grow freely in nutrient gelatine or agar, as above described, but some require particular media, as tubercle bacilli, gono-cocci, etc.; others do not grow in any of the known media, as the spirillum of relapsing fever, bacillus of syphilis, etc.

EXAMINATION AND STAINING OF BACTERIA.

Micro-organisms are very resistant to the action of acids and alkalies. This property was utilized to recognize them before the staining methods were used. If a section or specimen be treated by acetic acid, or with a 2% solution of caustic potash, all the components disappear, and the bacteria only remain visible. Some protoplasmic granules and crystals may resist the action of such treatment, but the special form of the micro-organisms, their uniform dimensions, and their peculiar grouping, make their recognition easy. This method is known under the name of its author,—Baumgarten's method. Günther has modified this method, principally when treating specimens kept for a long time in alcohol. In such cases it frequently happens that the granular protoplasm is affected neither by acids nor alkalies; Günther treats them with a 3% solution of pepsine, which, peptonizing all the albuminoids, clears the field, leaving the micro-organisms unaffected.

Staining.—The best method of staining micro-organisms is important; for by it the bacteria are made more distinct, and a chemical action takes place which in many cases is characteristic of a determined species.

Aniline colors are chiefly used for staining. Acid dyes have the property of diffusely coloring tissues; the principal ones are eosine, aurine, and acid fuchsine. Basic dyes have an elective tendency to color the nuclei of cells and bacteria: these are methyl blue, methyl violet, gentian violet, dahlia, basic fuchsine, Bismarck brown, etc.

Any water solution of a basic color will do for coloring bacteria. It is well to heat the specimens after staining with acetic acid, as in Baumgarten's method; by thus dissolving the nuclei, the colored bacteria are much more distinct. This method gives excellent results in the study of the spirillum of relapsing fever.

Not all micro-organisms can be colored by this method. Those that resist it may be treated with Löffler's solution (concentrated alcoholic solution of methyl blue 30 parts, $\frac{1}{10000}$ water solution of caustic potash 100 parts). After leaving the sections for a few minutes in this solution, treat them with 1% acetic acid, then pass to absolute alcohol and clear with cedar oil. This method shows very distinctly the bacillus of typhoid fever in tissues.

Gram's method is a general method for staining. The sections are first treated with water solution of aniline oil 100 parts; saturated alcoholic solution gentian violet 5 parts (the sections must have been kept, previous to staining, in concentrated alcohol). After a few minutes they are transferred to a solution of iodine (iodine 1 part, potassium 2 parts, water 300 parts), where they are left for two or three minutes; from this solution the sections are put in absolute alcohol, where they decolorize almost entirely; clear in oil of cloves and mount in balsam. By this method the micro-organisms are deeply stained, while the tissues remain colorless; such preparations can be stained with a contrast stain, as a water solution of Bismarck brown—and the relations of tissues and micro-organisms studied.

Günther has modified Gram's method, and his modification is to be recommended where old sections, kept in impure alcohol, are dealt with. The sections are kept for a minute at ordinary temperature in the violet solution, dried with blotting-paper, and transferred to the iodine solution for two minutes, then placed in alcohol for one minute, treated for exactly ten seconds by a 3% hydrochloric acid, alcoholic solution, and washed in alcohol again. Clear with oil of cloves, keep in balsam.

Weigert has also modified Gram's method: when the sections are taken from the iodine solution they are decolorized by aniline oil instead of alcohol; thus not only micro-organisms remain colored, but also fibrin, which is important in certain cases.

The micro-organisms that stain by Gram's method and its modifications are:

Pneumococcus of Friedländer.
Diplococcus pneumoniæ of Fränkel.
Bacillus anthracis (Davaine).
Bacillus lepræ (Hansen).
Bacillus tuberculosis (Koch).
Streptococcus erysipelatis (Fehleisen), all pyogenic bacteria and actinomyces.

Those that do not stain are:

Gonococcus (Neisser).
Bacillus typhosus (Eberth).
Diplococcus intercellularis meningitis (Weichselbaum).
Bacillus mallei (glanders).

Spirillum choleræ asiaticæ (Koch).

Spirochæte Obermaieri (relapsing fever).

Gonococci, diplococcus intercellularis, and bacillus typhosus stain well with Löffler's solution as already indicated; bacillus mallei, spirillum choleræ asiaticæ, and spirochæte Obermaieri, stain with ordinary water solutions of dyes, and afterwards are treated with 1% acetic acid.

All these preparations can be double-stained with a contrast color in water solution, or with picro-lithio-carmine, excepting the streptococcus erysipelatis. For this micro-organism the sections are first treated with any of the carmine solutions, washed in water, transferred to alcohol, and treated by Gram's method. Thus the nuclei and tissues remain colored by the carmine or picro-carmine solutions, while the bacteria are of a deep-blue color. These preparations are exceedingly beautiful, and the staining, first of the tissue and then of the bacteria, gives such distinctness that it ought to be recommended for general use.

The importance of tubercle bacillus in diagnosis is such that a special mention should be made of the particular method of its staining, which makes it easily distinguished from all known bacteria excepting lepra bacillus. The property of retaining the color when treated by strong mineral acids is peculiar to this bacterium. The sections, or cover-glass preparations (sputum, pus, blood, etc.), are first treated by a solution composed of—

Aniline water	100 parts.
Alcoholic solution of fuchsine, methyl blue, or methyl violet	11 parts.
Absolute alcohol	10 parts (Erlich).

If sections, they should be heated for a few minutes in this solution (rapid process), or kept for twenty-four hours at ordinary temperature; if cover-glass preparations, pass them over a burner three or four times until steam begins to arise. Then wash with—

Nitric acid	1 part
Water	2 or 3 parts,

until nearly decolorized, then wash in abundant water or alcohol, and stain with contrast color in water solution. If section, treat with alcohol, clear with oil of cloves, and keep in balsam.

By this method the tubercle bacillus alone remains colored after the action of the nitric acid; the contrast stain colors the tissues.

This is the classic method of staining tubercle bacilli, and is called—after the authors—Erlich-Koch's method. Many others have been proposed, but all are more or less a modification of the one given. Lepra bacilli also react in the same manner as tubercle bacilli. To distinguish them, pour four to six drops of a saturated alcoholic solution of fuchsine in a watch-glass filled with water, dip the cover-glass preparation for six to eight minutes in it, and afterwards treat with $\frac{1}{10}$% of nitric acid in alcohol for twenty seconds, wash in distilled water, and treat with aqueous solution of methyl blue, clear, and examine. If sections, treat with Erlich's fluid for two or three minutes; pass through the nitric alcoholic solution for one-half a minute; then stain with water solution of methyl blue; treat with absolute alcohol, then with oil of cloves; keep in balsam.

Thus only the lepra bacilli are colored, even if the tubercle bacilli are present (Baumgarten's method).

To keep ordinary preparations, balsam dissolved in xylol is good if they are cleared with xylol instead of other oils. Tubercle bacilli after a time lose their color. This is due to the acid retained in the preparation. To avoid such an inconvenience, after treating as usual, dry the preparations first with blotting-paper, then clear with xylol and keep in balsam.

BIOLOGY OF BACTERIA.

During the various stages of their life, bacteria present different forms according to the time at which they are examined; but it is established that one species of bacteria in the adult state and under the same circumstances presents constantly the same forms. Experimentation has also proved that each species has an individuality of its own, capable of reproducing itself under the same circumstances, with its characteristic form and biological properties. The principal forms are micrococci and bacilli. The micrococci, or spherical micro-organisms, are sometimes elliptical, but they never exceed in length twice their diameter. They can be grouped as follows: When alone, they are called micrococci; if in twos, diplococci; if in chains, streptococci; if in irregular bunches, staphilococci; if those bunches are surrounded by a gelatinous envelope, ascococci; when the cocci divide in two directions, forming squares, they are called merismopedia; if in three directions, forming packs or cubes, sarcina.

The elongated or bacillary forms are those whose length is more than twice their diameter. They are of various shapes; some are perfectly cylindrical, others elliptical, lanceolate, fusiform, spiliform. A distinction has been established by some authors between bacillary forms containing spores, and those where spores are absent; calling the first bacilli and the second bacteria. Such a distinction is not generally accepted. If the bacillary forms are curved, they are called spirilla; if corkscrew in shape, spirochæte; some of them have prolongations called flagella. Long and slender bacilli, without any difference between base and apex, are leptothrix; if they have false branchings, cladothrix; large bacilli, containing sulphur granules, are called beggiatoa. All these forms can readily be reduced to two general classes: the spherical, or coccaceæ; and the elongated, or bacillary.

The dimensions of bacteria vary; the unit of measure adopted by bacteriologists is the *micro*, which is the thousandth part of a millimeter, or the 25,000th part of an inch.

Structure.—Bacteria are cellular, formed of an involving membrane, the constituents of which are principally an albuminoid substance called microproteine. Their protoplasm is homogene. In addition to these constituents they may contain sulphur granules (beggiatoa) or amyloid substance.

Nutrition.—Nitrogen, oxygen, carbon, water and some mineral salts are elements indispensable to the life of bacteria. No bacteria grow without water, and most of them are seriously affected, if not destroyed, by a prolonged desiccation. Oxygen is essential to their life. Some need free oxygen, which they take from the air ; others take their oxygen from the media in which they live, and will not grow in the free air ; a third class obtains oxygen either from the air or the media, or from both. The first are called ærobii, the second anærobii, and the third indifferent, or anærobii by election. They also receive their carbon from the media in which they grow. They differentiate themselves from the plants with chlorophyl by not taking carbon from carbonic acid. They obtain nitrogen principally from the albuminoids. The salts required principally by bacteria are the sulphate and phosphate of magnesia and soda, of which they contain from three to six per cent. Bacteria produce such changes in the media in which they grow that they have been divided into three general groups, viz.: *chromogenic* or color-forming bacteria, *zymogenic* or putrefactive bacteria, and *pathogenic* or disease-producing bacteria. This is an empirical division that has no scientific base, but the division, from a practical standpoint, facilitates their study. Bacteria decompose the media in which they live, and give rise to new compounds, which may be a color, a putrid substance, or a virus. The temperature best suited to the life of microbes is between 20° and 40° Centigrade ; those that have been acclimated to the animal body need the temperature of 37° and 40° C. Extreme temperatures are antagonistic to microbes, although they can stand extreme low temperatures. Some lose their vitality at —37° C. and others only at —110° C. High atmospheric pressures destroy them. Light does not affect them ; electricity affects them only when the current is sufficiently intense to cause electrolysis.

Movement.—Most bacteria have movements of their own, whenever the conditions of their nutrition excite them. These movements are either rotatory, oscillatory, or dancing, and are produced either by the contraction of their protoplasm, or by the cilia in those bacteria that have cilia, or by both.

Reproduction.—Bacteria, under favorable conditions of existence, reproduce themselves by transverse scission in one, two, or three directions ; thus their name of schizomycetes (from σχίζειν). When they reach their maturity, their protoplasm contracts, they become trabeculated, and two or more cells are developed. If the media is exhausted, or other conditions interfere, another change takes place, viz., the formation of spores. When the spores are about to be produced, the protoplasm first swells in one or more points of the bacilli, and little by little gets brilliant until the spore appears as a very refulgent point. The remainder of the protoplasm undergoes a process of atrophy, and the spore remains free. Spores are much more resistant than the mother cell, and keep their vitality for a very long time, until they find the necessary conditions for their growth. When this happens, their contents get opaque, the spore swells, its membrane atrophies in one of its poles, and a new bacillus escapes, which begins again the process of reproduction.

Biological Action.—As has been stated, bacteria have been divided into three general classes, according to their influence on the media upon which they grow. The chromogenic and zymogenic are of little importance to the physician.

The third group, or *pathogenic bacteria*, are of the greatest interest to the physician, and their discovery has led to many practical and useful conclusions.

A micro-organism is pathogenic, according to Koch, when it answers to the following conditions :

1. It must be found in the humors or tissues of the animal suffering or dead from the disease.
2. The micro-organism must be cultivated out of the organism (if possible).
3. A pure culture inoculated in an animal must reproduce the disease.
4. The organism ought to be found in the humors or tissues of the artificially diseased animal.

Bacteria that fulfil such conditions are pathogenic. They produce disease whenever introduced into an organism which presents conditions favorable for their development. The second condition must be taken into consideration as much as the existence of the micro-organism itself. Myriads of germs float in the atmosphere and are taken into the blood; nevertheless the amount of disease produced by them is small. This can only be explained on the basis that the exposed organism has the power to resist the invasion of the germs. The tissue cells—principally the surface cells—are constantly at war with bacteria. If the vitality of the surface cells is normal, and the bacterial invasion is not overwhelming, the systemic infection is prevented; but if the vital resistance of the cells is not sufficient to arrest the action of bacteria, disease results. Hence there are two conditions which are necessary for infection, viz.: the specific germ and a low vitality of the organism.

Whether the micro-organism itself, or its products, is the cause of the disturbances observed in disease is still an unsettled question. Since the discovery of ptomaïnes by Gautier, and the experiments made by inoculating them in animals and producing the symptoms of the disease incited by the specific bacteria, scientists are inclined to attribute to the ptomaïnes, and not to the bacteria, the direct action in the development of the disease symptoms. Such facts, however, do not affect the doctrine of parasitism, as many of those opposed to it thought. The microbes generate the poison and are thus the cause of the disease.

Most of the infectious diseases have been studied bacteriologically, and many bacteria have been claimed as pathogenic of certain diseases which further experiments have shown to be innoxious.

Attenuation of Virus.—Bacteria are differently affected in their vital characteristics by the soil in which they grow, or by the atmosphere, temperature, light, electricity, etc. Some lose their primitive properties; their infective power is either changed to a higher degree or rendered almost inert.

Anthrax bacilli ordinarily, at a certain period of their growth, begin the formation of spores; but if kept at a certain temperature,—about 42°

C.,—for some time, spores cease to form, and their virulence is greatly diminished.

The virus of rabies, if passed through a series of rabbits, increases in virulence, while if subjected to a dry atmosphere it gradually loses its virulence.

Cholera bacillus, inoculated from man to pigeons, produces no results, but if inoculated first to guinea-pigs and from them to pigeons the phenomena of cholera are developed.

These facts, and many others stated, have given rise to the theory of attenuation of virus. Upon the basis of this theory, inoculation of the attenuated germs has been practiced with the purpose of producing a mild form of disease, which would render the individual unsusceptible to the more severe form. Practically this theory has been proved to be true, and inoculations of an attenuated virus are now employed as prophylactic or therapeutical measures. Among those can be mentioned vaccine, anthrax, rabies, etc. As to how they act upon the organism to render it refractory to the action of a new invasion of micro-organisms is a question not yet answered.

The discovery of ptomaïnes and their action tends to establish a chemical action of the virus. In such a case the micro-organisms would not be the agents of disease, but their exchange products act as so many poisons. By modifying bacillary vitality, such poisons may somewhat affect the composition or biological action of the bacilli themselves.

This assertion has to a certain extent been proved by inoculating the ptomaïnes of certain micro-organisms (tetanus, cholera, etc.), and producing symptoms similar to those observed in the diseases of which they are supposed to be the cause.

If the conditions of attenuation be carried to a further extent, the micro-organisms not only lose the power of producing disease, but also of existence, and die. Extreme temperatures, the lack of humidity, or the action of certain chemicals, are apt to produce it. In such cases we disinfect or sterilize the cultures. By properly applying those principles, the hygienist can avoid the spreading of infectious diseases.

Pathogenic Bacteria.—The specific character of some bacilli is well determined. The pathological processes which they incite, although essentially the same in character, are modified according to their localization. Some have a predilection for certain organs, but can localize themselves and excite pathological processes in any part of the body where they find proper conditions for their existence.

Acting thus as the cause of disease, they incite similar morbid processes, which receive different names according to the organ in which they are developed.

Let us take, as an example, inflammation of the lungs.

In addition to the micro-organisms usually regarded as the cause of pneumonia, many others can produce inflammation of the lung.

The intensity of the inflammations, or even the ultimate characters of the processes, may differ; but clinically it is impossible to distinguish the diseases produced by Fränkel's diplococcus, Friedländer's bacillus, typhoid bacillus, etc., from each other. Some bacilli destroy the tissues which they penetrate; others provoke hyperplasia of the anatomical elements, and either organize, or perish after a short existence.

Thus, lesions dependent upon a single cause vary. In one case a rapid pyogenic gangrene is established, while in another a neoplasm is developed, which may, as in tuberculosis, undergo coagulation necrosis, and become cheesy. Although many diseases are known to be of an infectious nature, their micro-organisms have not yet been determined. Many germs have been assigned as the causes of certain diseases, and afterwards proved to be harmless saprophytes.

In this sketch only those germs the pathogenic nature of which in man we know has been thoroughly proved will be mentioned.

PATHOGENIC MICROCOCCI.

Staphilococcus Pyogenes Aureus.—Found in pus. Cells grouped in bunches. Yellow gold color of cultures. Melts gelatine.

Staphilococcus Pyogenes Albus.—In pus; similar to above, excepting color of culture, white.

Staphilococcus Pyogenes Citreus.—Same as above, excepting *lemon* yellow color of culture.

Diplococcus Intercellularis Meningitis.—In exudation of cerebro-spinal meningitis. Cocci united in twos. Does not melt gelatine.

Diplococcus Pneumoniæ Lanceolatus.—In croupous pneumonia exudation. Cocci united in twos. Capsulated when in the lung; in cultures it loses the capsule. Does not melt gelatine.

Gonococcus.—In gonorrhœal pus. Cocci in twos, similar to grains of coffee. Cultures grow on agar and sugar or blood serum.

Streptococcus Pyogenes.—Chains of cocci found in phlegmons. Culture does not melt gelatine.

Streptococcus Erysipelatis. — In the lymph spaces of erysipelatous patches. Very fine cocci in chains. Does not melt gelatine. Considered by some authors identical with previous one.

PATHOGENIC BACILLI.

Bacillus Anthracis.—Rods single or in long chains when cultivated; found in the lymph and blood of animals suffering from splenic fever. Cultures melt gelatine.

Bacillus Tuberculosis.—Rods about one third the diameter of a red corpuscle in length; it appears as if formed of several minute buds. From tubercular tissue or sputum; it grows in blood serum, agar, potatoes, carrots, turnips, etc., very slowly; temperature of 37° C. the best. Its coloring reaction differentiates it from all other bacteria.

Bacillus Lepræ.—Found in the tubercles of leprosy. It resembles tubercle bacillus, as shown in colored plate. Grows very slowly in blood serum.

Bacillus Mallei.—Short, slender bacilli, resembling tubercle. Found in the tissues and secretions of animals affected with glanders. Grows as small shining drops on blood serum and potatoes.

Bacillus Œdematis Malignæ.—Rods straight, generally forming chains. Found in cases of malignant œdema in man and animals. It is anærobic, and grows under the gelatine, giving rise to the formation of gas bubbles.

Bacillus Typhosus.—Found in the dejections, mesenteric glands, and spleen of typhoid patients. Short rods with rounded ends; it grows slowly without melting the gelatine. On potatoes it gives a colorless growth.

Bacillus Pneumoniæ.—Short bacilli, single or in chains; capsulated when in the animal organism; without capsule in cultures. Does not melt gelatine, and forms a characteristic nail form; growth white in gelatine tubes.

Bacillus of Rhinoscleroma.—Found in rhinoscleroma tubercles. It resembles the diplococcus pneumoniæ of Fränkel in form and in cultures.

Spirillum Choleræ Asiaticæ.—Short, slender rods curved to resemble a comma, sometimes in S or corkscrew forms; it liquefies gelatine in a characteristic manner. Found in cholera discharges.

Bacillus of Syphilis.—Short rods found in the syphilitic lesions. It has not been cultivated.

Spirochæte Obermaieri.—Long spirilliform rigid threads, found during the exacerbation of relapsing fever. It has not been cultivated, although it has been inoculated in animals from man, producing the disease.

Pathogenic Fungi; Actinomyces.—Found principally in tumors in the jaws of cattle. It is a club-shaped fungus, growing as radii. It produces suppuration in man. Cultures on agar present themselves as small grayish dots.

Achorion Schönleinii.—Fungi of favus, found in patches of favus. Its growth on agar resembles the patches of favus in color.

The micro-organisms just mentioned are those which have been proven to be pathogenic in man as well as in animals. Many others have been described as related to disease. Some of them are peculiar to animals only, and are out of our limits; others, although considered as pathogenic in man, need to be more widely experimented with, in order to include them in the list of causes of disease.

CLASSIFICATION OF BACTERIA.

A scientific classification of bacteria has not yet been given. All of those proposed are defective, as they rest only on empirical basis. It is known that bacteria are plants, that they belong to the Thallophytes without chlorophyl. To this point our knowledge of the place bacteria occupy in the living world is scientific. The subsequent divisions are based only on the form of each germ. As some classification has to be adopted until a truly scientific one is established, bacteriologists generally accept that proposed by Cohn as the one which is more advantageous and nearer to the actual exigency of this new science.

SECTION I.

DISEASES OF THE RESPIRATORY ORGANS.

In considering diseases of the respiratory tract, I shall commence with

DISEASES OF THE NASAL PASSAGES.

The Medical diseases affecting these passages may be included under the general head of *Nasal Catarrhs,* for nasal catarrh appears, either as cause or effect, in all intra-nasal diseases. These diseases may be conveniently considered in the following order :

I. *Acute Coryza.*
II. *Chronic Coryza.*
III. *Hypertrophic Nasal Catarrh.*
IV. *Atrophic Nasal Catarrh.*
V. *Ozæna.*

ACUTE CORYZA.

Acute Coryza is an acute catarrhal inflammation of the nasal mucous membrane characterized by engorgement and tumefaction of the tissues over the turbinated bones. It is commonly called "cold in the head."

Morbid Anatomy.—At the onset of the affection, the anterior portion of the nasal mucous membrane is markedly congested, its normal pink and red hue being replaced by a bright arterial red, while the inner margins of the turbinated tissues assume the blue color of intense venous engorgement. The turbinated tissues are tumefied because of the distension of the venous spaces with blood. Having lost their normal tonicity, they are unable to retract and reduce the turgescence. The distension is, after a time, relieved by an exudation, and the blood gradually resumes its natural flow.

Etiology.—Acute coryza may result from momentary reduction of the surface temperature by chilling, which causes an unequal distribution of blood. As the nasal mucous membrane has less resistance than other mucous surfaces, its inflammation is the result. It may also be occasioned by external irritants, such as tobacco, dust, acrid fumes from chemicals, or by direct injury ; extensive desiccation of the nasal mucous membrane, inspissated mucus, and foreign bodies may also cause it.

Symptoms.—An acute coryza may be ushered in by a slight chill. It is often preceded by a pricking sensation in the turbinated tissues. These structures are rendered extremely sensitive by the intense hyperæmia, so that sneezing is provoked by touching them. The sudden tumefaction accompanying venous engorgement arrests secretion and causes an uncomfortable sensation of dryness ; this is somewhat relieved by a free mucous discharge, which in a short time becomes muco-purulent. The accessory cavities of the nose may become involved, producing headache.

Differential Diagnosis.—A persistent discharge, caused by foreign bodies in the nostrils, may be mistaken for an acute catarrh. Concealed scrofulous and syphilitic ulcers, with co-existing coryza, cause similar symptoms, but will hardly be mistaken for it.

Prognosis.—The nostril generally returns to its normal condition in a few days. Repeated attacks of acute coryza may result in permanent structural changes, and lead to a chronic nasal catarrh. The ear, lachrymal duct, accessory nasal sinuses and cavities may secondarily become involved.

Treatment.—An effort to abort the disease in its incipiency may be made by the administration of ten or fifteen grains of quinine, given alone, or with an equal quantity of bromide of sodium. A full dose of opium at its very onset may restore the vascular equilibrium and abort the affection. Inhalations of ammonia, carbolic acid and alcohol, the vapor of solutions of carbolic acid inhaled through a cone, and fumes of iodine and cologne, inhaled through a sponge, often relieve the engorgement and tension of the mucous membrane by exciting exudation. Depletion of the turbinated tissues by puncturing them with sharp needles, relieves the turgescence and favors their retraction. Spraying of the parts reduces the congestion by chilling the turgid tissues, and gives comfort and hastens recovery by removing acrid discharges.[1] Chloride of sodium, bicarbonate of soda and borax (two grains to the oz. of water) make efficient cleansing solutions.

CHRONIC CORYZA.

(*Chronic Nasal Catarrh.*)

CHRONIC CORYZA is a chronic catarrhal inflammation of the nasal mucous membrane characterized by an excessive and persistent discharge of mucus.

Morbid Anatomy.—There is intense and persistent congestion of the nasal mucous membrane without pronounced tumefaction of the deeper tissues covering the turbinated bones. The abnormal cell production is not accompanied by a marked increase of the sub-mucous cellular tissues. A continuous discharge of watery mucus generally escapes anteriorly. Inspissation of the mucus seldom occurs, as the nares are constantly moist. Excoriation of the lips and integument about the nostril is often caused by the action of the acrid discharges.

Etiology.—Constant watery mucous discharges are sometimes caused by the lodgment of foreign bodies in the nostril. It is frequently observed in young women having a constitutional tendency to catarrh. Cachectic children readily acquire the disease. An irritating atmosphere or abnormally moist or impure air may develop it.

Symptoms.—Annoying paroxyms of sneezing are of frequent though not invariable occurrence. A burning sensation of the anterior nasal orifices is often complained of. Discomfort is sometimes caused by the excoriation resulting from the acrid discharges.

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[1] Delano's, Goodyear's, or Richardson's hand atomizers are best for this purpose.

Differential Diagnosis.—Failure to find a foreign body in the nasal passages will prove that the discharge is not caused by mechanical irritation. Absence of ulceration demonstrates the catarrhal nature of the affection. The chronicity of the complaint and freedom from nasal obstruction distinguish it from acute coryza. The distinctive feature of hypertrophic nasal catarrh, thickening of the intra-nasal tissue, does not occur in chronic coryza. In hypertrophic nasal catarrh the secretions usually flow posteriorly. Concentration and inspissation of the nasal mucus rarely occur in chronic coryza.

Treatment.—The nares should be first thoroughly cleansed, the acrid discharge being best removed by means of syringes and sprays.[1] Weber's nasal douche is convenient for cleansing. Solutions of bicarbonate of soda, boracic acid, and chloride of sodium, five to ten grains to the ounce of water may be employed, the fluid always being warmed before using.

Alkaline solutions may be thrown into the nasal cavities by means of atomizers. After the nares have been thoroughly cleansed, it is well to use astringents. They should be applied in the form of sprays. Of these, ferric alum, five grains to the ounce of water, sulphate of zinc, and tartaric acid, three grains to the ounce of water, are the most efficient. Iodoform, applied by means of Ely's powder-blower, acts as a local anæsthetic, and sometimes affords great relief. Vaseline and benzoated oxide of zinc ointment may be employed to protect the margins of the nostril and heal excoriations. Constitutional vices and tendencies should be corrected by appropriate treatment. The general health should be improved by tonics and an out-of-door life.

HYPERTROPHIC NASAL CATARRH.

(*Chronic Naso-Pharyngeal Catarrh.*)

HYPERTROPHIC NASAL CATARRH is a chronic catarrhal inflammation, characterized by hypertrophy of the turbinated tissues and intra-nasal mucous membrane.

Morbid Anatomy.—The nasal mucous membrane is in a constant state of engorgement, especially the soft turbinated structures, whose cavernous sinuses are so distended as to interfere with free nasal respiration. Hypertrophic changes are most extensive in the loose turbinated tissues; the hypertrophy is due to the general increase of the soft turbinated structures. Permanent dilatation of the turbinated blood cavities takes place, with consequent loss of "turbinated erection." Localized thickening of the tissues

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[1] Dobell's is an efficacious cleansing solution, viz.:

℞ Carbolic acid	gr. j.
Bicarbonate of soda	
Borax	āā gr. v.
Glycerine	ʒ j.
Water	℥ j.
M.	

over the septum, with deviation of this structure, is of common occurrence. The nasal mucus, with epithelial débris, being retarded in its escape by the thickened mucous membrane, inspissates and accumulates in the nostril. The turbinated hypertrophies have a light pink hue; when complicated by acute coryza they vary in color from arterial red to a venous blue.

Etiology.—This disease sometimes results from repeated attacks of acute coryza. The constant irritation caused by a deviated septum, pressing upon the turbinated tissues or nasal mucous membrane, frequently gives rise to it. Distortion of the nasal gutter from deflection of the septum, with consequent accumulation of mucus, always develops it. The growth of gelatinous polypi is encouraged by the abnormal vascularity, and the irritation they produce intensifies the affection. The dusts of cities, factories, and certain occupations act as excitants. Damp localities, irregular living, insufficient clothing, and prolonged exposure to cold and wet, also predispose to it.

Symptoms.—The symptoms of this affection are usually due to turbinated enlargement and accumulated secretions. Thick, tenacious mucus collects in the nostrils, and small masses drop into the pharynx, and thence pass into the larynx, producing irritation, coughing and retching. The nostrils may be permanently or temporarily occluded. Hearing is impaired, either by extension of the catarrhal processes, or collapse of the Eustachian tube from suction induced by nasal stenosis. Obstinate sneezing, headache, suffusion of the eyes, or the ordinary symptoms of cold in the head, may occur as the result of acute exacerbations. The pressure exercised by the thickened tissues sometimes produces persistent neuralgia. Nasal stenosis, combined with laryngeal irritation, may develop symptoms resembling spasmodic asthma.

Differential Diagnosis.—The chronicity of the affection will prevent it from being mistaken for an acute coryza. A careful examination will show whether the disease is simple, or dependent upon the presence of foreign bodies or growths. The mistake of confounding turbinated hypertrophies with gelatinous polypi may be avoided by comparing the gelatinous appearance, mobility, and clear contour of the polypus, with the fleshy hue, firm attachment, and irregular shape of the hypertrophied turbinated tissues.

Prognosis.—The cure of hypertrophic nasal catarrh depends upon the general health, the nature of the structural changes, and the complications. Patients, otherwise in good health, who have acquired the disease from prolonged exposure, may be permanently relieved by a change of climate, after the removal of the nasal obstruction. The part played by a deviated nasal septum, in chronic nasal catarrh, is pronounced, and its excision is as much called for as the removal of a foreign body. Turbinated hypertrophies obstruct nasal drainage and respiration, and provoke and continue the disease which produced them. There are cases in which the simple excision of the offending structures, by removing the cause of the disease and restoring normal respiration, circulation and drainage, effects a cure. When the health is much impaired, with a tendency to profuse catarrhal discharges, and with great distortion of the interior of the nostril, the prognosis is unfavorable. More or less relief, however, can be afforded in all cases.

Treatment.—The treatment of hypertrophic nasal catarrh consists in the restoration of the symmetry of the nasal chambers, and the local cleansing of the part, with the prolonged local use of certain medicinal agents. Caustics and cutting instruments are now employed to remove obstructions and restore drainage and nasal respiration. The exfoliation produced by cauterization with chromic acid and nitrate of silver slowly reduces intranasal hypertrophies. They may be used when the tissues are easily accessible. The platinum knife, made incandescent by the galvanic current, is extensively used to diminish hypertrophied tissues. It acts either by eschar and cicatricial contraction, or by excision. Glacial acetic acid, applied by means of Bosworth's nasal applicator, forms an eschar which can be easily limited, and immediately neutralized by an alkaline spray. Turbinated hypertrophies and gelatinous polypi can be safely removed by the cold wire, with Jarvis' Écraseur. After the removal of the hypertrophied tissues, Dobell's solution, or the bicarbonate or chloride of sodium, may be employed to cleanse the parts; the addition of an antiseptic like carbolic or salicylic acid, or the permanganate of potash, is often of service. Nasal washes are most effective when employed with the post-nasal syringe. Cleansing can be practised by the patient, by means of Warner's nasal douche. Astringents should only be used after thorough cleansing. They are usually applied by means of sprays. Insufflations of boracic acid and iodoform are sometimes advantageously employed. Insufflation should only be practised with powder-blowers working on the Ely principle.

ATROPHIC NASAL CATARRH.

(*Dry Nasal Catarrh.*)

ATROPHIC NASAL CATARRH is a chronic catarrhal inflammation resulting in atrophy and desiccation of the nasal mucous membrane.

Morbid Anatomy.—The nasal cavities are very much enlarged from atrophy of the soft and osseous turbinated structures. The mucous membrane becomes thin, and loses part of its epithelium and some of its mucous glands; and there is an unnatural dryness of the nasal and naso-pharyngeal mucous membrane from deficient glandular secretion. Crusts of decomposing mucus adhere to the turbinated ridges, and occupy the sinuosities of the nostrils.

Etiology.—This disease is local in character. It may be brought on by rapid absorption of moisture from the nasal mucous membrane, hence the frequency of its occurrence in persons continually subjected to the desiccating influence of a dry or dusty atmosphere. Occupations which favor this desiccation, as wood-turning, soldering, milling, cigar-making, develop the affection. It is sometimes associated with a hypertrophic nasal catarrh, and in such cases probably owes its existence to a local modification of the catarrhal processes by which the inflammatory activity is largely expended upon the glandular follicles. Accumulation of the nasal mucus is favored by the destruction of the cilia of the epithelium. The prolonged contracting effect of inspissating nasal mucus promotes progressive atrophy of the soft tissues and bones.

Symptoms.—There is usually entire loss of the sense of smell; a feeling of fullness in the nose is sometimes complained of, the sensation resembling that produced by a foreign body. Neuralgia and dull frontal headache are often present. An annoying fetor, not an invariable occurrence, results from the decomposition of retained nasal secretions. Obstructed nasal respiration from accumulation or lodgment of crusts causes much discomfort. Tenderness and extreme sensitiveness of the mucous membrane may follow the detachment of the incrustations.

Differential Diagnosis.—The glazing and desiccation of the surface demonstrate the existence of dry catarrh; its chronicity will distinguish the affection from the temporary dryness of an acute catarrhal inflammation. Accumulations of crusts, with the co-existing musty stench, are often associated with the atrophic changes. In the advanced stages the unnatural roominess of the nares from general shrinkage of the soft and osseous structures is a distinguishing feature of the affection.

Prognosis.—The disease does not perceptibly shorten life. Beneficial results, or complete restoration of the glandular function, may be obtained by careful stimulation. An artificial supply of moisture relieves the dryness and promotes a return of the normal secretion. Antiseptic cleansing prevents decomposition, and removes a disagreeable feature of the disease. Loss of tissue from atrophy of the soft and solid structures is permanent. Relief of the dryness, nasal obstruction, offensive odor, and general discomfort is always possible.

Treatment.—Thorough cleansing with alkaline solutions should be practised to remove nasal incrustations. Dobell's solution or carbolized water rendered alkaline by the addition of soda or potash, as recommended for hypertrophic nasal catarrh, should be thrown into the nasal cavities by means of the post-nasal syringe. At first it may be necessary to remove the crusts by mechanical means. Cleansing should be practised daily by the patient. Warner's douche may be used for this purpose. Accumulation and inspissation of the nasal mucus are effectually prevented. The painful spots sometimes produced by detachment of crusts are best treated by the application of iodoform. Fluid cosmoline or melted vaseline thrown into the nostril by an atomizer will prevent the reformation of catarrhal crusts. As the sensibility of the mucous membrane is largely obliterated by the atrophic changes, stimulating powder may be employed to provoke secretion, and thus promote the discharge of the accumulated débris. When there is marked general constitutional impairment, change of climate or occupation may be advisable. with the administration of tonics and alteratives.

OZÆNA.

(*Fetid Nasal Catarrh.*)

The term OZÆNA is applied to necrotic and ulcerative processes in the nose and its neighboring cavities, accompanied by fetor due to putrefaction.

Morbid Anatomy.—The thick mucus, dry crusts, and peculiar processes of atrophic catarrh are discoverable in catarrhal ozæna of the nasal cavity; muco-purulent secretions are imprisoned beneath the dried follicles of mucus adhering to the turbinated ridges and interspaces, and then undergo decomposition. When associated with the disease of the accessory cavities, the ethmoidal cells, the sphenoidal sinuses, and the antrum of Highmore, there is continually or intermittently a discharge of glutinous offensive muco-pus. In antral ozæna there usually exists a chronic catarrhal inflammation of the mucous membrane lining the maxillary sinus with a constant formation of muco-purulent matter. The tertiary ulcers of syphilitic ozæna progress rapidly, eroding the soft tissues, denuding the turbinated bones, and inducing necrosis. Their favorite situation is upon the cartilage of the septum. Perforation of the nasal septum by syphilitic ulceration is of common occurrence. In scrofulous ozæna the mucous membrane may be ulcerated, or an accessory cavity may be involved in an asthenic catarrhal inflammation ; when it is catarrhal in character, a peculiar bluish viscid discharge may be discovered oozing from the cavity or sinus involved.

Etiology.—The ozæna of atrophic catarrh is due to the decomposition of secretions which adhere to the mucous membrane. When the ethmoidal cells or sphenoidal sinuses are involved, the offensive odor is due to decomposition of pent-up muco-purulent matter. Antral ozæna may originate in the catarrhal inflammation set up by a tooth projecting into the antrum. Catarrh of the antrum may also result from the extension of inflammation to it from the nares. Syphilitic ozæna may be caused by osteitis, or by fetid catarrh and decomposing débris of ulcers. It may be associated with disease of the pneumatic nasal cavities. Necrosed bone acts as an irritant, provoking and keeping up the fetid discharges. Scrofulous ozæna may originate in an ulcer or in catarrh of the accessory nasal cavities. Ozæna is seldom produced by the decomposition of retained secretions in hypertrophic nasal catarrh.

Symptoms.—In the ozæna of atrophic catarrh, the patient is seldom disturbed by the odor, as he is unable to distinguish odors. When the disease implicates a cavity, the sufferer is usually painfully aware of the ozænous taint. The detachment of incrustations may be followed by slight or alarming hemorrhage. Violent retching or vomiting may be caused by the passage of loosened crusts into the pharynx. The constant effort to free the nostril from incrustations proves extremely wearisome ; a dull headache is often associated with disease of the sinuses, its location being determined by the situation of the cavity. Ulcerative ozæna is seldom accompanied by acute pain. Occasional burning sensations and severe headache are its only painful symptoms.

Differential Diagnosis.—The ozæna of atrophic catarrh is diagnosticated by the rules already given for the diagnosis of that affection. Failure to find a source for ozæna in the nasal cavity, discovery of the peculiar fetid discharge already described as trickling from the superior meatus, the condition being usually unilateral, are evidences of the existence of ozæna of the sphenoidal sinus or ethmoid cells. Antral ozæna is easily recognized by the source of the discharge and the situation of the pain. Non-ulcerative strumous or syphilitic ozæna is usually confined to the accessory nasal cavities. The scrofulous diathesis and history or manifestations of syphilis determine its nature. In the ulcerative ozæna of syphilis the ulcers resemble similar lesions in other parts of the respiratory tract. Their edges are ragged, and the red areola encircling the ulcer fades away into the surrounding normal or congested mucous membrane. There is often a puffiness and infiltration of the tissues about the sore. In the ulcerative ozæna of scrofula the edges of the ulcer are usually smooth and red, occasionally, however, they are pale. The ulcer and areola are clearly defined in the pale anæmic membrane. Pale granulations, or a spongy mucous membrane, may surround the margin of the ulcer. Its chronicity, the enlarged cervical glands, cachexia, and general evidences of strumous diathesis, establish the nature of the ulcer. Perforation of the septum from constant removal of scabs, with consequent abrasion, should not be confounded with the ravages of specific disease.

Prognosis.—The prognosis as regards life is almost always favorable. The conditions determining the course of atrophic nasal catarrh apply also to the ozæna complicating this affection. Scrofulous catarrhal ozæna of the sphenoidal sinuses and ethmoidal cells is dependent upon the general health, and may persist for months, or even years. Syphilitic non-ulcerative ozæna is more amenable to treatment than the scrofulous. Antral ozæna, due to simple catarrhal inflammation or the presence of irritants, responds to proper anti-syphilitic treatment. The cure or relief of syphilitic or scrofulous ulcerative ozæna depends upon the healing of these ulcers. When the disease depends upon the presence of dead or dying bone its removal will often produce a cure.

Treatment.—The treatment recommended for an atrophic nasal catarrh relieves also the ozæna complicating this affection. When the disease is confined to the accessory cavities it is only possible to remove the discharges as they accumulate by means of Dobell's solution, or any of the antiseptic alkaline washes recommended for hypertrophic or atrophic nasal catarrh. The patient can accomplish much in this direction by faithfully using Warner's nasal douche. Careful applications of powder made with an Ely powder-blower prove beneficial. Its odor may be disguised by tincture of musk, tonga bean, or the aromatic essential oils. Antral catarrh due to a dead tooth, or misplaced filling, may require the removal of an upper molar and perforation of the alveolus. Superficial syphilitic ulcers when within reach are readily healed by local applications of chromic acid, or of nitrate of silver fused upon a probe. The same local treatment does not apply to the deep syphilitic ulcers. Iodoform frequently applied gives the best results in the treatment of this form. Local applications should be always preceded by careful cleansing by means of sprays or the syringe. Specific constitutional medication should be combined with the local treatment. It prevents the recurrence of the syphilitic lesions and materially hastens cicatrization. In the deep ulcers rapidly increased doses of iodide of potassium, with or without mercury, are required to facilitate healing. Local stimulation does not, as a rule, benefit scrofulous ulcers, on account of their low vitality; the cleansing and iodoform treatment is preferable. Cod-liver oil emulsions, hypophosphites, iron, nutritious diet, with the usual hygienic observances, constitute the general treatment. The removal of dead or dying bone may hasten healing or complete the cure.

DISEASES OF THE LARYNX.

Laryngeal affections may be primary or secondary; *primary*, when the larynx is first affected, and the affection is local; *secondary*, when the laryngeal disease occurs as a complication, and depends upon some morbid state of the general system. I shall consider them under the following heads:

I. *Acute Catarrhal Laryngitis.*
II. *Chronic Catarrhal Laryngitis.*
III. *Chronic Laryngitis of Phthisis.*
IV. *Chronic Laryngitis of Syphilis.*
V. *Œdema Glottidis.*
VI. *Croupous Laryngitis.*
VII. *Laryngeal Ulcers.*
VIII. *Neuroses of the Larynx.*
IX. *Tumors of the Larynx.*

The most important to the general practitioner in this list are the inflammations.

ACUTE CATARRHAL LARYNGITIS.

ACUTE CATARRHAL LARYNGITIS is an acute inflammation of the mucous membrane of the larynx, which gives only the products of a catarrhal inflammation. It may occur at any age, and be mild or severe in type; the severity varies in proportion to the extent that the submucous areolar tissue of the larynx participates in the morbid processes.

Morbid Anatomy.—The anatomical changes which take place in this affection are characterized by redness, swelling and softening of the laryngeal mucous membrane; its surface, at first dry, the function of the mucous glands being arrested, is soon coated with mucus which contains epithelial and pus-cells. When the deeper tissues are affected, the inflammatory products accumulate beneath the mucous membrane, in its substance and upon its surface, causing tumefaction of the parts, which in this situation is attended with danger. On the other hand, when the inflammatory process is superficial, and all the products are upon the surface of the membrane, there is little danger. At the post-mortem examination there is often less redness and swelling of the laryngeal membrane than were observed during life, owing to the richness of this mucous membrane in elastic tissue. The redness and swelling are due to hyperæmia and infiltration of the mucous membrane. The mucous follicles may be so enlarged as to be recognizable by the naked eye, and each may yield on pressure a drop of muco-pus. The mucus is their natural secretion greatly increased in amount, containing pus-cells and swollen epithelial cells. If the follicle continues to suppurate, it is destroyed, leaving a small depression or ulcer. This inflammation usually runs a rapid course, yet in some cases it becomes chronic. It may produce superficial erosions, it may also be accompanied by ecchymoses of the membrane, and an escape of blood in the secretions; it is then said to be hemorrhagic, and the hemorrhage is due to rupture of capillary vessels. Again, in some cases it is limited to a portion of the larynx, more especially to the epiglottis; then it is usually associated with inflammation of the mouth, fauces and pharynx. The danger in this form of laryngitis is due not only to the submucous inflammation, but also to the spasm of the glottis, which the infiltration causes, partly by reflex action, partly by direct irritation of the adductor muscles of the vocal cords.

Etiology.—Badly nourished cachectic subjects, rather than the strong and healthy, are predisposed to acute catarrhal laryngitis; those constantly exposed to changes of temperature in the open air are less liable to be affected with it than those who are rarely subjected to such exposures. There is also a peculiar vice of constitution that renders certain persons especially liable to catarrhal inflammation, and consequently predisposes them to attacks of catarrhal laryngitis. Among the exciting causes of this affection may be named chilling of the surface by exposure to wet and cold, particularly that of the neck and feet. Mechanical violence to the larynx, inhalations of irritating vapors and acrid liquids may give rise to the most intense laryngeal catarrh.

Laryngitis may also be developed secondarily during the course of the exanthematous fevers, typhus fever, diphtheria, syphilis, and phthisis; not infrequently it is the result of the extension of inflammation from parts adjacent to the larynx, as in tonsillitis, erysipelas, etc. Acute bronchitis is usually attended by a mild form of acute laryngeal catarrh.

Symptoms.—The symptoms that attend the development of acute catarrhal laryngitis vary with the extent and severity of the inflammatory process; it usually comes on insidiously, and a very mild laryngeal catarrh may suddenly become very severe. Usually at first there is soreness of the throat, accompanied by a sense of constriction, or a tickling sensation with a tendency to cough; the larynx is tender on pressure, there is difficulty in swallowing, which becomes more and more marked as the disease progresses; to this is soon added difficulty of breathing. The character of the respiration varies with the seat of the inflammation. If it is confined exclusively to the upper portion of the larynx, as it often is at its onset, the difficulty will be with inspiration only, which will be prolonged and accompanied with stridor; if the lining membrane of the whole larynx is involved, and the calibre of the larynx becomes contracted from œdematous infiltration and spasmodic approximation of the vocal cords, there will be difficulty with both inspiration and expiration, and both will be protracted and wheezing; in severe cases the patient will be unable to lie down. There is a harsh stridulous cough, with (at first) little or no expectoration; if there is any, it is tenacious; later it may become thick, purulent and abundant. The voice is hoarse or is reduced to a whisper. These local symptoms are accompanied by a flushed face, a hot, dry skin, the temperature often rising to 105° F. The pulse is frequent and hard in character. In severe cases, as the disease advances, both acts of respiration become more and more labored, the cough more and more metallic in character, the patient's distress increases, symptoms of imperfect aëration of the blood are developed, the countenance becomes pale and anxious, or livid. During the exacerbations caused by spasm of the laryngeal muscles, suffocation seems imminent; in the intervals the patient becomes drowsy, the vesicular murmur over both lungs is feeble or inaudible, the capillary circulation is obstructed, the lips and nails become blue, a cold perspiration breaks over the surface, the respiratory sounds become gasping in character, and finally delirium and coma close the scene.

As soon as the characteristic symptoms are manifest, a laryngoscopic examination will show the mucous membrane of the larynx to be of a bright red color; if the case is severe, œdema soon appears, the parts being red, swollen and semi-transparent. The tumefaction will be most marked on the ventricular folds, which may entirely conceal from view the vocal bands; this redness and tumefaction may extend into the trachea, or may be confined to the mucous membrane of the larynx and free borders of the epiglottis. Death may occur in a few hours or it may be delayed five or six days; it is caused either by a complete closure of the rima glottidis from tumefaction of the mucous and submucous tissues, or the patient struggles on with obstructed respiration and dies from pulmonary or cerebral congestion and œdema. If death takes place suddenly, it is caused by the combined effects of œdematous swelling and spasm of the glottis. When fatal, its course is usually rapid and severe; when recovery takes place, it is mild in character, and extends over a period of seven or eight days.

Differential Diagnosis.—The affections which may be confounded with acute catarrh of the larynx, are *croupous laryngitis*, *diphtheria*, *œdema of the larynx*, *spasmodic asthma*, *hysterical laryngeal spasm*, and *thoracic aneurism*. In very young children it is often impossible to distinguish between catarrhal laryngitis and croupous laryngitis; but when the laryngoscope can be used, the presence or absence of false membrane decides the question. The history of the attack, the accompanying constitutional symptoms, and a careful laryngoscopic examination, will enable one readily to distinguish between the laryngeal symptoms of acute laryngitis and those of diphtheria, œdema glottidis, or laryngeal spasm; while a physical examination of the thorax determines the existence or the non-existence of spasmodic asthma and thoracic aneurism.

Prognosis.—Age is the most important element in prognosis. In young children this disease is always attended with danger; in adults the danger depends upon the amount of œdema present. The tendency, even in severe cases, is to recovery. In those that tend to a fatal termination, when death is imminent it may be averted by the performance of tracheotomy. There is always danger that the inflammation will extend into the trachea, and lead to a bronchitis or pneumonia.

Treatment.—When active febrile symptoms are present in the early stage of acute catarrhal laryngitis, occurring as a primary disease in a strong, robust adult, venesection to syncope *may* be of service; but if the symptoms which indicate imperfect aëration of the blood are present, or if the laryngitis is a secondary affection, venesection is not only useless but does positive harm. There is no reliable evidence that local depletion by leeches, the application of blisters, or the internal administration of antimony or calomel, has any power to arrest the progress or alleviate any of the distressing symptoms of this disease. It has been claimed that when the disease involves but a small portion of the larynx, the direct application of a solution of nitrate of silver, eighty or even ninety grains to the ounce, to the inflamed portion, by means of a sponge, camel's-hair brush, or with a laryngeal nebulizer, speedily relieves the dyspnœa and ameliorates the general symptoms. Few, however, have the requisite skill and experience in topical medication of the larynx to make such applications efficaciously, or at least to accomplish what is claimed for them. Most practitioners would do much more harm than good were they to attempt to make such applications. Externally, both hot and cold applications are recommended. The rubber or metallic coil is the most efficient means of employing either, and should be applied upon the earliest manifestation of the disease. Cold applications, when employed, are most efficacious at the onset of the inflammation, but my experience leads me to prefer hot applications throughout its entire course.

For the successful management of this disease, a warm, moist, and uniform temperature is essential; the temperature of the apartment should never be allowed to fall below 76° F. When the submucous areolar tissue is either not at all or only slightly involved, vapor inhalations unquestionably give the greatest relief, and have greater power in arresting the inflammatory process than all other local measures; they should be commenced early and perseveringly continued. Aconite, in ½ ♏ doses, repeated every half hour until dryness of the throat is produced, is often of value in adults as well as with children.

When the temperature reaches 103° F. at the onset, one or two full doses of quinine will be of service. If the inflammatory process is not arrested by the combined action of these remedies, œdema is almost sure to follow, and the parts should be freely scarified; in the adult this may readily be done with a laryngeal lancet by the aid of the laryngoscope. Should this treatment fail or be impossible, and should the dyspnœa be of a threatening character, and the signs of imperfect aëration of the blood be well marked, tracheotomy must not be delayed. Intubation by O'Dwyer's method, or even the passage of a soft catheter through the larynx, may frequently be employed in place of the more serious operation with equally good results. Many lives have been lost by too long delaying operation. For the successful management of acute laryngitis, rest to the larynx is all important

SIMPLE CHRONIC CATARRHAL LARYNGITIS.

This is essentially a chronic inflammation of the lining membrane of the larynx, the submucous tissue being slightly involved. When once established its tendency is to remain stationary. Like the acute, it may be general or partial.

Morbid Anatomy.—When the disease is fully developed the mucous surface of the larnynx is always more or less coated with mucus or pus. Its tissue is dark colored, sometimes of a grayish red or bluish hue, owing to previous ecchymosis; it may be either softer or firmer than natural; the mucous glands are large and prominent, the submucous tissue is thickened, and the vocal bands may either become relaxed or stiffened, and hence vibrate less than in health. Paresis of certain laryngeal muscles may result from the thickening and infiltration. When the trachea is involved, the portion of the mucous membrane covering its rings is reddened, while the intermediate portions are of a dark gray color.

Etiology.—This affection may occur as a primary disease, or as a sequela of a mild form of acute laryngitis; not infrequently it is the result of the extension of a pharyngeal inflammation in those who constantly use the voice in public speaking or singing. It constitutes the chief morbid condition in what is termed "clergyman's sore-throat." It is frequently secondary to a chronic nasal catarrh—the catarrhal process extending from the nasal passages. The constant inhalation of irritating particles may be a cause of chronic laryngitis; but it most frequently occurs as an accompaniment of other affections, as syphilis, pulmonary phthisis, and laryngeal morbid growths. When it occurs, as it frequently does, in phthisical or syphilitic subjects, it is described as laryngeal phthisis, and syphilitic laryngitis. The variety which is the result of the extension of a follicular pharyngitis, is sometimes separately described as chronic glandular laryngitis, but it does not differ from simple laryngeal catarrh, except that in it the minute racemose glands are principally affected. The sudden development of the larynx in males which takes place at puberty is often attended by a mild form of chronic laryngeal catarrh. In every variety of chronic bronchitis, especially that occurring in old age, there is more or less chronic laryngeal catarrh,—in many instances the laryngeal catarrh is secondary to the bronchitis.

Symptoms.—The symptoms of chronic catarrhal laryngitis are altogether local in character. The most characteristic are the changes which occur in the voice; in some it is hoarse and husky, in other cases the patient is only able to speak in a husky whisper. The voice is most often reduced to a whisper in the dry variety of laryngeal catarrh. Accompanying or preceding the vocal changes, there is a hoarse, stridulous cough, with more or less abundant muco-purulent, or purulent expectoration; not infrequently the expectoration is streaked with blood, and of a fetid odor. Inspiration and expiration are more or less impeded, and are often accompanied by a whistling or stridulous sound, and moist râles can usually be heard over the larynx. There is often soreness and tenderness of the laryngeal cartilages when pressed laterally or backward against the spine. In some cases, the act of swallowing fluids or solids is attended with no inconvenience; in other cases it excites spasm of the glottis, and thus occasions fits of distressing dyspnœa. If constitutional symptoms exist, they are due to sympathetic irritation, and are in no way characteristic of the disease. The principal danger is from chronic laryngeal œdema, but this is of exceedingly rare occurrence. The laryngoscopic appearances correspond to those changes already described under the head of morbid anatomy of the disease. The laryngeal mucous membrane is of a deep red color, verging on purple. The change in color is most marked over the vocal cords and arytenoid cartilages; sometimes the larynx has the appearance of being very much dilated, at other times it is apparently contracted. The mucous surface may be covered over with a muco-purulent secretion, or may present a dry and shining appearance; the enlarged orifices of the glands may be seen as pale specks on the congested membrane, or as red circles studding a pale membrane. In addition to these more common appearances, more or less extensive thickening and papillary excrescences are sometimes visible.

Differential Diagnosis.—The diagnosis of chronic laryngitis is readily made; the changes in the voice at once direct the attention to the larynx, and the laryngoscope will determine the nature, extent and exact seat of the disease. From the general and local symptoms, chronic laryngitis may be confounded with laryngeal growths and nervous affections of the larynx, but a careful laryngoscopical examination will correct any error. Phthisical and syphilitic laryngitis may be distinguished from simple catarrhal laryngitis by the presence of ulcers, by a careful physical examination of the lungs, and by the history of the patient. If both phthisis and syphilis can be excluded, however extensive the disease, it must be regarded as of primary origin.

Prognosis.—The prognosis in this affection depends on its pathological associations. All forms of simple chronic laryngitis, unless complicated, may be recovered from; at least, it rarely, if ever, leads to a fatal termination. It is always difficult, however, and sometimes impossible to cure chronic laryngitis in old people.

Treatment.—The most efficient agents in the treatment of this variety of laryngitis are local applications, within the larynx, to the parts affected. These topical applications may be made at the time of a laryngoscopical examination, either by means of a sponge or camel's-hair brush carried within the larynx, by the inhalation of vapor impregnated with some volatile substance, or in the form of nebulized liquids. The most certain and most satisfactory method is the use of atomized liquids, applied by means of the laryngeal spray—the mechanical chilling produced by the spray adds to the efficiency of the astringent solutions. Sponges and brushes are apt to irritate, and produce hyperæmia of the larynx. When local applications are to be used for a long time, mild astringent solutions are preferred. A solution of alum, perchloride of iron, tannin, or the sulphate of zinc, from one to twenty grains to the ounce of water, may be used. If the applications are made directly to the diseased tissues, and sufficiently often, it matters very little what astringent solution is used. When the laryngeal secretion is excessive, the local application of turpentine sometimes does good. For steam inhalations, a few drops of oil of creosote, oil of pine, or oil of juniper, added to half a pint of water at a temperature of 150° F., may be employed. Neither the steam nor spray inhalations should be continued more than five minutes at a time; they may be repeated three or four times during the twenty-four hours. A solution of carbolic acid (two grains to an ounce of water), either as a spray or as a steam inhalation, may be used with benefit in cases where the laryngeal secretion has a fetid odor. In such cases the nares should be carefully examined, and the passage of offensive nasal discharges into the larynx should be prevented. In addition to the local treatment, the patient must be removed from all sources of laryngeal irritation. The vocal organs must have perfect rest; the patient, if possible, should change climate, removing to such as he finds best suited to his individual case; as a rule, a warm, dry atmosphere best agrees with this class of patients.

The constitutional treatment of each patient will be governed by his general condition, and the pathological relations of the laryngitis. The general hygiene of the patient should always be carefully regulated.

CHRONIC CATARRH OF THE LARYNX IN PULMONARY TUBERCULOSIS.

Simple catarrhal laryngitis associated with pulmonary phthisis is often modified by the phthisical cachexia. When the inflamed surface becomes the seat of tubercular development, and the secretions are found to contain bacilli, it is called *laryngeal* tuberculosis, or phthisis.

Morbid Anatomy.—The laryngeal structure, sympathizing with the general impairment of nutrition, is pale in the earlier stage of the affection. As the inflammatory and tissue changes advance, the mucous membrane becomes irregularly vascular and of a peculiar ashy-red hue, but its tissues become progressively thickened. A pear-shaped enlargement over the mucous membrane which covers the arytenoid cartilage is a characteristic feature of pronounced laryngeal phthisis. The epiglottis in the catarrhal stage of the affection is often enlarged and shaped like a turban. The submucous tissues become infiltrated with small cells. These cells show a tendency to cluster and form cheesy centres, thus paving the way to the development of tubercular ulcers. Œdema from serous transudation within the loose mucous tissue often occurs. Later, localized abrasions occur where the tissues are in contact, or at points where the friable tissues are in constant motion. The ulcerative stage may become pronounced, and superficial sores of an ashy-gray hue occur at points most favorable for their development. These ulcers may enlarge by coalescence or individual extension until a large portion of the ventricular bands, vocal cords, and epiglottis becomes involved. They often advance in depth, descending the laryngeal skeleton and eroding the cartilages; the roughened structures are constantly covered with the tenacious muco-purulent discharges which come from the lungs, or are excited by the intense local irritation.

Etiology.—The phthisical peculiarity of the inflammation receives its impress from the development of tubercle. Constant hawking or coughing, provoked by the passage of the irritating discharges over the laryngeal structure, and the constant movement of the parts, favor the development of the laryngeal complication. The general cachexia, with attendant impaired nutrition, modifies the nature and course of the catarrhal processes, and favors the development of tubercle.

Symptoms.—The early subjective symptoms are generally those of a modified chronic laryngitis, associated with constitutional manifestations of pulmonary phthisis. The respiration may be hurried or embarrassed. Hoarseness or complete aphonia is produced by the structural changes, and the irritating discharges excite distressing paroxysms of coughing. An examination of the laryngeal secretions will reveal the presence of tubercle bacilli. Painful deglutition is induced by counter-pressure of the inflamed or thickened abraded mucous membrane, the disturbance of elevated ulcers, or the friction of food. A harassing nervous cough exists in the ulcerative stage of the affection. The patient hesitates to take food on account of the pain caused by the passage of the bolus. This hastens the general emaciation.

Differential Diagnosis.—The peculiar pyriform thickening over the arytenoid prominence distinguishes the hypertrophic from simple chronic catarrhal laryngitis. The ashy-gray hue, irregular vascularity, and gray infiltrated vocal cords can readily be contrasted with the bright inflammatory redness of the laryngeal structures seen in simple catarrhal laryngitis. The non-existence of ulcers in simple chronic laryngitis will also aid in differentiating between the two affections. The contour of the syphilitic ulcer and its bright red areola and irregular margin are not easily mistaken for the pale and peculiar thickened mucous membrane of laryngeal phthisis, and its irregular ashy-gray ulcers. The syphilitic ulcer, moreover, advances rapidly, but usually painlessly; the phthisical ulcer progresses slowly, and is accompanied with much pain. The presence of tubercle bacilli in the secretion which covers the laryngeal surface establishes the diagnosis.

Prognosis.—Healing of the ulcers is usually considered the cure of a laryngeal phthisis. This, however, will depend upon the site and size of the lesions. When superficial ulcers upon the ventricular bands, vocal cords, or arytenoid prominences are seen early, they are sometimes curable. Ulcerations of the epiglottis are incurable, and often the indirect cause of death. Erosions of the cartilages are always exceedingly troublesome, and generally lead to a fatal issue; the extent of the pulmonary disease and the general cachexia modify the prognosis in laryngeal phthisis. Extensive hypertrophy of the laryngeal mucous membrane does not seriously influence the prognosis. Judicious local and general treatment will afford relief in every case, and frequently postpone the fatal issue.

Treatment.—Treatment should be both constitutional and local. The general treatment is determined by the laryngeal symptoms, the condition of the patient, and the co-existing pulmonary disease. Cough may be quieted by the internal administration of bromide of sodium or solution of morphia (U. S. P.). Its relief removes a source of irritation which is constantly acting on the inflamed or ulcerated larynx, and by affording rest favors repair or retards the advance of the local disease. It also soothes the disturbed nervous system, and allows the patient to gain strength by undisturbed sleep. In connection with the constitutional and hygienic treatment of the accompanying pulmonary disease, great relief can be afforded and life prolonged by the following local management of the laryngeal ulcers. The surface of a phthisical ulcer should be thoroughly cleansed by means of an alkaline spray;—a one per cent. solution of the phosphate, or carbonate of soda, with glycerine and carbolic acid or thymol, is most serviceable. Finely pulverized iodoform should then be blown upon the ulcerated surfaces. When there is much pain Magendie's solution, or, better, a 4% solution of cocaine, may be sprayed upon the ulcerated or inflamed tissues. The laryngeal sponge or brush is always contraindicated. Tracheotomy to rest the larynx and lessen the suffering is sometimes justifiable. It is of doubtful curative value, and may hasten the impending crisis by accelerating the progress of the pulmonary lesions.

CHRONIC CATARRHAL LARYNGITIS OF SYPHILIS.

The course, symptoms, and appearance of chronic catarrhal laryngitis may be modified by constitutional syphilis. Constitutional infection may cause a syphilitic non-ulcerative laryngitis, as a secondary or tertiary manifestation. In its advanced stage it is often complicated by ulceration. The vascularity of the mucous surface does not show the rapid variations seen in ordinary catarrhal laryngitis; it is far more persistent. The arterial red of catarrhal laryngitis is replaced by a deep red or even a livid hue. The discomfort which it causes does not, as in simple laryngitis, correspond in severity with the intensity of the congestion. A syphilitic history and the disappearance of local manifestations under appropriate internal medication aid in determining its character. The treatment consists in the application of tincture of iodine by means of sprays, brushes, or vaporizers, with or without the insufflation of iodoform; simple applications of iodoform by means of Ely's laryngeal powder-blower are often markedly beneficial. The parts should be thoroughly cleaned with alkaline sprays before each application. In all cases local and constitutional treatment should be combined. A rapid increase in the size of the dose of iodide of potassium is often required to effect the desired changes.

ŒDEMA GLOTTIDIS.

Œdema Glottidis is a term which has been used to indicate the occurrence of a dropsical effusion or inflammatory exudation into the areolar tissue beneath the laryngeal membrane above the vocal bands. Strictly speaking, it is not œdema of the glottis, but of the upper portion of the larynx. Its gravity, and the necessity of its prompt relief, make the early recognition of its existence, and of the pathological conditions which lead to its occurrence and attend its development, very important.

Morbid Anatomy.—The effusion, which is almost always serous, takes place in the loose cellular tissue beneath the mucous membrane of the upper part of the larynx, principally in the ary-epiglottic folds, and at the base of the epiglottis; as a consequence, these parts become prominent and the epiglottis swollen. On either side there may be a tumor an inch or more in diameter, projecting into the cavity of the larynx and pharynx; in some cases these tumors touch each other, completely occluding the laryngeal cavity. The mucous membrane may be either red or pale. On pricking the tumors, a clear, or turbid, or even a purulent fluid may escape; after which the parts previously distended collapse, and the mucous membrane is left wrinkled and folded. The effusion may occur wholly or principally on one side. Not infrequently after death, owing to the disappearance of the effusion, the wrinkled condition of the mucous membrane is all that is found, or, at least, there is much less effusion than might have been expected from the appearance of the parts during life.

Etiology.—Œdema of the glottis rarely if ever occurs as a primary affection, but is secondary to, or is a complication of, some local laryngeal disease or constitutional disorder. Any inflammatory affection of the larynx or of the adjacent tissues may give rise to it, such as acute laryngitis (especially that due to local irritation), erysipelas of the neck, deep-seated cervical abscesses, and acute tonsillitis. It occasionally occurs as a complication of the laryngeal ulceration of typhus and typhoid fever, small-pox and scarlatina. Sometimes it is the immediate cause of death in the general dropsy of Bright's disease, and in the venous obstruction which attends some forms of cardiac disease and thoracic aneurism.

Symptoms.—The prominent symptom of this affection is dyspnœa, and the difficulty in breathing is mainly confined to inspiration. No difficulty in swallowing is experienced, nor is there tenderness on pressure over the larynx. Fever and the other constitutional symptoms which attend acute laryngitis are absent. It is accompanied by paroxysms of extreme dyspnœa—suffocative breathing being usually the first indication of its occurrence; there is also an uneasy sensation in the region of the larynx, and a constant inclination on the part of the patient to rid the upper part of the throat of some supposed secretion. If the index finger be carried below the epiglottis, œdematous tumors may be distinctly felt. Hoarseness or huskiness of voice may not be present, unless laryngitis coexist. The laryngoscope reveals two tense, smooth, rounded swellings immediately behind the epiglottis; these swellings, after meeting in the centre, with a sulcus between them, appear oval. The œdema is usually most marked at the ventricular folds, which explains the nature of the urgent dyspnœa. Whenever, during the progress of any disease in which œdema glottidis is liable to occur, there is the slightest difficulty in respiration, the difficulty being limited to inspiration, the possible occurrence of this complication should be remembered.

Differential Diagnosis.—The circumstances under which œdema glottidis is developed, the suddenness of its occurrence, the peculiar character of the respiration, indicating obstruction at the upper portion of the larynx, and the absence of febrile excitement, constitute a group of symptoms almost pathognomonic. When to these is added the presence of œdematous tumors at the upper portion of the larynx, the differential diagnosis between it and other laryngeal affections is easily made.

Prognosis.—The tendency of this affection is to speedily destroy life, but in most instances death may be averted by prompt and efficient surgical interference.

Treatment.—There is no time to be lost in fruitless medication. In extreme cases, laryngotomy or tracheotomy must be performed early. It is recommended, by some, to scarify the edges of the œdematous epiglottis or ventricular bands and ary-epiglottic folds, so as to give free exit to the effused fluid before resorting to these operations. This scarification can rarely be accomplished, except by an experienced hand, and, in extreme cases, the delay and danger which attend such an attempt are hardly justifiable.

ACUTE CROUPOUS LARYNGITIS.

(*Membranous Croup.*)

Croupous inflammation of the larynx differs from catarrhal in the nature of its inflammatory products. The inflammatory process may be limited to the larynx, or it may extend into the trachea; and broncho-tracheitis so frequently accompanies it that the disease has received the name of *cynanche trachealis*, but in all instances the tracheal inflammation is secondary to the laryngeal. Croupous laryngitis is a local inflammation.

Morbid Anatomy.—When fully developed, there exists over a varying extent of the mucous membrane a whitish, or yellowish-white, fibrinous layer, often spotted here and there with dots and lines. This membranous exudation may be limited to a few patches, or form a cylinder which may extend into the trachea and bronchi. At one time it is firm in its consistence, and tenaciously adheres to the subjacent membrane, while at another it is soft and easily separated from it. Its thickness varies; sometimes it is scarcely perceptible, at others it may be a line or more in thickness. Its surface is smooth, it adheres firmly to the vocal cords and the upper part of the epiglottis. After the membrane is once formed, it may be cast off in the form of a cylinder, in bands, or shreds. Its separation is effected by the secretion of the follicles which have been obstructed, as well as by a serous exudation from the previously inflamed membrane. It may break up into threads, and be expectorated as such, or it may undergo a granular, fatty, or, more rarely, a mucous degeneration, and so become a fluid resembling mucus.

In its earlier stages, the mucous membrane of the larynx presents the same appearance as in catarrhal inflammation. When the false membrane has formed, it takes the place of the epithelium, and is situated on the homogeneous boundary layer of the mucous membrane which exists in the greater part of the larynx. The tissue is pale, except when it is dotted with ecchymoses, which correspond to similar spots in the membranous exudation. The laryngeal membrane is somewhat swollen, and moister than normal. Generally, the submucous tissue is only slightly involved. A microscopic examination of this membranous exudation shows it to consist of a homogeneous, shining net-work, in the meshes of which are inclosed pus-cells, rarely epithelium; it may be made up of alternate layers of fibrin and cells. As the membranous exudation is cast off, the epithelium is quickly replaced, and the laryngeal membrane returns to its normal condition. Sometimes, after the membrane is thrown off, it is reproduced. The submucous tissue is more liable to be involved in adults than in children. Very frequently membranous pharyngitis precedes and is associated with croupous laryngitis. Pulmonary congestion, œdema, atelectasis, emphysema, and lobular pneumonia not infrequently occur as complications in the course of this disease.

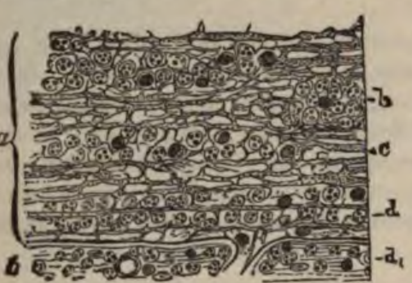

FIG. 12.

Croupous Membrane from the Trachea.

a, Section through the false membrane.
b, Upper layer of mucous membrane, infiltrated with pus-corpuscles (d_1).
c, Filaments and granules of fibrin.
d, Pus-corpuscles. After Zeigler. × 250.

Etiology.—Age is the most prominent predisposing cause. It is rare in adults (except from traumatic causes), or in very young infants. The time of its greatest liability is between the period of dentition and puberty. There is no evidence that its development is due to any specific atmospheric poison. Following Bretonneau, many authors have regarded croup as depending upon the same specific poison as diphtheria, but representing a milder grade of infection. I am unable to see that either pathological researches or clinical facts are sufficient to establish this position. Exposure to cold and moisture, with sudden alterations of temperature, are among its most frequent exciting causes. It occurs more frequently during the winter and spring months. Delicate, weakly, and ill-nourished children, rather than the strong and healthy, are liable to it. It not infrequently follows the sudden disappearance of eczematous eruptions on the head and face; occasionally it follows measles, scarlatina, and variola, and sometimes complicates diphtheria; and when diphtheria is prevailing it is often difficult to draw the line between them.

Symptoms.—Acute croupous laryngitis usually begins with the ordinary symptoms of a simple cold; at its commencement there is nothing to distinguish it from an ordinary catarrh. If the throat is examined the whole visible mucous membrane will be found red and tumefied. Usually the first symptom that attracts attention is a slight hoarseness; a little later the respiration becomes difficult, the expiration noisy, and it is accompanied by a high-pitched stridulous cough. The inspiration that immediately follows the cough is accompanied by a loud crowing noise. Although there is no pain or tenderness in handling the larnyx, there is some difficulty in swallowing, and the child frequently puts its hand to its throat as if to remove some obstruction. With the first croupy paroxysm, however slight, the pulse is accelerated and becomes full and hard; there is increased heat and redness of the surface, especially of the face, with injection of the conjunctival vessels; the axillary temperature may range from 102° F. to 104° F. These febrile symptoms somewhat subside as the paroxysm passes off, to return, however, with greater intensity on the return of the next paroxysm. At the commencement of the attack the paroxysms of dyspnœa are more frequent and severe at night than during the day.

As the disease advances the voice is entirely lost; the patient speaks and cries in a whisper; the cough becomes more and more stridulous in character, without expectoration; the head is thrown back; the respiration grows more and more difficult, and with each inspiration there is contraction of the lower part of the chest and sinking in of the soft parts above the clavicles. The vesicular murmur over both lungs is feeble or inaudible; with every inspiration the epigastrium, instead of projecting, is strongly depressed, and the outward movement of the lower ribs is arrested. Every muscle that can aid in expanding the chest is brought into violent action. During these laborious efforts at inspiration the nostrils are dilated. As the laryngeal obstruction increases, the paroxysms of dyspnœa become more urgent and without remission; there is a restless tossing of the limbs, and the greatest terror is depicted on the face, which, at one time, is pale, at another livid; the pulse becomes rapid and feeble; the temperature falls sometimes below the normal and the extremities become cold. Gradually, as the blood becomes imperfectly aërated, the patient becomes drowsy, at times rousing up and gasping for air, and springing from one place to another to find relief; the lips and nails become blue, the respiration shorter and shorter, until at last, after a violent paroxysm of dyspnœa, the patient becomes unconscious and quietly ceases to breathe. The disease always attains its height by the end of the third day; death may occur within forty-eight hours after its commencement; its whole duration rarely exceeds five days.

In accordance with its symptomatology, croupous laryngitis may be divided into three stages: a precursory or *catarrhal* stage, a stage of *development*, and a *suffocative* stage, or stage of collapse. The most important fact connected with its clinical history is, that in a large proportion of cases before the urgent symptoms come on, the membranous exudation can be seen on the tonsils. In most cases the membrane is first formed on the tonsils or in their immediate vicinity. As the membrane extends into the larynx there is loss of voice, a stridulous cough, difficult breathing, and the face is alternately flushed and pale. For a day or two, while the membrane is extending and becoming thicker, the patient remains in about the same condition, gradually growing weaker, the capillary circulation on the surface becoming more and more imperfect, the respiration more and more labored, the paroxysms of dyspnœa more and more frequent and severe, until there is little hope of recovery. Sometimes all the urgent symptoms are suddenly relieved, the patient coughs, and a stringy matter is expectorated, he struggles for a moment in a violent paroxysm of dyspnœa, and a perfect membranous cast of the larynx, and perhaps of the trachea, and larger bronchi is expectorated. Now he passes into a quiet sleep, and recovery seems certain; but still there is great danger—from the formation of a new membrane; from the extension of the inflammation into the minute bronchial tubes, giving rise to capillary bronchitis and pneumonia; and from the exhaustion that has occurred before the membrane was thrown off. A laryngoscopic examination is rarely possible in this class of patients.

Differential Diagnosis.—The two affections of the larynx which are most liable to be confounded with this form of laryngitis, are *simple catarrh* of the larynx, called spasmodic or pseudo-croup, occurring in nervous subjects, and purely *spasmodic affections* of the larynx. In both, the laryngeal spasms give rise to croupy symptoms. In spasmodic croup or simple catarrh of the larynx, the croupous phenomena come on suddenly, the attack usually occurs at night, it is not preceded or accompanied by active febrile symptoms, there is no complete loss of voice, and there is absence of membranous exudation on the tonsils and epiglottis. All of these conditions are important diagnostic features of croupous laryngitis. Within twenty-four hours after the commencement of an attack of catarrhal croup, auscultation of the chest furnishes signs of incipient bronchial catarrh. Spasm of the glottis, which may give rise to croupy symptoms, is excited in infancy by a variety of causes. Among these are dental irritation, gastric irritation, enlargement of the thymus gland, giving rise to what is called thymic asthma, and undue excitability of the nervous system, the result of hereditary predisposition. These laryngeal spasms may be recognized by the suddenness and violence of the attack, by the absence of the catarrhal and febrile symptoms, by the absence of alteration of the voice, and by the speedy and complete relief which immediately follows the spasm. Diphtheria involving the larynx, sometimes mistaken for croup, may be distinguished from it by the following characteristics: *first*, either diphtheria is epidemic, or there is a history of contagion; *second*, the development of the throat symptoms is preceded or accompanied by constitutional disturbances; *third*, the glands at the angle of the jaws are usually enlarged, and the laryngeal symptoms are at first not urgent; *fourth*, the pharynx presents the characteristic diphtheritic appearance before any laryngeal symptoms are present.

Prognosis.—There are no data from which to estimate the ratio of mortality in this disease; unquestionably it is one of the most fatal diseases of childhood. When the diagnosis is based upon the presence of the membranous exudation on the tonsils and epiglottis, recovery seldom occurs. The signs of a favorable termination are, diminution in the frequency and severity of the paroxysms of dyspnœa, with less distress in breathing during intervals, a gradual return of the voice, and a moist sound with the cough. If, on the other hand, the paroxysms of dyspnœa become more frequent and violent, the restlessness and dyspnœa increase during the intervals, and the cough is less powerful and more stridulous, the blueness of the lips and nose more marked, and the patient becomes more and more drowsy, recovery is scarcely possible. The younger the patient, the greater the danger. In fatal cases, the duration of the disease is from three to seven days. If recovery takes place, it is slow, weeks often elapsing before the voice returns, during all of which time the patient is subject to violent paroxysms of dyspnœa.

Treatment.—I do not purpose to discuss the merits of the various plans of treatment which have been proposed for the management of membranous croup, for under every plan the disease has proved fatal in the majority of cases. Simple catarrh of the larynx is so liable to be mistaken for croupous, that it is difficult to estimate the real value of the different remedial agents which have been claimed to have a controlling power over it. Statements that certain plans followed, or agents employed, have been successful in the majority of cases arouse distrust of the diagnosis; the existence of croup should be asserted only upon positive evidence of the presence of the membranous exudation. With the written history of membranous croup before us there is no evidence that calomel, blood-letting, or antimony has any power either to arrest the progress of the inflammatory action or to prevent the membranous exudation. In the treatment of this affection it is of the first importance that the patient should be placed in a large, well-ventilated apartment, the temperature of which should be kept uniformly at 75° F. to 80° F., and the air rendered moist by steam. In the case of children, a tent may be made over the bed by means of blankets, into which is made to pass a constant current of steam from a kettle containing boiling water. As soon as evidence of imperfect oxygenation appears, a continuous stream of oxygen gas should be carried into the tent, or arrangements should be made so that it will be constantly inhaled by the patient; sometimes, in addition to these means, lime vapor, produced by slacking large quantities of quicklime in the room, will be found of service. During the whole course of treatment external application of moist heat should be constantly maintained. This is best accomplished by a metal coil bound upon a thin flat sponge. Whenever there are indications that loosened portions of membrane act as causes of dyspnœa, an emetic may be administered; the sulphate of zinc acts most promptly and efficaciously. The frequent administration of emetics should be avoided, on account of their depressing influence. Topical applications are not to be resorted to in its treatment, as they intensify rather than relieve the laryngeal spasm, which plays so important a part in producing the paroxysms of dyspnœa, and there is no evidence that they have any control over the inflammatory process. It is all-important that this class of patients, from the onset of the disease, should receive a most nutritious diet; and as failure of the vital powers becomes apparent, stimulants may be freely administered upon the same principles as govern all adynamic diseases. As regards internal medication, I have little confidence in any of the so-called specifics. After the formation of the membranous exudation, the vapor inhalation and the oxygen gas are the only means which afford any hope that the patient can be safely carried through the disease. In regard to the propriety of tracheotomy the opinion of the profession is divided. The statistics of this operation in this disease are not to be relied upon. The only question to settle is, Has one life been saved by it? If this can be answered affirmatively, the operation is justifiable. It never should be resorted to with a promise even of relief; if there are evidences that the membranous formation has reached the bronchi, and even when the membrane has formed in the trachea, temporary relief from the dyspnœa is all that can be promised. The operation, to be successful, must be performed early, and not be delayed, as it usually is, until the patient is beyond hope of recovery. Its success also undoubtedly depends much upon the manner of its performance and the subsequent management of the case. As in other obstructive conditions of the larynx, intubation may often be substituted with success, unless the fauces and posterior nares are implicated and filled with the exudation.

ULCERS OF THE LARYNX.

The remaining laryngeal affections come more directly within the province of the specialist, and only the most prominent points in their history will be considered. The different forms of laryngeal ulcerations are included under the following heads: the catarrhal, the follicular, variolous, typhous, phthisical, and syphilitic. The most common forms are those occurring in phthisis and syphilis.

Morbid Anatomy.—*Catarrhal* ulcers are usually superficial, and at first may be either rounded or oval; afterwards, as the loss of substance becomes more extensive, they coalesce and have an irregular outline. The *follicular* ulcer, as a rule, is superficial, with a limited area of extension. It sometimes constitutes a serious complication of laryngeal phthisis. Though of common occurrence in the pharyngeal and faucial cavities, it is seldom seen in the larynx. These ulcers may be situated upon any portion of the laryngeal membrane; when they are located upon the anterior or posterior ends of the vocal bands, they have a tendency to spread lengthwise.

Variolous Ulcers are the result of small-pox pustules on the laryngeal membrane. They commence by the formation of soft, non-umbilicated pustules, which after a little rupture and form a rounded ulcer, which readily heals.

Typhous Ulcers are generally of large size, and deep, penetrating through the mucous membrane, and sometimes involving the cartilages. The edges of these ulcers are everted, and of a dark purple color; their commonest seat is the posterior wall of the larynx, and the edges of the epiglottis.

Phthisical Ulcers may be superficial or deep; the most frequent seat is the inter-arytenoid commissure. They are complications of laryngeal phthisis. The deficient vascularity of the pale and thickened arytenoids favors the occurrence of these ulcers. They may have their inception in an inflammation of the follicles of the epiglottis or neighboring tissue, and spread by coalescence; sometimes they produce deep destruction of tissue. The epiglottis is often eroded at its margin, and the cartilage may be exposed or perforated. Calcification, as well as necrosis of the laryngeal cartilages, occasionally follows phthisical ulceration. The necessary movement of the arytenoid, and the irritant action of the pulmonary discharges, may induce ulceration of the vocal cords.

Syphilitic Ulcers of the larynx are usually met with among the tertiary manifestations of syphilis, and rarely if ever occur as secondary lesions of the disease. These tertiary ulcers frequently begin on the epiglottis and spread rapidly; they often involve the mucous membrane of the entire larynx, and cause great destruction of tissue. They have an irregular outline, with everted edges and yellow hue, excavated base, and at times present a more or less gangrenous appearance. In some cases they extend to the pharynx. They may originate in the breaking down of syphilitic tubercle or gummata. They often heal at the point attacked, while the ulceration advances in other places. The scars which result from the healing of the ulcers have a tendency to contract and narrow the calibre of the larynx. The papillary growths which surround these ulcers are especially characteristic of their syphilitic origin.

Etiology.—Catarrhal laryngeal ulcers are rarely the result of acute laryngeal catarrh, but, as has been mentioned, are of not infrequent occurrence in chronic catarrhal laryngitis, especially that which accompanies pulmonary phthisis. The follicular variety generally results from the extension of a follicular faucitis from the pharynx to the larynx, or at least the two are frequently associated.

Variolous ulcers have their origin in the propagation of the exanthem from the mouth and pharynx.

Typhous ulcers have their origin either in diphtheritic infiltration or imperfect nutrition of the mucous membrane of the larynx.

Syphilitic ulcers depend upon a specific constitutional poison acting in conjunction with a catarrhal inflammation of the mucous membrane of the larynx.

Phthisical ulcers are always secondary to the development of tubercular tissue, which usually appears in the form of gray nodules, and may develop about the base of any chronic ulcerative process.

Symptoms.—All forms of laryngeal ulcers are attended by the general symptoms of chronic laryngeal catarrh. When a patient with a harsh, stridulous cough of long standing (the expectoration containing pure blood and laryngeal tissue), with hoarseness at times amounting to aphonia, complains of a burning, smarting, pricking sensation in the larynx, with tenderness on pressure, which is increased by speaking, and of difficult and painful deglutition, attended by a wavy laryngeal respiration, there is reason to suspect the existence of a laryngeal ulcer; but a positive diagnosis cannot be made from these symptoms alone, as extensive ulceration may exist and all of these symptoms be wanting, and they may be present where there is only laryngeal catarrh without ulceration. The appearance of the posterior wall of the pharynx is always of great diagnostic importance. The use of the laryngoscope clears up all doubt as to the existence or nonexistence of laryngeal ulcers. By a careful laryngoscopic examination, the existence, as well as the seat and extent of these ulcers may be determined.

Having determined their existence, the history of the patient and a careful auscultatory examination of the chest will enable one to decide their character.

Prognosis.—The prognosis depends entirely upon their character. The catarrhal, follicular, typhous, and variolous are usually readily recovered from; while the phthisical and syphilitic rarely, the former perhaps never, entirely heal; or, if healed, the destruction of the parts is so great that the remaining cicatrix permanently interferes with the functions of the larynx.

Treatment.—The treatment of laryngeal ulcers has been considered under the heads of chronic laryngeal catarrh, phthisical and syphilitic laryngitis. Ulcers which arise independently of these conditions require no special treatment.

NEUROSES OF THE LARYNX.

The true neuroses which are met with in the larynx are due to defective innervation of the recurrent laryngeal nerve. They will be considered under the following heads:—

I. *Recurrent Laryngeal Paralysis.* II. *Paralysis of the Abductors of the Vocal Cords.* III. *Paralysis of the Adductors of the Vocal Cords.* IV. *Paralysis of the Tensors of the Vocal Cords.*

Each form of paralysis may be limited to one side, or may affect both sides of the larynx.

Morbid Anatomy.—Recurrent laryngeal paralysis may be unilateral or bilateral. All the muscles supplied by the recurrent laryngeal nerves are usually affected in this form of paralysis. There is no constant morbid change in the tissues which compose the larynx; frequently, however, there is thickening of the laryngeal mucous membrane, and the vocal cords lose their pearly lustre; such conditions are most likely to be met with in pulmonary phthisis. In the absence of appreciable morbid appearances, this paralysis is produced by insufficient or unequal supply of nerve force. In unilateral paralysis of the adductors, only one of the recurrent nerves is diseased, either primarily or secondarily, or an inflammatory degenerative process may have been established in the muscle involved. Bilateral paralysis of the adductors is caused by compression of both recurrent nerves by tumors, aneurisms of the aorta, innominata, or subclavian arteries, and by degenerative processes in the nerves. Pathological changes at the apices of the lungs, or in the lymph glands in contact with the nerves, may also give rise to it. In bilateral paralysis of the abductors of the vocal cords, or openers of the rima glottidis, there is generally advanced atrophy of the laryngeal muscles, which is evidently dependent upon interruption of nerve force, either from cerebral disease or local pressure on the vagi, or on both recurrent nerves. It is frequently associated with constitutional syphilis. Unilateral paralysis of the abductors is usually of central origin, and is rare. Unilateral paralysis of the vocal cords, of a functional nature, is a rare affection. It is sometimes observed in cases of chronic lead or arsenical poisoning. Bilateral functional paralysis is of common occurrence—it is met with most frequently in hysterical females. In unilateral paralysis of the abductors, local pressure by different kinds of tumors is most frequently met with, and the wasting of muscular tissue, which attends such pressure, is usually limited to one side. It is stated that paralysis of the tensor muscles of the vocal cords indicates changes in the spinal nerve. This condition is not, as is frequently stated, that of functional disturbance. There may be organic lesions present, such as follow contusion or laceration of nerve tissue. Atrophy of the spinal accessory nerves, consecutive to compression in their passage through the foramen lacerum posterius, has occasionally been met with.

Etiology.—The etiology and morbid anatomy of laryngeal paralysis cannot be separated. A common general cause of laryngeal paralysis is some local change in the mucous tissues of the larynx. Women rather than men are subject to it. Pressure on, or traction of, the pneumogastric or recurrent laryngeal nerves, by tumors, enlarged glands, and thoracic aneurisms, is a frequent cause of laryngeal paralysis. Diphtheria, typhus and malarial fevers and other acute blood diseases are occasionally followed by laryngeal paralysis; under these circumstances the paralysis is undoubtedly due to the direct effects of the special poison of the disease upon the nerve centres. The action of certain metallic poisons, such as lead, arsenic, mercury, etc., upon the larynx, after months or years of exposure to their poisonous influence, may cause it. Central diseases in the brain or upper portion of the spinal cord are sometimes its cause. Whenever there is bilateral paralysis of the abductors its cause may be found in some more or less defined lesion of the brain. Paralysis of the laryngeal muscles may occur as a late manifestation of constitutional syphilis. In rare instances laryngeal paralysis may be due to atrophy and degeneration of the laryngeal muscles, and comes on without any assignable cause. Temporary laryngeal paralysis, occurring in connection with hysterical manifestations, has no cause save the erratic one of hysteria, appearing and disappearing without any apparent cause. Mechanical violence not infrequently causes paralysis of the tensors of the larynx, as when a blow is struck, or there is a fall on some projection; it also may occur as a sequela of too loud, too frequent, and too prolonged exercise of the voice in public speaking.

Symptoms.—The phenomena which attend the different forms of laryngeal paralysis are for the most part local in character. In paralysis of the muscles supplied by the recurrent laryngeal nerve, the patient is voiceless and unable to cough. When this form of paralysis is of hysterical origin, the voice comes and goes most capriciously—now it is normal, and in a short time the patient may become completely aphonic without any apparent cause. A laryngoscopic examination of the larynx will show that during attempted phonation the vocal cords remain apart, midway between extreme abduction and adduction: they may be perfectly motionless. In unilateral recurrent laryngeal paralysis, the voice may be but slightly impaired. In rare instances, it will be unchanged during ordinary conversation, and will only be impaired when an endeavor is made to sound the higher notes in singing, or after some extraordinary, continued effort of the vocal organs. The sound produced during coughing, sneezing, and laughing is usually much changed and weakened. The laryngoscope shows that one vocal band does not act when the patient attempts to speak or cough. As has already been stated, this form of paralysis is due to some cause acting directly on the nerve of the affected side.

Bilateral paralysis of the abductors is often accompanied by decided hoarseness and huskiness of the voice, rarely by entire loss of the voice; articulate speech is often almost normal, and then suddenly, as though the current of air were interrupted, the patient is unable to make himself understood, so feeble, so utterly lost, has his phonetic power become. The prominent symptom of this form of paralysis is dyspnœa, with noisy, stridulous inspiration, which is always more or less marked, but becomes greatly aggravated after violent exertion, or on deep inspiration. A laryngoscopic examination shows both vocal bands in juxtaposition, near the median line, and they do not separate when a full inspiration is made; on the contrary, a forced inspiration makes them approach even to touching, while a forced expiration separates them a little. In unilateral abductor paralysis, the voice is shrill and discordant, and dyspnœa is present only after physical exertion. During inspiration the paralyzed band does not move, but its edge is concave. It frequently remains stationary, near the median line, but usually it remains in the median line on account of the unopposed contraction of the abductors. The band seems shorter than normal, and usually is congested, especially after attacks of dyspnœa. Generally there is no difficulty in deglutition in any form of laryngeal paralysis. In those where the bands do not approximate sufficiently to guard the entrance to the larynx, there may be slight dyspnœa.

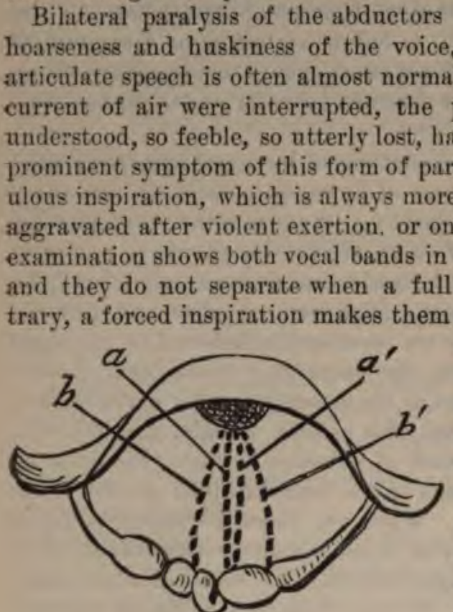

FIG. 13.

Diagram showing position of the vocal bands in abductor and adductor paralysis as seen with the laryngoscope.

The dotted lines a and a′ indicate the position of the bands in bilateral paralysis of the abductors. b and b′ indicate the position in bilateral paralysis of the adductors.

Differential Diagnosis.—Laryngeal paralysis is easily recognized when a careful laryngoscopical examination of the larynx is made. The character of the respiration in paralysis of the abductors, and of that in paralysis of adductors, is usually sufficiently marked to distinguish the one from the other. In adductor paralysis the respiration is always performed with ease; while, in paralysis of the abductors, dyspnœa and stridulous breathing are always present in a greater or less degree. In other forms of laryngeal paralysis, the respiration is normal.

Prognosis.—In those cases where paralysis of the vocal bands depends upon a morbid condition of the nerve centres, or is due to compression of the nerves by aneurisms or new formations, the prognosis is always grave.

On the other hand, it is favorable when it is due to functional causes, or originates in catarrhal inflammation of the mucous lining of the vocal organs. When there is paralysis of the adductors, usually the prognosis is favorable; while with paralysis of the abductors the patient is always in great danger.

Treatment.—In recurrent laryngeal paralysis, where any method of treatment can be of service, the surest and best is the application of the electric current, galvanic or Faradic, one pole being placed over the thyroid or cricoid cartilage, and the other in contact with the vocal cords. These applications must be employed at regular intervals, and only for a short period at any time. As adjuvants, stimulating inhalations may be employed, such as ammonia, creosote, etc., and local applications of iron, nitrate of silver, etc. Whenever the abductor muscles have lost power, it becomes a question whether tracheotomy shall or shall not be performed; if the dyspnœa becomes so intense as to be a source of immediate danger to the patient, tracheotomy should be performed without delay, for it affords the only chance of prolonging life. Rest of the voice is an all-important element of treatment, where there is deficient action of the muscles; and, in obstinate cases, electricity, with the induced or galvanic current, may be used with advantage to the patient. In all forms of laryngeal paralysis, general treatment is indicated.

SPASMODIC AFFECTIONS OF THE LARYNX.

The only spasmodic affection of the larynx which I shall consider, is the common form known as spasm of the glottis, or *laryngismus stridulus*, which is occasioned by temporary spasm of the adductors of the larynx; this gives rise to temporary paroxysms of dyspnœa and stridulous breathing.

Morbid Anatomy.—There are various opinions in regard to the pathological nature of spasms of the glottis. According to some, there exists an altered or abnormal condition of the nerve centres—especially is this the case in children; while other authorities recognize an excessive susceptibility of the glottic nerves to receive reflex impressions. When an adult is affected, there is frequently some catarrhal or other inflammatory condition of the mucous membrane of the larynx, which acts as an efficient cause of the spasm; in children, the mucous lining of the larynx is usually perfectly healthy. In adults, the brain is normal in appearance; in children, serous effusion is frequently found in the ventricles and on the surface of the brain. Evidences of rickets are frequently apparent in the osseous system of children subject to laryngismus. The condition of the pneumogastric nerve has been variously reported by those who have written on this subject. Unquestionably, reflex irritation in the larynx may arise from a great variety of causes.

Etiology.—There can be little doubt but that spasm of the glottis is usually due to a nerve impulse originating in some form of irritation and conveyed by the laryngeal nerves. The seat of the irritation may be in the brain, or at a point in the course of the nerves, or peripheral and reflex. Laryngeal spasm is most frequently met with in children, when indigestion, teething, and impressions of external cold are usually assigned as causes; yet, in most cases of this class, cerebral irritation, due to some other cause, already exists. Scrofulous and cachectic children are said to be especially subject to spasm of the glottis. In adults, it is observed in connection with hysterical manifestations, and is sometimes the result of pressure on the nerves; it also occurs in connection with irritation from foreign bodies. It has been met with as a sequela of whooping-cough.

Symptoms.—In children, the laryngeal spasm usually comes on at night, during sleep. The dyspnœa attending it is often intense, the respirations are stridulous and crowing in character, and the child presents the appearance of deficient oxygenation of the blood. It is sometimes attended by general convulsions, in which there is extreme contraction of the flexor muscles of the extremities; strabismus and involuntary discharge of fæces and urine are sometimes present. The spasm usually subsides suddenly, the recovery is complete, and is never accompanied or followed by fever. One of the characteristics of this affection is the tendency to recurrence of the attacks. Death from suffocation during the paroxysm may occur, but it is exceedingly rare. In adults a spasmodic affection of the larynx is either hysterical in its nature or it depends upon interrupted pressure along the course of the recurrent nerves. It gives rise to symptoms similar to those already described, except that the paroxysms are less severe and are more persistent.

Differential Diagnosis.—The only disease liable to be mistaken for the one under consideration is croup, and its diagnosis has already been considered under that head.

Prognosis.—Those cases which depend upon reflex causes generally recover. The prognosis in every case will depend, however, upon the violence and frequency of the spasm, the age of the patient, and, above all, upon the cause of the spasm; a spasm of the glottis depending upon uninterrupted pressure of an aneurism on the recurrent nerve, is not infrequently the immediate cause of death.

Treatment.—If spasm of the glottis is due to reflex irritation, the cause of the irritation should be immediately removed. In children, dentition or an overloaded stomach is most frequently the source of the irritation. In prolonged attacks, inhalation of ether or chloroform may be tried, or a hot bath, or an emetic may be promptly administered. During the interval between the paroxysms careful attention must be paid to the diet and general hygiene of the patient. If the spasms are severe and prolonged, and the patient seems to be sinking, the trachea must be opened and artificial respiration resorted to. When laryngismus occurs in the adult, those means which have been proved beneficial for children may be employed for its relief. When laryngeal spasm occurs as an hysterical phenomenon, it must be treated in the same manner as any other hysterical symptom. If it occurs in connection with pressure upon any portion of the pneumogastric nerve, one must be prepared at any moment to perform tracheotomy for temporary relief.

TUMORS OF THE LARYNX.

Laryngeal growths may be divided into two classes, *benign* and *malignant*.

Morbid Anatomy.—I shall only briefly consider the morbid anatomy of those laryngeal growths with which one should become familiar on account of their frequency; other forms are more especially interesting on account of their rarity. Morbid growths, as they occur in the larynx, may have a broad base which attaches itself to the interior lining membrane of the larynx, or they may hang, as it were, into the interior of the larynx, from a narrow neck or pedicle. They may vary in size, shape, consistency, and number. They may fill up the cavity of the larynx so as to impair respiration, or they may be of such small size as to pass unnoticed. Three-fifths of all the benign growths which occur in the larynx are *papillomata;* where these growths are congenital, the proportion is even greater. These tumors grow rapidly; sometimes they attain a considerable size in the space of a few months. For the most part, their structure is similar to that of the normal papillæ. Their basic substance is formed of connective-tissue, which receives into its interior, vessels and nerves, while the surface is covered with a layer of epithelium. They have decidedly a villous appearance; some of these growths contain spaces filled with colloid matter; after removal, they are quite likely to recur. Relations have been traced between benign papillary growths and warty cancers. Some cases are related where papillomata have become softened, fatty and cheesy, and have been removed by coughing.

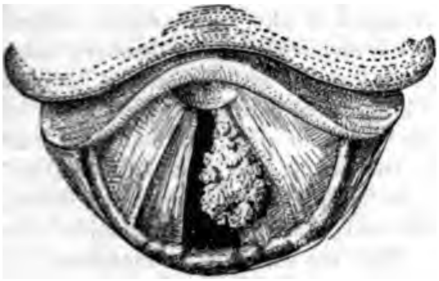

FIG. 14.

Multiple Papilloma of the right Vocal Chord as seen with the Laryngoscope.

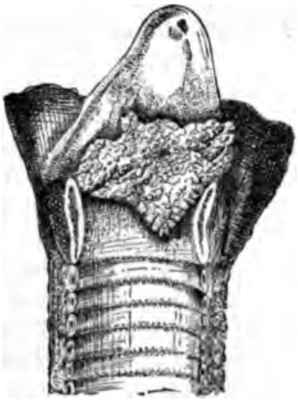

FIG. 15.

The Trachea laid open, showing the same Tumor as seen in Fig. 8.

Fibromata are of less frequent occurrence than papillomata ; they grow less rapidly, and are never congenital. These growths are composed of white fibres, diverging from, and interlacing one another in different directions; after removal they do not return. They are generally smooth, rounded, pedunculated and vascular.

Fibro-cellular growths are composed of fibro-cellular tissue. They usually contain a serous fluid, are of slow growth, single, and after removal show no disposition to return.

Cystic tumors are due to enlargement of the glands in the mucous membrane. They contain a white, sebaceous-like material, and have thick walls. This variety of tumor is less frequently met with than any of the other varieties.

Glandular growths take their origin in the larynx, where the glands and follicles are most abundant. They sometimes attain considerable size.

Carcinomatous growths in the larynx are of two varieties, epithelial and medullary. The epithelial is the more frequent. The medullary is not so liable to ulcerate as is the epithelial, but produces more displacement. Sometimes profuse hemorrhage occurs in connection with epithelial cancer of the larynx.

Etiology of Laryngeal Growths.—The most frequent cause of laryngeal growths is unquestionably chronic or frequently recurring laryngitis. In some cases a more or less constant irritation of the vocal organs seems to give rise to their development, such as is met with among teachers, singers and public speakers. Around the ulcerations of syphilis and of laryngeal phthisis these growths are found. Those whose calling subjects them constantly to the inhalation of irritating vapors or dust, are especially liable to them. Non-malignant tumors of the larynx are always associated in their origin with local hyperæmia. In malignant growths, in addition to the local changes, there are constitutional influences in operation which impart to them a specific character. They are sometimes congenital.

Symptoms.—The symptoms which attend laryngeal growths are for the most part local in character, and these local symptoms will necessarily vary with the size, situation, and nature of these morbid growths, as well as with the size of the larynx. The development of these tumors is rarely accompanied by pain, but sometimes there is a sense of uneasiness as though a foreign mass were in the larynx. Respiration may be more or less interfered with, and there may be severe dyspnœa ; but usually it is present only after violent physical exertion, running, jumping, going up a long flight of stairs, etc. The breathing is sometimes stridulous in character, and frequent suffocative attacks due to spasm may come on. When the growth is above the glottis, all the difficulty in breathing is on inspiration ; the expiration is quite free. The voice is always more or less changed; it is not only altered in quality and liable to sudden changes in intensity, but sometimes it is completely lost. Cough is present in many cases ; it is usually due to accompanying laryngitis ; not infrequently it is voluntarily excited by the desire on the part of the patient to get rid of the laryngeal obstruction. In the expectoration, which is usually increased by coughing, fragments of the growth are sometimes found; as a rule there is nothing which can be considered as distinctive about it. Dysphagia is present in the advanced stages of many laryngeal growths, especially when they are malignant. The most positive evidences of laryngeal growths are furnished by the laryngoscopic examinations. By moderately expert laryngoscopic examinations the seat, size, and, in some cases, the nature of the laryngeal growths will be readily determined.

Differential Diagnosis.—The interference with the functions of the larynx will direct attention to this organ, and if the laryngoscope is used the existence of these growths will rarely be overlooked; when seen it will hardly be possible to confound them with any other disease. The study of the histories of such cases as are recorded in laryngoscopic manuals will be of great assistance in making a differential diagnosis.

Prognosis.—If the growth be pedunculated, of moderate size, and single, with ordinary condition of tolerance, the voice can, in many instances, be entirely restored. If the contrary condition exists, relief may be looked for, but never complete restoration of the voice. As to length of life, other things being equal, the prognosis is more favorable in adults than in children, for the reason that evulsion of the growth by the intra-laryngeal methods is more readily and certainly accomplished in the former than in the latter. Whenever these growths are cancerous in nature they terminate fatally.

Treatment.—If a laryngeal growth is small, and does not interfere with the voice or respiration, the rule is to let it alone; if, on the other hand, it is of considerable size, and is increasing rapidly, endangering life, operative measures, either intra- or extra-laryngeal, must be resorted to. These more properly fall within the province of the specialist than of the general practitioner. Whenever there is great obstruction to respiration, and suffocation seems imminent, tracheotomy should be immediately performed, after which the intra-laryngeal methods of procedure may be resorted to. In malignant laryngeal growths, all remedial measures are only palliative.

Ossification and calcareous infiltration of the cartilages of the larynx are met with in those cases where there has been chronic and frequently recurring laryngitis; not infrequently the calcareous condition of the cartilage, which is sometimes present in connection with chondritis or perichondritis, is preceded by its ossification.

BRONCHITIS.

Bronchitis is essentially an inflammation of the mucous membrane of the larynx, trachea, and bronchial tubes, which may vary in *extent, intensity, duration*, and in the *nature* of its pathological products. Thus it may be limited to the larynx, trachea, and larger bronchi, or it may extend into the capillary tubes; it may be mild or severe in character, run a rapid course, or be indefinitely protracted. It may also be produced by a variety of causes, some external, some internal, some accidental, and others constitutional. It may be *primary* or *secondary*,—primary, when the result of exposure, or produced by the inhalation of irritating gases; secondary, when it arises from constitutional vice, or from previously existing disease. Again, it may occur as a complication during the course of other diseases, such as acute blood disease, pulmonary phthisis, pulmonary emphysema, and cardiac disease. It affects all ages and either sex. One attack predisposes to a second. Bronchitis, clinically and pathologically, may be divided into the following varieties:—(1) *Acute Catarrhal Bronchitis of the large tubes*; (2) *Acute Capillary Bronchitis*; (3) *Chronic Catarrhal Bronchitis*; (4) *Fibrinous or Plastic Bronchitis*; and (5) *Bronchiectasis.*

ACUTE CATARRHAL BRONCHITIS.

This form of bronchial inflammation occurs at all ages. In childhood and old age it most frequently involves the smaller bronchi; in adult life it involves the larger bronchi. It may be mild or severe in type.

Morbid Anatomy.—The morbid anatomy of this variety of bronchitis does not differ essentially, whether it has its seat in the large or small bronchial tubes. In either case it rarely originates in the tubes themselves, but is the continuation of a similar process affecting the nasal, pharyngeal and laryngeal

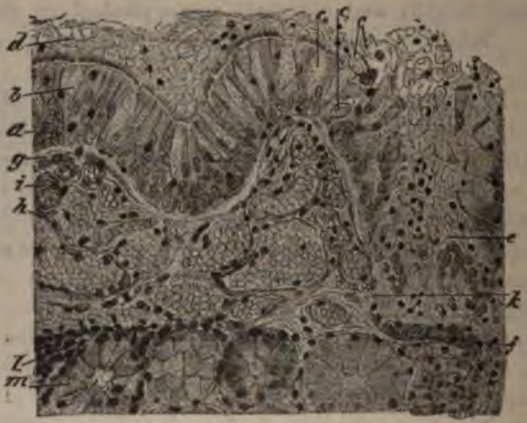

FIG. 16.

Transverse Section of a Portion of a Medium-Sized Bronchial Tube in Acute Catarrhal Bronchitis.

a, *New cells forming.*
b, *Epithelium of mucous membrane.*
c, *Desquamated cells* (c_1 c_2).
d, *Mucus on epithelial surface.*
e, *Opening of mucous gland.*
f, *Attached epithelium.*
g, *Internal fibrous layer.*
h, *i*, *Engorged vessels.*
k, *Extravasated red-blood corpuscles.*
l, *Round cell infiltration.*
m, *Hypertrophied glands.* × 300.

mucous membrane, or is the extension to the smaller tubes of an inflammation commencing in the alveoli. As a rule, the simple variety does not advance beyond the larger bronchi; when the smaller tubes are involved it is denominated *capillary*. In some cases the mucous membrane is swollen and reddened, either uniformly or in points or patches. Its surface may be roughened by the presence of enlarged papillæ or granulations. It is usually softer and moister than natural—occasionally ecchymoses are observed in it. The natural longitudinal rugæ of the membrane are effaced, giving a smooth appearance to the reddened surface. The bronchi at first contain a clear transparent mucus, which, as the disease advances, becomes opaque, whitish, yellowish, or greenish. The change in the color of the secretion is owing to pus-cells contained in the fluid; at the onset there are but few present. The presence of desquamated epithelium in the tubes after death is for the most part owing to the separation of the cells from the membrane between the time of death and the making of the autopsy. In a small proportion of cases, the only evidence of bronchitis which is found at the post-mortem is the presence of mucus or muco-pus in the tubes. Sometimes the tubes are more rigid than normal. These changes exist whether the larger or smaller tubes are involved. Generally, the tubes on both sides are equally affected. In the weak, the very young and the very old, or when there is some condition which prevents or enfeebles the cough, the mucus or muco-pus sometimes gravitates from the larger into the smaller tubes, and gives rise to yellow spots near the surface of the lung; this is especially liable to occur in young, feeble children.

There may be complications with acute bronchitis. The swollen mucous membrane, or the accumulation of mucus or muco-pus may produce a temporary air distention of the alveoli—a condition frequently met with at autopsies, and sometimes mistaken for vesicular emphysema. Fully developed emphysema, as well as atelectasis, may occur as the result of these bronchial obstructions. Atelectasis is specially liable to occur in young children. In these patches of collapsed lung, or as the result of the extension of inflammation from the bronchi to the alveoli, lobular pneumonia is not infrequently developed as a complication. This is rare in the acute bronchitis of the adult, but frequent in children. Pulmonary congestion and œdema are not infrequent complications of general capillary bronchitis. Temporary bronchial dilatation often occurs in children, when the disease affects the smaller tubes, and lasts more than a week.

Etiology.—The most marked predisposing causes of acute bronchial catarrh are infancy and old age, indulgence in enervating habits, or debility from any cause, constitutional diseases, chronic pulmonary affections, the breathing of impure air in badly ventilated apartments, and sudden changes in temperature. It is comparatively rare in continuously hot or cold climates. In our climate it prevails most in the spring and fall. The disease, when primary, is either due to some sudden atmospheric change, to some morbific agent in the atmosphere, or to the action of cold on the surface of the body when imperfectly protected, causing a chilling of the surface. It occurs secondarily in connection with blood-poisoning, as in measles, typhoid and typhus fevers, gout, rheumatism, etc. In the course of other pulmonary affections, and in chronic cardiac diseases, it is of quite frequent occurrence. It may be produced traumatically by the inhalation of irritating gases, particles of dust, etc., which act directly upon the mucous membrane. Those who live in the open air are less liable to it than those living in-doors. At times bronchitis prevails epidemically, associated with influenza and due to the action of some unknown atmospheric influence.

Symptoms.—A common "cold" may be regarded as a bronchitis of the larger tubes. This simple form of bronchial catarrh does not extend below the second division of the bronchi, but expends itself on the larynx, trachea, and large bronchi. Its invasion is commonly marked by coryza, lachrymation, sore throat and slight hoarseness, with chilliness scarcely amounting to rigor. The occurrence of the coryza, with an uneasy sensation in the frontal sinuses, gradually passing from the nasal passages to the larynx and trachea, is diagnostic of its primary character. The pulse is slightly increased in force and frequency, there is aching in the back and limbs, but the general febrile symptoms are usually mild ; in very young and weakly children convulsions may occur. As the bronchial inflammation becomes fully established, more or less pain and discomfort are felt behind the sternum ; there is a sense of tickling, rawness and soreness at the upper portion of the chest, which amounts to actual pain on coughing ; the respirations are somewhat increased in frequency, and there is a sensation of constriction with oppressed breathing which may be somewhat laborious, but there is no evident dyspnœa. The cough, an essential feature of the disease, at first is dry and hacking, sometimes incessant, especially on lying down, and on waking after a long sleep ; it may be paroxysmal in character. After one or two days the cough becomes loose, and is attended with an expectoration of frothy mucus, of a yellowish color and a saline taste ; gradually this becomes muco-purulent and even purulent. As soon as the expectoration becomes free the patient is relieved. The disease lasts from four or five days to two or three weeks, and ends in complete recovery or in chronic bronchitis.

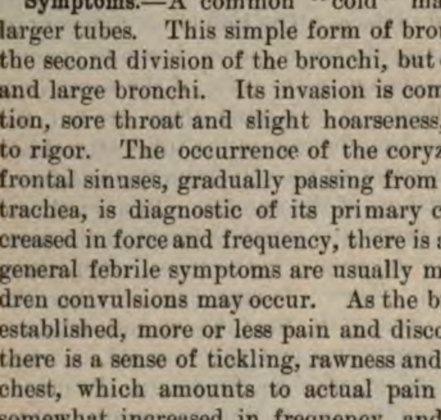

FIG. 17.

Diagram illustrating the Physical Signs of Bronchitis.

Physical Signs.—In slight attacks of acute bronchial catarrh of the larger tubes, there may be no physical signs to indicate its existence. The severer forms are attended by easily recognized physical signs. As a rule, *inspection* and *palpation* give negative results. The *percussion* sounds are normal, unless there is a very considerable accumulation of mucus in the bronchial tubes ; in such cases, the normal resonance is diminished posteriorly in the infra-scapular region. *On auscultation* over the affected tubes, the respiratory murmur is feeble, temporarily suppressed, or sonorous in character. In the dry stage, sibilant and sonorous râles may be heard on both sides over the whole chest, more distinctly posteriorly. In the stage of secretion with the sibilant and sonorous râles, moist râles, large and small in size, are heard on both sides of the chest. These râles are inconstant, coming and going, and changing their situation; after a violent fit of coughing, they may entirely disappear for a time. When they are abundant and very loud, they often altogether mask the respiratory murmur. When the secretion is watery, they have a "rattling" sound. In some cases, secretion takes place so rapidly that moist râles are heard from the first. Vocal resonance in bronchitis is normal.

Differential Diagnosis.—It is hardly possible to confound bronchitis of the large tubes with any other pulmonary affection. The absence of lancinating pains in either side, the bronchial character of the cough and expectoration, the coryza and hoarseness which precede the attack, are usually sufficient to distinguish it from pneumonia and pleurisy; besides, its physical signs, if properly appreciated, render the diagnosis easy and positive in all cases. The early stage of whooping-cough may be confounded with it, until the characteristic cough is heard.

Prognosis.—This form of bronchitis, unless it occurs in the very young, or very old and feeble, never directly destroys life. It usually terminates by resolution in from three to four days to two or three weeks; sometimes it becomes chronic: in such cases the inflammation is likely to extend into some of the smaller tubes, giving rise to circumscribed capillary bronchitis.

Treatment.—In the majority of cases, this form of bronchitis is easily managed. In mild attacks the patient is not sufficiently ill to consult a physician; it is simply regarded as a severe cold. At the onset, while the coryza is present, it may generally be arrested by a Dover's or Tully's powder and a warm bath at night, followed in the morning by a brisk saline purge—in the case of children by a full dose of castor oil. The patient should remain in a warm, moist, equable temperature for a day or two. gr. xx. of quinine or of salicylic acid acts oftentimes as an abortive in adults. If this plan has not been resorted to, or has not proved successful, then moderate but continued action of the skin and kidneys should be induced by the administration of mild diaphoretics and diuretics, the patient remaining in a warm, even temperature. In the early stage of the disease, especially in the case of children, great benefit is often derived from steam inhalations. Counter-irritation, by means of cups and mustard sinapisms, to the upper part of the chest, is of great service in its late as well as in its early stages. If the disease shows a tendency to pass into the chronic stage, or to extend into the smaller tubes, from eight to ten grains of the sulphate of quinine should be daily administered; in children, cod-liver oil with lime-water should be given. A succession of small blisters applied to the posterior portion of the chest will be of service after the acute stage is past. When simple bronchitis occurs in those of a gouty or rheumatic diathesis, colchicum must be given in connection with alkalies.

ACUTE CAPILLARY BRONCHITIS.

When acute catarrhal inflammation invades the small-sized bronchial tubes, it is termed capillary bronchitis. It is also known as "catarrhus senilis," bastard pleurisy, and suffocative catarrh. In most instances, this form is an extension of simple bronchitis, whose characteristic symptoms have preceded; but sometimes the smaller as well as the larger bronchial tubes are affected, or the smaller bronchi may be the primary seat of the inflammatory process. General capillary bronchitis is much more frequently met with in infancy and old age than during any other periods of life. If the inflammation is limited, and only a few of the smaller tubes are involved, it is called localized capillary bronchitis; but when the bronchial inflammation is intense, and diffused over the lining membrane of all the bronchial tubes, it is termed general capillary bronchitis. In the symptoms which attend its development, and in its tendency to destroy life, it differs very much from bronchitis of the larger tubes. The morbid anatomy of this form of bronchitis has been already sufficiently described; but its symptomatology, prognosis, and treatment require separate consideration. The causes which give rise to capillary bronchitis are similar to those which have been named in connection with the etiology of simple bronchitis, except in those instances where it occurs as a secondary affection. The danger from acute catarrhal inflammation of the smaller tubes in patients with Bright's disease, typhus fever, measles, and the chronic bronchitis of old age, should never be lost sight of. Some of the worst cases are met with in connection with emphysema of the lungs.

Symptoms.—The milder types of this form of bronchitis are usually preceded by inflammation of the larger tubes, and the symptoms of invasion are not marked. In fact, the capillary element of the disease might not be recognized, were it not for its physical signs, and difficult or labored respiration. On the other hand, the severe forms may be ushered in by distinct chills, high febrile excitement, and great dyspnœa. The patient is unable to lie down on account of the difficulty of breathing, and the countenance is anxious and flushed. Paroxysmal orthopnœa is not uncommon. The respirations are accelerated, reaching 60 and 70 in a minute, attended by great muscular effort. The pulse is feeble, beating from 100 to 130 in a minute. The axillary temperature is raised to 103° F., but as the disease advances it may fall to 100° F., although the pulse and respiration remain frequent. The patient, at the commencement of the attack, has an incessant hacking cough, which is often so violent as to compel him to sit up, bend forward, and hold his sides. At first, there is little if any expectoration; if expectoration is present, it is a thick, tenacious mucus; later, it becomes more abundant and less tenacious. So viscid and tough is the expectorated material, that cast-like masses of the bronchioles may be formed. When some of the sputa is put in water, the froth floats and is connected by filaments with the heavier masses underneath the surface. The cough may be accompanied by a rattling sound in the trachea. There is great exhaustion. If the disease progresses, all the phenomena of deficient oxygenation are developed. The face betokens great distress and has a livid aspect, the lips become blue, and there is blueness of the finger-ends, with fulness of the jugular veins. The respiratory acts become more and more labored and imperfect, the expectoration becomes more and more abundant, and the matter expectorated thin, frothy, and less tenacious. There is great restlessness, with signs of impending suffocation, and the surface of the body is covered with a cold, clammy sweat. As death approaches, the pulse becomes small and thready, the respiratory efforts are less violent and less frequent, muttering delirium comes on, or the patient lies in a state of partial coma, both cough and expectoration cease, and he dies asphyxiated. These symptoms vary somewhat with the age and peculiarities of the individual affected, and with the diseases which it may complicate. In aged persons, or in those who are constitutionally weak from any cause, the fever is very apt to take on an adynamic type. When it occurs in connection with acute blood diseases, it is likely to come on very insidiously, without any of its usual symptoms being prominent.

Physical Signs.—In addition to the signs belonging to simple bronchitis, the *percussion* sound in the early part of the disease may be somewhat exaggerated in the infraclavicular regions, the percussion resonance may be diminished on account of the attendant pulmonary œdema, and the accumulation of morbid products in the small bronchi. While resonance is diminished in the lower portions, the superior portions of the lung may give an emphysematous percussion note.

Auscultation.—If the bronchitis is extensive, the vesicular murmur over both lungs is feeble or suppressed, and the inspiration may be masked by high-pitched, hissing, sibilant râles ; as the disease advances the subcrepitant distinctive râle of capillary bronchitis is heard all over the chest, but especially in the infra-scapular region. (*See* Fig. 17.) If the subcrepitant râles are abundant and are heard over the whole chest, they indicate very positively the existence of a general capillary bronchitis. These râles may be present over circumscribed spaces posteriorly, as the result of the gravitation of the fluid secretion from the larger into the smaller tubes. If they are confined to the apex or base of one lung, with resonance on percussion, they indicate the existence of a localized capillary bronchitis.

Differential Diagnosis.—Capillary bronchitis may be confounded with *pneumonia, pulmonary œdema, and phthisis.* It differs from *simple bronchitis* in the higher temperature, greater frequency of the respiration, the extreme dyspnœa, the interference with the general capillary circulation, and the presence of the hissing, sibilant, and subcrepitant râles. It is distinguished from *pneumonia* by the absence of pain in the side, prolonged initial chill, and the characteristic pneumonic sputa, by the greater frequency and labor of respiration, and the more intense dyspnœa, by its lower temperature, by the normal or exaggerated resonance on percussion, by the presence of the subcrepitant râles on *both* sides of the chest, and by the absence of bronchial breathing and of increase in the vocal fremitus. The points of differential diagnosis between capillary bronchitis and *phthisis* will be considered under the latter head. The existence of the physical signs of capillary bronchitis at the apex of one lung, accompanied by evidence of pulmonary consolidation at that point, always leads to the suspicion of incipient phthisis. The physical signs, pyrexia, and history of the patient will suffice to distinguish it from asthma.

Prognosis.—General capillary bronchitis is a disease attended with great danger, especially when it occurs in infancy or old age, or when it supervenes upon some grave organic disease, as phthisis, Bright's, heart disease, and acute blood diseases. When it occurs in persons suffering from pulmonary emphysema, although for a time the symptoms are urgent, it rarely proves fatal. It usually lasts from three to five days; but when very extensive may prove fatal in twelve hours. Among the unfavorable symptoms may be named great difficulty of expectoration, shallow breathing, cessation of cough, urgent dyspnœa with evidences of incipient asphyxia, and the presence of adynamic symptoms. In this disease, death results from asphyxia caused by imperfect oxygenation of the blood.

Treatment.—All the so-called antiphlogistic remedies lessen, if they do not destroy, the chances of recovery. From the commencement of the attack, the treatment must be supporting. In general capillary bronchitis, the patient must be kept in bed, the surface of the body covered with flannel, the temperature of the apartment must range from 75° to 80° F. and the air must be moistened with steam. Children should be placed in the steam tent, as advised in the treatment of membranous croup. Dry cups should be applied over the whole surface of the chest, after which it should be covered with an oil-silk jacket. The inhalation of steam usually increases the bronchial secretion, facilitates expectoration, and for a time, at least, relieves the difficult breathing. If symptoms of imperfect oxygenation are developed, the inhalation of oxygen gas in connection with the steam will often afford the most marked relief. The internal administration of muriate of ammonia, or chlorate of potash in five or ten grain doses every two hours to the adult (two grains may be given to a child two years of age), often seems to have a controlling influence over the inflammatory processes. Iodide of potassium is of benefit in children threatened with atelectasis and lobular pneumonia. The so-called expectorants are of little service. Sometimes, when suffocation is imminent and the power of expectoration is entirely lost, stimulating emetics will be found of service, especially in very young children; the action of the emetic seems to supply the want of voluntary power to expectorate, and it dislodges the accumulated secretion in the bronchial tubes; care must be taken not to repeat emesis so often as to produce exhaustion.

In the advanced stage of the disease, when the pulse becomes small and thready, quinine and stimulants must be freely administered. The chief object of treatment in this disease is to sustain the life of the patient until the inflammatory process has passed through its different stages. As regards the use of stimulants, there is no disease (especially of childhood) in which their judicious use is so markedly beneficial. They should be commenced early, and given in sufficiently large quantities to overcome the signs of exhaustion, which are present very early. To allay spasm of the bronchial tubes, which is occasionally present in this form of bronchitis, and gives rise to the most distressing paroxysms of dyspnœa, full doses of hydrocyanic acid may be given, and this is often followed by most marked relief. Opium should never be given, for by its action the power of expectoration is often diminished, and it favors the dangerous accumulation of inflammatory products in the bronchial tubes. Each case should be studied by itself, with attention to the constitutional conditions under which it occurs, and the treatment should be so modified as to meet the indications. The general management of capillary bronchitis associated with Bright's disease is very different from that of capillary bronchitis occurring in a person previously healthy. During the whole course of the disease when this complication is present the patient should receive the largest possible amount of concentrated nutrition—the yolk of eggs and milk are generally well borne by this class of patients. Precaution must be taken against the slightest exposure to changes in temperature during convalescence.

There are certain peculiarities which attend the capillary bronchitis of young children. It differs from the bronchitis of adults in the greater liability to extension of the bronchial inflammation to the alveoli, with consequent lobular pneumonia; also, in the liability to the occurrence of atelectasis or collapse of the lobules, the result of the plugging up of the small bronchi by accumulation of secretion in them, with intense swelling of the mucous membrane. The occurrence of lobular atelectasis cannot be determined with certainty either by the rational or physical signs. It may be suspected in young children whenever physical signs indicative of extensive capillary bronchitis are associated with extreme dyspnœa and evidence of defective oxygenation of the blood, the physical signs and other symptoms of broncho-pneumonia being absent. The development of lobular pneumonia is certain to follow lobular atelectasis, if the life of the patient is sufficiently prolonged after the occurrence of the latter. In the treatment of bronchitis of young children, the liability to these complications should always be borne in mind.

CHRONIC CATARRHAL BRONCHITIS.

This is a very common disease, and results from any cause which excites and keeps up a low grade of inflammation of the bronchial mucous membrane. It is usually a disease of adult life. One of its chief characteristics is its tendency to recurrence; the attacks increase in severity and duration at each return, until the individual is rarely free from it. Chronic bronchitis may be *primary* or *secondary*. *Primary*, when it is the result of exposure to wet or cold, or when it is excited by the daily inhalation of dust, vapors, or other irritating substances. *Secondary*, when due to some constitutional vice, as gout, rheumatism, syphilis, etc.; or some local affection, as cardiac or renal disease. It may occur as a complication of other pulmonary affections, as phthisis, pulmonary emphysema, etc.

Morbid Anatomy.—As in acute bronchitis, any portion of the bronchial and tracheal membrane may be the seat of the inflammatory action. Thus it may be limited to the large bronchi, or it may extend into the capillary tubes. Usually, the inflamed membrane has a slaty, reddish blue, or even a violet color. In the more chronic cases, its tissue is frequently hypertrophied, its glands are enlarged and prominent, and their ducts so increased in size that their mouths are readily visible. The mucus secreted may be in transparent gelatinous masses and small in quantity, it may be muco-purulent, or a serous fluid may be exuded in great abundance. As a rather infrequent occurrence, the surface of the membrane presents an uneven appearance, due to the presence of little villosities covered by normal epithelium; occasionally follicular ulcerations are met with. These papillary excrescences and ulcerations are usually arranged longitudinally. In the early stage, the other coats of the bronchial tubes may be weak or yielding; later, an increase in connective-tissue takes place, leading to thickening and induration. The cartilages are sometimes normal, at other times hypertrophied, and at times calcified. In the posterior wall of the trachea and the larger bronchi, separation of the muscular fibres, and relaxation of the bronchial wall occur, with a protrusion of the mucous membrane through fissures in its middle coat. These diverticuli may involve a large or small extent of the posterior bronchial wall. The submucous coat shows increase in connective-tissue. In very old subjects the ultimate bronchi may be changed into calcified cylinders, each with a minute canal running through it.

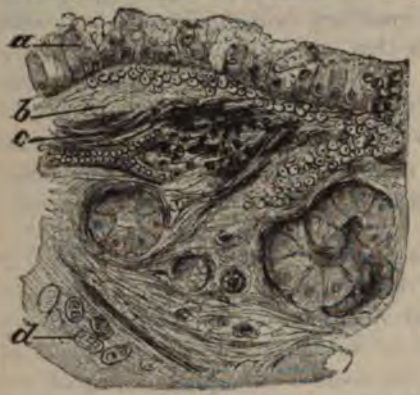

Fig. 18.
Transverse Section of Bronchial Wall in Chronic Catarrhal Bronchitis.

a. Epithelium covered with mucus.
b. Internal elastic coat.
c. Muscular layer. Round cell infiltration seen in one place.
d. Submucous tissue containing hypertrophied cartilage, increased connective-tissue, enlarged glands and vessels. × 250.

In very chronic cases, where there has been a puriform secretion for a long time, the bronchial mucous membrane not infrequently presents slight, or no apparent alteration. The results of chronic bronchitis are dilatation and stenosis of the bronchial tubes, an accumulation of secretion in a state of cheesy degeneration more or less obstructing their calibre, pulmonary emphysema, and induration of lung tissue adjacent to the inflamed bronchi. Ulcerations of the bronchial membrane rarely occur; if present they are slight and superficial, and for the most part are found in the bronchitis which accompanies phthisis. In old age deep ulcers may be formed, and fistulous communications may be established with the œsophagus, aorta, pleural cavity, large blood-vessels, pulmonary parenchyma, or, very rarely, externally. In tertiary syphilis, chronic bronchitis may be accompanied by gummy tumors of the mucous membrane of the trachea and primary bronchi, or by a fibrous induration which leads to stenosis.

Fetid Bronchitis.—An excessively fetid odor of the breath and of the matter expectorated in the course of a chronic bronchitis, may find no explanation after death, except decomposition of the accumulated bronchial secretion. This decomposition usually takes place in bronchial dilatations; it may arise independently of any bronchial dilatation. It is claimed that germs, usually atmospheric, enter, and, lodging in a cavity, cause putrescence. This decomposition of the secretion may exert no special injurious influence, or it may give rise to gangrene of the bronchial mucous membrane, and may thus involve the adjacent lung tissue, causing more or less extensive gangrene of the lungs. About the tubes the characteristic changes of *peribronchitis* are nearly always found; these changes are best marked at the periphery of the lung. The changes that take place in the small bronchi, in that form of bronchial catarrh which accompanies phthisis, will be considered under the head of phthisis.

Etiology.—The most interesting part of the history of chronic catarrhal bronchitis is its etiology. When *primary*, it arises almost always from external causes; such as exposure to cold and wet, the inhalation of dust or unwholesome air. It is unquestionably the exception for chronic bronchitis to be developed from exposure to what are termed the ordinary causes of "taking cold," without some special predisposition, such as long-continued mechanical irritation of the bronchial membrane, constitutional vice, or some previously existing organic disease. Acute bronchitis may frequently be the result of some temporary exposure, but if it becomes chronic, there will almost invariably be found to exist a predisposing cause. Bronchial irritation may exist, perhaps for years, as the result of some mechanical irritation (as in the case of stone-cutters, grain-heavers, etc.), and not particularly inconvenience the individual, until an acute catarrh is developed from exposure; this invariably becomes chronic, and sooner or later leads to the development of broncho-pneumonia, and a condition called knife-grinders' or stone-cutters' phthisis follows.

Secondary chronic bronchitis, or that which arises from some previously existing acquired or congenital dyscrasia, is of more frequent occurrence. An hereditary tendency to gout frequently manifests itself in a form of chronic bronchitis. Sometimes in the same individual attacks of bronchitis and gout alternate. In some instances the gouty diathesis only produces a strong predisposition to bronchitis, which requires for its development some external exciting cause much slighter than would produce the disease in health; in other instances, there is for a long time a slight bronchial catarrh, which, as life advances, slowly merges into chronic. Not infrequently chronic bronchitis occurs in connection with psoriasis and eczema, and these affections alternate one with the other; as one disappears the other manifests itself; under such circumstances it seems evident that these different affections are manifestations of the same constitutional vice. Pulmonary emphysema is produced in many instances by chronic bronchitis; sometimes, however, it occurs independently of it, and then it is a strong predisposing cause to the development of the latter. Disease of the left side of the heart predisposes to bronchitis, which is sub-acute in character and chronic in duration. Chronic bronchitis is very often associated with asthma. Chronic alcoholismus is one of its frequent causes.

Symptoms.—The symptoms of this form of bronchitis vary with the constitutional and local causes under the influence of which it is developed. There are, however, certain prominent characteristics common to all varieties, the most constant of which are cough and expectoration. The peculiarity of the cough, and the quantity and quality of the matter expectorated, determine to a great extent the character and severity of the bronchitis. In some cases the cough is slight, the expectoration moderate in quantity, and muco-purulent in character; this occurs in the mildest variety—a variety which comes on in the winter and disappears, or is mitigated, in summer. After a time it becomes permanent, and is liable to exacerbations in cold, damp weather. It is the simplest form of chronic bronchial catarrh. In another class of cases the cough is violent and more constant, severest in the morning—the expectoration is either tenacious and scanty, or thin, semi-transparent and abundant; it is sometimes streaked with blood, and frequently is difficult to expectorate. So severe is the cough that vomiting is very commonly induced, the contents of the stomach and bronchi being simultaneously expelled. The matter expectorated varies in color from an ashy-yellow to a deep green; it is slightly aërated, and not infrequently sinks in water. Its odor varies: sometimes it is sweet and nauseous; at other times it has a fetor similar to that of gangrene of the lungs. The microscope shows it to be composed of granular matter, broken down epithelial and pus-cells, and sometimes blood-globules and small filaments of bronchial tissue. Some cases of this form of bronchitis are attended by loss of flesh, fever, and night sweats. It occurs most frequently in strumous, broken-down subjects, especially those given to alcoholic excess. More or less extensive bronchial dilatations are usually present in this variety of bronchitis.

Again, there is a class of cases in which the cough is exceedingly troublesome and paroxysmal in character—the expectoration is scanty, consisting of small, rounded, semi-transparent masses of tough mucus. This variety is met with almost exclusively in connection with pulmonary emphysema, gout, spasmodic asthma and irritant inhalations, and has received the name of "dry catarrh." There is also a variety of chronic bronchitis, not infrequently met with in old people, especially in connection with heart disease, in which the cough is paroxysmal, and often violent, and the paroxysms are attended by a peculiar flux from the bronchi. The expectoration often amounts to four or five pints in twenty-four hours, and is either watery and transparent, or gelatinous and ropy, resembling an emulsion of white-of-egg and water. The patient often finds great relief after a paroxysm of coughing and expectoration. In some cases this variety of bronchitis is accompanied by loss of strength and flesh; it has received the name bronchorrhœa. In some cases of simple chronic bronchitis the sputa are moulded in the form of the smaller tubes. Blood in the sputa indicates superficial ulceration. A brownish fluid expectoration is sometimes present; and in this are fatty granules and crystals of cholesterin and margarin. In all these varieties there is dyspnœa and labored respiration—the respiration is much more accelerated in other chronic pulmonary affections than in bronchitis, but it is never so labored. The pulse in a purely chronic bronchitis does not exceed the normal frequency, and on this account it may readily be distinguished from pneumonia and phthisis; besides, in chronic bronchitis the temperature is rarely much above the normal, excepting in those cases which are accompanied by a fetid expectoration. A little uneasiness or soreness is often felt behind the sternum, which is increased by violent coughing; but pain in the side is rarely present. Individuals with any form of chronic bronchitis are unable to sustain prolonged physical exertion without great exhaustion, and they are markedly affected by atmospheric changes.

Physical Signs.—These are very nearly the same as in acute bronchitis.

Inspection shows labored respiration with diminished expansion on inspiration. The chest may appear more convex than normal.

Palpation.—Vocal fremitus varies: if the bronchial walls of the larger tubes are thickened, it is exaggerated; if the tubes are obstructed, or much dilated, it is diminished or absent. In the simple forms of chronic bronchitis the vocal fremitus is normal.

The *percussion* sound rarely differs from that in health: if the accumulation of a thick secretion gives rise to obstruction in some of the bronchi, then localized temporary dulness on percussion is the result.

On *auscultation*, the vesicular murmur is more or less deficient over the whole chest, and the respiratory sound is coarse, loud, and harsh, with prolonged expiration. After free expectoration, it will often be audible at points where it had been inaudible a moment before; it is accompanied, and sometimes entirely masked, by râles of every variety, but chiefly sonorous and sibilant. Large and small mucous râles are present in those cases in which there is abundant liquid secretion. These râles are constantly varying in size and character—at times they may be altogether absent; they are altered in character and position by coughing and by full inspiration. Vocal resonance may be normal, diminished, or slightly exaggerated. Large and persistent gurgles in the lower portion of the lung suggest the existence of bronchiectasis.

Differential Diagnosis.—The diagnosis of chronic bronchitis is rarely attended with difficulty, except in connection with pulmonary phthisis. It may be distinguished from *pleuritic effusions*, not only by the cough and expectoration which attend it, but by the continuance of vocal fremitus, and the existence of resonance on percussion. From *pneumonic* consolidation, by the absence of bronchial breathing, of rusty expectoration, accelerated breathing, and high pulse-rate and temperature. In those cases of chronic bronchitis in which the general health suffers, emaciation takes place and bronchial dilatation occurs. The bronchitis sometimes so closely simulates *phthisis* in its rational and physical signs, that the differential diagnosis is exceedingly difficult; the points of difference will be more fully considered under the head of pulmonary phthisis.

Prognosis.—This disease rarely, if ever, directly destroys life; but when it occurs in the old and feeble, it is always attended with danger, on account of the frequent occurrence of acute attacks involving the small bronchi. Any pulmonary affection associated with chronic bronchitis renders the condition of the patient more serious, on account of the liability to bronchial obstruction from the accumulation of the secretion in the bronchial tubes. It is very apt to lead to the development of pulmonary emphysema, pulmonary collapse, dilated bronchi, and fibrous phthisis. It is rarely recovered from when it occurs in those past middle life. Hepatic congestion, abdominal dropsy, and general anasarca are frequent attendants of chronic bronchitis. Seventy-five per cent. of such cases are complicated by the presence of small, granular kidney.

Treatment.—The one important fact to be borne in mind in the treatment of this affection is, that it rarely occurs as a primary disease, but is due to some constitutional disorder. The patient must be removed from every possible source of bronchial irritation, and be protected from exposure to sudden changes of temperature; flannels should be worn next the skin, and if a suitable climate cannot be obtained, the patient must keep in-doors during bad weather, in well-ventilated apartments, the temperature of which should range from 65° to 70° F. Night air and cold winds must be avoided. The region best adapted to patients affected with any of the forms of bronchitis, is one with a moderately warm, dry atmosphere, protected from cold winds, and of moderately high altitude. In cases that are attended by emaciation, a long sea-voyage is often of the greatest benefit. The diet at all times should be most nutritious. As regards the use of stimulants, no definite statement can be made, but, as a rule, moderate stimulation is of service.

In no disease is a careful study of each individual case more important. The immediate and remote cause of the affection must, if possible, be determined. If the bronchitis is the result of an irritant inhalation, removal from exposure to this is of the greatest importance. If cardiac disease exist, which keeps up the bronchial affection by inducing hyperæmia of the mucous membrane, the treatment should be directed to the cardiac affection, and, if possible, the heart's action regulated. If a gouty or rheumatic diathesis exist, the use of colchicum and alkalies is indicated. Steam inhalations of hyoscyamus, conium, or stramonium are often of great service in gouty bronchitis. When pulmonary emphysema is associated with, or is the apparent cause of the bronchitis, the internal administration of iodide of potassium will be followed by most marked relief. Dilute nitric acid, and the ethereal extract of the acetate of iron are beneficial. In general anæmia accompanying bronchitis, preparations of iron are indicated; in fact, in the majority of cases of chronic bronchitis, a general tonic plan of treatment is attended by the most marked benefit. Quinine, mineral acids, bitter vegetable infusions combined with iron, often prove of great service. Bronchial catarrh, alternating with chronic skin affections, yields most readily to preparations of arsenic and sulphate of zinc.

The treatment of the immediate symptoms must depend upon the quantity of the expectoration, the degree of difficulty which attends its discharge, and the presence or absence of any spasmodic action of the bronchial tubes. When the bronchial secretions are excessive in quantity, steam inhalations of tar, creosote, copaiba, and naphtha are often of great service in limiting their formation; the vapor of iodine, muriate of ammonia, and the different balsams are also of service in accomplishing the same purpose. These remedies may be given internally at the same time. When the power of expectoration is deficient, owing to the adhesive character of the expectoration, stimulating expectorants are indicated, such as senega, serpentaria, camphor, tincture of benzoin, combined with such alkalies as carbonate of potash and soda. In those cases where the bronchial membrane is extremely irritable, the secretions scanty, and the cough attended by violent paroxysms, narcotics and sedatives should be administered in full doses; opium, hydrocyanic acid, hyoscyamus, belladonna, and conium are the most trustworthy agents of this class. Where there is much spasm of the bronchi, shown by the breathing and cough, a few drops of ether or chloroform may be inhaled; when the tendency to the spasm is great, the narcotics and sedatives already referred to should be administered. Tincture of cannabis indica acts well in some of those cases. In all varieties of chronic bronchitis, localized counter-irritation over the seat of the most extensive bronchial changes may sometimes be employed with benefit, such as may be produced by dry cups, sinapisms, blisters, croton-oil, and turpentine. It is never necessary or desirable to abstract blood, either locally or generally. Occasionally, emetics may be employed with benefit, when the bronchial secretion accumulates in the larger tubes and cannot be expectorated. The close connection of chronic bronchitis with dilatation of the bronchi renders it necessary to consider briefly some of the prominent features of the latter.

BRONCHIECTASIS.

Bronchiectasis, or *dilatation* of the bronchial tubes, is closely connected with *chronic bronchitis*.[1]

Morbid Anatomy.—It may be *general* or *partial*. When partial, it is called saccular or ampullar. The dilatation may be cylindrical, fusiform, or sacculated. When dilatations are connected together by tubes of *normal* calibre, the condition is distinguished as the "*moniliform*" dilatation of Cruveilhier. In bronchiectatic cavities, the result of chronic bronchitis, the walls are hypertrophied, the mucous membrane is thickened, and may be covered over with small papillary outgrowths. The submucous tissue is hypertrophied and loses to a great extent its elastic fibres, and the mucous glands are atrophied. The muscular fibres are often dissociated.[1] Bronchiectasis is rarely met with independent of some stenosis; we often find alternate stenosis and dilatation. On the tracheal side these bronchial dilatations usually communicate with a slightly enlarged bronchial tube; but, on the peripheral side, the continuity of the tube is almost or entirely lost by narrowing or *actual obliteration.* Cystic cavities may be found; these are isolated bronchial dilatations, whose supplying bronchus has become permanently obstructed. Pus, muco-pus, crystals of margarin, fibres of lung-tissue, and even chalky *débris* have been found in these cavities. The lung tissue close to the bronchiectases is altered in various ways; there may be fibroid induration, emphysema, lobular pneumonia, and atrophy. The contents of a bronchiectatic cavity may decompose and, ulceration occurring, *gangrene* or *abscess of the lung* may result; but neither gangrene nor abscess occurs with bronchial dilatation as often as collapse and fibroid thickening. The small bronchi, and the bronchi in the lower lobes, are the parts most often involved in bronchiectasis.

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[1] First described by Laënnec.

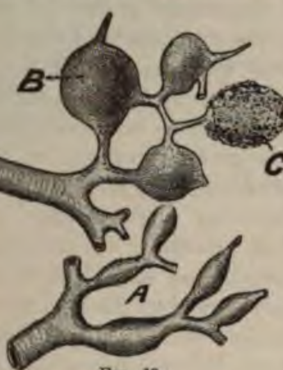

FIG. 19.

Diagram illustrating forms of dilatation of the Bronchi.

A. "*Moniliform*" *dilatation—the tube between the enlargements being of normal size.*
B. "*Sacciform*" *dilatation—several of the ampullar enlargements connecting.*
C. *Formation of a Cyst—by atrophy of the connecting tube.*

Etiology.—Chronic bronchitis is the most frequent cause. Atelectasis, lobular collapse, fibroid induration, and old pleuritic thickenings also cause it. Phthisical processes are nearly always accompanied by more or less bronchial dilatation.

Symptoms.—Many of the symptoms are referred to under the head of "*fetid bronchitis.*" An abundant, fetid, purulent, and often nummular expectoration, frequent and paroxysmal cough, a very fetid breath, some emaciation, occasional profuse hæmoptysis, and not infrequently night sweats, associated with the symptoms of chronic bronchitis, are the characteristics of bronchiectasis. The pulse is accelerated, and there is hectic fever during its advanced stage.

Physical Signs.—*Inspection* shows retraction, prolonged expiratory motion, with diminished expansion on the affected side, or of the whole of the chest if both sides are involved.

Palpation.—There is increased vocal fremitus.

Percussion elicits dulness if the dilatation is filled, or if it is surrounded by consolidated lung. There will be extra resonance if the dilatation is empty and superficial, and there may be a cracked-pot resonance if the dilatation is very large, surrounded by fibrous tissue,and near the surface.

Auscultation.—The respiratory sounds may be harsh, blowing, bronchial, cavernous, or amphoric, according to the seat, size, and condition of the dilatation. Large and small gurgles are often heard.

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[1] In children these bronchiectases not infrequently disappear when the bronchitis which caused them disappears.

Its *differential diagnosis* will be considered in connection with phthisis. This condition cannot be cured. It may exist for many years without materially impairing the general health. Death may be caused by gangrene, abscess, exhaustion, or some complication.

Treatment.—Its treatment is that of chronic catarrhal bronchitis (*q. v*). It is benefited by the daily use of antiseptic sprays of creosote, carbolic acid, etc.; by a residence in a moderately high, warm and dry locality; by a carefully regulated, nourishing diet, and a proper hygiene; and in most instances by tonics in addition to cod-liver oil.

FIBRINOUS BRONCHITIS.

Under this head will be considered croupous, pseudo-membranous, or plastic inflammation of the bronchial mucous membrane, as it occurs independently of laryngeal croup on the one hand, and of croupous pneumonia on the other, or of that form of catarrhal bronchitis during the course of which a few membranous flakes are expectorated. This disease may pursue either an acute or chronic course. Both forms are rare; the acute is the more infrequent.

Morbid Anatomy.—It differs from catarrhal bronchitis in the character of the exudation, as plastic material is poured out into the tubes in the form of casts, which are either solid or hollow, according as the small or large tubes are affected. In the chronic form, the membranous exudation occurs over a circumscribed portion of the bronchial membrane; in the acute, it is distributed over a greater portion of the bronchi. The membrane may be firmly adherent or loosely attached to the mucous surface. These casts are of a whitish color, sometimes dotted over with blood-spots. Microscopically, they consist of fibrillated fibrin, abundant granular matter, oil-globules, exudation corpuscles, and fusiform ovoid cells. They always consist of concentric laminæ. Acetic acid causes them to swell. In some cases no membrane exists; the bronchial membrane is pale and congested.

Etiology.—There is no known special exciting or predisposing cause to this disease—it is supposed to be due to some diathetic state. It is most frequently met with in young adults, and occurs more frequently in females than in males, and in those of feeble, delicate constitutions, rather than in those who are strong and healthy. It has been seen associated with asthma and emphysema. The strumous and phthisical are markedly predisposed to it.

Symptoms.—The acute form is usually preceded by catarrhal symptoms of short duration. It is attended by fever, by dyspnœa (often severe), by a dry, hoarse, ringing cough (not as stridulous as in croup), and by a sense of constriction and oppression across the chest. After severe paroxysms of coughing, either fragments of membrane, or membranous casts or cylinders are expectorated, usually in small masses. The membranous expectoration, in rare instances, is wanting, and occasionally not even cough is present. There are no symptoms of laryngeal obstruction. When the disease progresses toward a fatal termination, the dyspnœa rapidly increases in severity, and is finally superseded by those phenomena which precede death by asphyxia.

The chronic form is generally preceded by catarrhal bronchitis, which sometimes has lasted for a long time; severe hæmoptysis may have preceded its development. Not infrequently, in pulmonary phthisis, where hæmoptysis has occurred, casts of bronchial tubes are expectorated, which are nothing more than decolorized blood-clots. The history of the chronic form of plastic bronchitis is rarely a continuous one, but is made up of intervals of health and paroxysms of disease; during the latter, expectoration of membrane in fragments or casts occurs. Their removal is often preceded by fits of severe coughing, and by paroxysms of dyspnœa of variable intensity, lasting usually a few hours, sometimes a day or more; at other times, simple sneezing effects their removal. Generally, along with the membrane, there is catarrhal expectoration, in which small portions of membrane may be hidden. In about one-third of the cases, hæmoptysis (generally slight) has either preceded or accompanied the membranous expectoration. The membranous exudation, if it comes from the large bronchi, is in the form of casts; if from the small, it is in the form of cylinders. Occasionally, there is mucus or blood in the interior of the casts, while streaks of blood are often present on the exterior. The casts are of variable thickness and length—usually two or three inches long, laminated, and of a whitish or grayish color. Microscopically, they are composed of a structureless mass, more or less fibrous in character, in which cells are imbedded, more particularly pus cells. During the interval between the paroxysms, in uncomplicated plastic bronchitis, the general health is good and fever is not present.

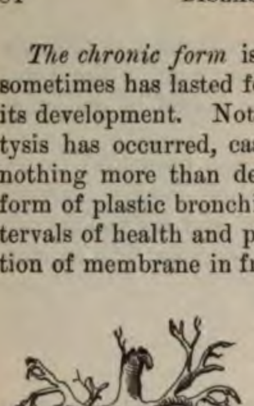

FIG. 20.

Mould of bronchial twigs expectorated in a case of Plastic Bronchitis. One-half the original size.

Physical Signs.—These depend upon the obstruction produced by the membrane, sometimes upon the vibration of a portion of it, and on coincident catarrh. When the bronchial tubes are obstructed, there is feebleness or absence of the respiratory murmur,—in the chronic form, over a limited portion of the chest, in the acute, over a large extent. At the same time, the percussion note may be normal, extra resonant or dull; the latter existing when collapse of the lung has taken place, disappearing, it may be, immediately after membranous expectoration, while the respiratory murmur regains its normal character, thus masking the exact seat of the disease. Flapping and rubbing sounds have been described as a result of vibration of the membrane. Dry and moist râles are also usually present, due either to the narrowing of the tubes, or to coincident bronchial catarrh.

Differential Diagnosis.—This form of bronchitis may be mistaken for *acute catarrhal bronchitis, pneumonia*, or *pleurisy*. The history of the case, the character of the paroxysm, the membranous expectoration, and the accompanying physical signs, will generally enable one to make the diagnosis of plastic bronchitis; without the membranous expectoration, however, the differential diagnosis between acute croupous and acute catarrhal bronchitis cannot be made. The absence of the symptoms which usually attend pneumonia and pleurisy serves to exclude them from the question of diagnosis.

Prognosis.—With the acute form, more than one-half die; with the chronic form, if death occurs, it is due to some complication ; so that, in uncomplicated cases of chronic plastic bronchitis the prognosis as regards life is good ; but the disease, having once occurred, is very apt to return. The duration of the disease varies. In the fatal cases, when the disease is acute, it lasts from three to ten days ; in those cases that recover, it lasts from ten to fourteen days. In the chronic form, the paroxysms usually last ten or twelve days, and recur, at longer or shorter intervals, for months or years. Complete recovery is rare. Croupous bronchitis is very likely to lead to pneumonia and pulmonary phthisis.

Treatment.—The acute form is to be treated the same as croupous laryngitis. In the chronic form, during the paroxysm, alkaline steam inhalations should be resorted to, with the hope of removing the membrane as quickly as possible. The patient should be kept in a warm, equable temperature. During the interval, the general system should be invigorated in every possible way, and all exposure to the causes of bronchial irritation should be avoided. The internal administration of iodide of potassium has been highly recommended ; quinine, iron, and cod-liver oil are often called for. If the paroxysms continue to recur, a change to a warm climate, or a long sea voyage must be tried. There is no known remedy or plan of treatment which promises a cure in this disease.

BRONCHIAL ASTHMA.

This is a spasmodic affection of the bronchial tubes, which gives rise to dyspnœa of a paroxysmal character. The spasmodic contractions may be regarded as due to a *neurosis which depends upon the existence of a peculiar diathesis.* Some muscular spasm or contraction of the circular muscular fibres of the bronchial tubes is the essential element of the asthmatic paroxysm ; and the consequent narrowing of the tubes is a necessary mechanical result. Bronchial catarrh, when present, may precede the paroxysm, or it may not come on until its close. Although bronchitis plays an important part in the development of asthma, it only acts as an exciting cause, as there must exist a special neurotic condition, without which the paroxysm would not occur. This neurosis undoubtedly has its seat in the vasomotor system, and in some instances—notably cases of hay asthma—appears to result in paralysis rather than spasm of the bronchial and vascular walls.

Etiology.—Unquestionably, the primary cause of asthma is some constitutional idiosyncrasy, which is frequently hereditary. Heredity is traced in about forty per cent. It is a diathetic disease, and, like all such diseases, may be readily transmitted from parent to offspring. It is believed by some to be connected with a gouty or rheumatic diathesis. No period of life is exempt from it; I have seen a well-marked paroxysm of asthma in an infant six weeks old, born of an asthmatic mother.

The *exciting causes* of the asthmatic paroxysms may be grouped into three classes :

First.—Those cases in which the bronchial spasm is produced by some material respired which acts directly on the bronchial mucous membrane. In this class are included all those cases of asthma in which the paroxysms are excited by irritating inhalations, such as ipecacuanha powder, many chemical vapors, smoke, dust, fog, emanations from newly mown hay, stables, roses, sulphur matches, burning sealing-wax, certain atmospheric conditions, and the emanations from certain animals, cats, horses, etc., etc. A pure mountain air excites it in some, and relieves it in others. Asthmatics present remarkable peculiarities as to the conditions of atmosphere which suit them best. *Second.*—Those cases which are of more distinct reflex origin. In this class are included those in which the asthmatic paroxysms follow errors in diet, an overloaded rectum, uterine irritation, the sudden application of cold to the surface, the irritation of an enlarged prostate gland, or of hemorrhoidal tumors. Chronic inflammations and obstructive diseases of the nasal cavities are frequent causes of reflex asthmatic paroxysms, which may develop into a permanent asthma. *Third.*—Those cases which occur as complications, or in connection with bronchitis, heart disease, or emphysema, and are most likely to occur after fatigue and physical exertion. Bronchial catarrh is one of its most frequent causes.

Each individual subject to asthma is susceptible only to his own peculiar exciting cause. The retrocession of gout and rheumatism, syphilis, renal diseases, disappearance of chronic skin eruptions, the stoppage of an habitual discharge, or the healing of old ulcers may be followed by asthmatic paroxysms. Certain organic diseases of the brain induce it. Angina, neuralgia, gastralgia and asthma often alternate. That form of asthma termed hay asthma, which is produced by emanations from newly mown hay, or other vegetable emanations, is always preceded or accompanied by coryza and bronchitis; persons may have the coryza and bronchitis for years without having the asthmatic paroxysm, yet the paroxysms are certain to come sooner or later, and differ in no respect from other asthmatic paroxysms.

Symptoms.—An attack of asthma may or may not be preceded by precursory symptoms; the majority of those suffering from asthma know when the attack is coming on, by some peculiar symptom which they alone recognize. There may be languor, drowsiness, depression of spirits, or the opposite condition, abnormal buoyancy of spirits. Often a large amount of "hysterical" urine is passed before the attack, or there may be wakefulness, headache, itching of the chin, etc. Ordinarily, the individual goes to bed as well as usual, and quietly falls asleep; after an hour or two, while he is still asleep, the characteristic wheezing commences, and soon he is awakened by a most distressing attack of dyspnœa. He feels as if his chest were compressed, and he were about to be suffocated; sits up in bed and rests his elbows on his knees, and with fixed head, elevated shoulders and mouth open, labors for breath. There may be immoderate sneezing, attended by running at the nose. There may be flatulent distention of the abdomen. His face becomes red, turgid or livid, his eyes prominent, his surface covered with perspiration; he springs out of bed, and hastens to an open window in search of air; respiration is noisy and wheezing, the inspirations are short and jerking, while the expirations are prolonged, and terminate with a sudden effort at expulsion. The number of respirations varies from sixteen to thirty a minute. All the auxiliary muscles of respiration are brought into play; yet the chest remains almost motionless. The labor may be so great that the patient is in a dripping perspiration. The mouth is wide open, the nostrils are dilated, the cervical and facial veins are turgid. If the bronchial spasm is prolonged, the surface temperature falls below the normal, the extremities become cold, blue and shrunken, and the patient seems to be dying. The pulse during the paroxysm is small, rapid, thready, and feeble in proportion to the intensity of the dyspnœa. The duration of the paroxysm varies; at one time it lasts only a few minutes, at another an hour or two, in rare instances it may continue two or three days without intermission.

As the paroxysm passes off, the patient begins to cough and expectorate; in some patients the expectoration consists of a few small, rounded masses like pearls of mucus, and contains neither pus nor watery constituents; in others it is profuse and watery. The expectoration occurs after the bronchial spasm has ceased; it does not cause its cessation; and even when the attack begins "dry," there is some expectoration at the end of the paroxysm. Occasionally in severe attacks, there are blood-streaks in the sputa, and sometimes quite profuse *hemorrhage* occurs. The paroxysm recurs after intervals of varying length; some experience an attack only annually, others monthly, and others only when subjected to their own peculiar exciting cause. Less violent attacks may last five to six days. During the interval, if the asthma is not due to any organic disease, the condition of the asthmatic subject varies; some are perfectly well; others constantly have a sense of thoracic constriction, which renders the breathing somewhat labored, especially during active exercise. Some suffer severely from a bronchial or nasal catarrh. When the catarrhal element predominates, the asthmatic paroxysms are excited by slight exposure. The longer the attack, the less abrupt its cessation and more profuse the sputa. Immediately after a paroxysm, there is usually a feeling of exhaustion, aching and "soreness," which passes off in a few hours, and the individual experiences a sense of relief, and has for a time an almost certain immunity from a repetition of the attack. Some claim that the *urine* exhibits a remarkable diminution in chloride of sodium and urea just *after* an attack, while later both return to the normal.[1] Analysis of expired air shows oxygen to be almost wholly replaced by carbonic acid, which may be eleven per cent. above the normal quantity.

Physical Signs.—During the paroxysm *inspection* shows labored respiration, while the upper part of the chest is almost motionless, and the muscles of the neck rigid; the inferior costal and abdominal respiration is labored, the act of inspiration is slower than in health, and expiration is more active and violent, and also longer than normal. Vocal fremitus and vocal resonance are normal. The *percussion* sound is slightly exaggerated. On *auscultation* the respiratory murmur is jerking and irregular; sometimes it is exaggerated, at other times it is suppressed. Sibilant and sonorous râles of a high-pitched, hissing and wheezing character are diffused over the whole chest, often loud enough to be heard at a distance from the patient. These are best marked over and between the scapulæ. The respiratory murmur is very faint or absent, especially in the old and where expiration is prolonged and low pitched. All sounds are loudest during expiration. The râle sounds are often musical. These râles are constantly changing their character and site, disappearing at one point and making their appearance at another. At the close of the paroxysm some moist râles may be heard ; or if bronchial catarrh exists then the sounds will be moist throughout.

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[1] Sidney Ringer.

Differential Diagnosis.—Spasmodic asthma will rarely be confounded with any other disease, if its rational and physical signs are properly appreciated. The phenomena of a paroxysm are quite distinctive, while the physical signs are unmistakable. The affections with which there is a possibility of its being confounded are *spasmodic affections of the larynx, acute capillary bronchitis, angina pectoris, hydrothorax, pulmonary œdema and congestion and emphysema.* It is easily distinguished from *laryngeal affections* by the absence of the change in the voice which is so characteristic of laryngeal spasm, and by the presence of auscultatory signs never heard in spasm of the glottis. It is distinguished from *bronchitis* by the slowness of respiration, by the absence of subcrepitant râles and pyrexia, and by the suddenness of its advent. In *angina pectoris* there are no sibilant and sonorous râles. Angina pectoris is accompanied by lancinating pain—absent in asthma —and there are no attendant physical lung symptoms. In *hydrothorax* there is no resonance on percussion over the entire thorax, no succussion, and no change in the line of dulness. In *pulmonary œdema* there is dulness ; in asthma extra resonance. Liquid, bubbling, stationary râles are heard in œdema ; in asthma the râles are dry and constantly change position and character. There is profuse watery expectoration in œdema, absent in asthma. Asthmatic dyspnœa and *cardiac dyspnœa* are sometimes confounded ; in some respects they resemble each other—both are paroxysmal, both are intense, and both generally occur at night. There is little wheezing in cardiac dyspnœa. In both, the respiration may be perfectly normal between the attacks, but a careful physical examination will enable one to determine whether the dyspnœa is asthmatic or cardiac.

Emphysema has a vesiculo-tympanitic percussion note, not present in asthma. Prolonged low-pitched expirations exist in emphysema; in asthma the expirations are never *low* pitched. The barrel-shaped chest and change in the position of the heart are absent in asthma, and are notable signs of emphysema. The two conditions are often found together. In *croup* the dyspnœa is inspiratory ; in asthma it is expiratory. The condition most likely to be mistaken for asthma, in old age, is *latent pericarditis with effusion.* Spasmodic dyspnœa often accompanies it ; but it is marked by a feeble, often an irregular pulse, diminished cardiac impulse, obscure heart sounds and increase in precordial dulness, and there are no râles present.

Prognosis.—Death rarely, if ever, occurs from uncomplicated asthma. Asthmatic patients are frequently long-lived, which may be accounted for by the fact that they are compelled to observe the most rigid hygiene in order to avoid their asthmatic attacks. The fact that a person has had one asthmatic attack, is presumptive evidence that he will have another. The prognosis, as to recovery, is hopeful in proportion to the youth of the patient. If the attacks only come on at long intervals and are not severe and prolonged; if during the intervals the patient is well and there is no organic disease; if the paroxysms can be traced to some obvious cause which may be avoided, the prognosis as to the complete recovery is good. At first, the attacks are violent and exhausting; then they lose their periodical character and run together, as it were, so that the patient is never wholly free from asthmatic dyspnœa. The periodicity varies from one year to one month. Emphysema, chronic passive pulmonary hyperæmia, right cardiac hypertrophy and dilatation, and chronic bronchitis are among its complications.

Treatment.—There are two things to be considered in the treatment of this affection, viz., the relief of the paroxysms and the prevention of their recurrence. The first thing is to ascertain the exciting cause of the paroxysm, and, if it is still in operation, to remove it if possible. If the paroxysm is dependent on an overloaded stomach, an emetic should be administered; if upon a loaded rectum, an enema should be given; if smoke, dust, or any animal or vegetable emanation is the cause of the attack, it must be removed. "Rose fever" and "hay fever" are only relieved by removal from places where there are roses or hay. If, in a certain locality, the attacks of asthma are of frequent occurrence, the patient should remove to one where he is free from asthmatic paroxysms. Not infrequently the removal of the exciting cause will be all that is necessary for the relief of the patient. If the exciting cause cannot be removed, or if its removal is not followed by relief of the paroxysm, free ventilation should be secured, and the patient should be placed in a position in which respiration may be carried on with as little mechanical impediment as possible. Curtains and obstructions to the free entrance of the air should be removed from the room. Usually the best position during an attack is the sitting posture—in a chair which will give support to the arms and so elevate the shoulders. Some *old* people are relieved by sitting before a hot open fire in a close room.

Having placed the patient under the most favorable circumstances for the relief of the paroxysm, the next thing is to select those remedial agents best adapted to the case. This selection will be very much influenced by the patient's own experience and idiosyncrasy. The great majority of asthmatics know the remedies that will give them relief. The remedies that give relief to asthmatics may be divided into three classes: *depressants*, *sedatives*, and *stimulants*. Among the *leading depressants* are antimony, ipecacuanha, tobacco, and lobelia. Ipecacuanha is to be given in quantities *just* sufficient to produce nausea, and tobacco till it begins to sicken. If a patient has been previously relieved by the use of depressants, it is well to inquire which one of this class he made use of. The relief obtained by this class of remedies is by bringing the asthmatic into a condition of complete relaxation. Sometimes this may be accomplished by the administration of one full dose of ipecacuanha.

Sedatives seem to act in two ways: some act locally on the nervous supply of the lungs, but the majority give relief by their action on the general nervous system. Those which experience has shown to be most efficacious in arresting the asthmatic spasm are stramonium, chloroform, belladonna, conium, assafœtida, the bromides, ether, opium, cannabis indica, hyoscyamus, and the fumes of burning nitre paper. Smoking stramonium often relieves when the internal use of the extract is inert. The *datura tatula* is by many regarded as more efficacious than the *datura stramonium.* Smoking tobacco often relieves a paroxysm. Some will be promptly relieved by the inhalation of the fumes of stramonium leaves; others by the inhalation of chloroform. Perhaps there is no agent in this class that will so speedily and completely relieve the spasm as chloroform; but the relief is only temporary; so soon as the stupefying effects have passed away the paroxysm generally returns with increased violence.

Ether is safer to inhale than chloroform, and has no such after-effects. A combination of the two is often efficacious. Trousseau advocates inhalation of vapor of ammonia. Many asthmatics find immediate but temporary relief by spraying the naso-pharyngeal passages with a four per cent. solution of cocaine. This measure is especially valuable in "hay asthma." Quebracho has proved efficacious in a few instances. Recently *grindelia robusta* has been strongly advocated. In this class of remedial agents, that which I have used most successfully is opium given in full doses—small doses are unavailing. One half a grain of the sulphate of morphia should be administered at once. I prefer its hypodermic use. Atropine may be combined with the morphia; there are cases which are quickly relieved by this combination, which are not relieved by the use of either of these drugs alone. Conium, hyoscyamus and belladonna act much less certainly. They should be tried, however. Nitrite of amyl is not so successful as its physiological action would indicate. Iodide of ethyl is advocated by some.[1] Fumes of nitre paper is one of the oldest and best remedies. The paper is prepared by dipping filter or blotting paper in a solution of saltpetre. How it acts is not well understood; certainly not by relieving the bronchial irritation, for, as a rule, the patient is not relieved if bronchitis is associated with the asthma. When this remedy is employed it is necessary that the apartment occupied by the patient should be filled with its fumes. If it acts favorably it will do so quickly, and its administration must not be prolonged if relief is not promptly obtained.

Among *stimulants,* the two principal remedies are coffee and alcohol. Coffee is the more efficacious. It should be taken strong without milk or sugar, and as hot as it can be swallowed; it should always be taken on an empty stomach. Not infrequently a paroxysm of asthma can be warded off by taking two or three cups of strong coffee immediately upon the accession of the first asthmatic symptom. Alcohol is another stimulant which experience has led me to regard very highly. It is of little importance what alcoholic stimulant is employed, but it must be taken hot and strong, that is, as a "hot toddy," and in sufficiently large doses for the patient to feel its intoxicating effects. As a rule asthmatic patients will bear large quantities of alcoholic stimulants without becoming intoxicated. By whichever class of remedial agents the patient is relieved, after a time the remedy will fail or cease to have the desired effect. The three most reliable remedies are ipecacuanha as a depressant, opium as a sedative, and coffee as a stimulant. Compressed air I have never found to give the relief promised by its advocates; nor does inhalation of oxygen allay the paroxysms as a rule. Certain mineral springs are beneficial for asthmatics; the most noted are *Cauterets*, *Mont-Doré*, and *Eaux Bonnes*. Faulkner paints the track of the pneumogastric in the neck with iodine and claims remarkable results. Nitro-glycerine and pilocarpin have both been used.[1] In the intervals the treatment is hygienic; no known remedies can prevent the return of the paroxysms, whereas the observance of certain hygienic rules may often prevent or delay their return. It is claimed by good observers that the removal of nasal obstructions, as deviated septa, turbinated and mucous hypertrophies, etc., is followed by marked or even permanent relief from the asthmatic paroxysms. When skin diseases alternate with attacks of asthma, arsenical preparations are beneficial. Where the cause is undiscoverable, many state that iodide of potassium not only prevents or delays an impending attack, but even effects a permanent cure. Asthmatic patients are usually dyspeptic: and it is a noticeable fact that in such cases, as long as they exercise proper care as to diet, they are free from asthmatic attacks. This is not to be overlooked in the management of asthmatics *between* the paroxysms. A change of residence is all-important in cases dependent for their development upon certain atmospheric causes. There is no definite rule for this change; each one must decide for himself, finding by experience the place in which he is free from his attacks. If the patient is anæmic and poorly nourished, cod-liver oil and iron must be administered during the interval. I have quite a number of asthmatics under observation who, by taking daily from gr. xv. to gr. xx. of the sulphate of quinine, can prevent paroxysms of asthma. As soon as the daily use of the quinine is discontinued, the asthmatic symptoms begin to manifest themselves, and soon culminate in a paroxysm. Helmholtz found relief by injecting into the nostrils a solution of quinia when he suffered from "hay fever." The *diet*, especially in old age, should be strictly regulated. No fats, no water within one hour before dinner or supper, or till three hours after—these should be laws never to be broken. Flannel must be worn next the skin. Exercise should be constant, but moderate. Should the chest development be defective, however, active gymnastic exercise is then to be advised.

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[1] Gazette Médical de Paris, 1878, p. 69.

HÆMOPTYSIS.[2]

In the majority of instances, when blood is expectorated in considerable quantities, the source of the hemorrhage is the bronchial mucous membrane.

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[1] British Medical Journal, Vol. I. 1880, p. 900.

[2] Hæmoptysis means spitting of pure blood only: *i. e.*, the rusty blood-stained sputa of pneumonia do not constitute an hæmoptysis.

Spitting of blood occurs in pulmonary congestion, pulmonary apoplexy, and in the inflammatory processes affecting the lungs and bronchi; but hemorrhage from the bronchial tubes is by far the most frequent cause of blood-spitting or *hæmoptysis;* and when blood comes from the bronchial tubes the hemorrhage is properly called *bronchorrhagia.*

Morbid Anatomy.—If the bronchial mucous membrane is examined soon after or during a bronchial hemorrhage, at the seat of the hemorrhage it will be found swollen, relaxed, bleeding on slight pressure, and of a uniformly dark-red color, with here and there spots of ecchymosis. The lungs are pale but are marked by bright pink spots, corresponding to the air-cells into which blood has been inhaled. If the examination is made some time after the bleeding, the bronchial membrane will either present a pale and bloodless appearance, or no traces of the seat of the hemorrhage can be found. If the examination is made during or soon after the hemorrhage, the bronchi may be found more or less completely filled at points with blood-clots; these clots are usually exsanguinated. The healthy portions of the lungs are inflated. In hæmoptysis occurring in advanced phthisis there will either be aneurism of a pulmonary vessel in a cavity, or ulceration of an exposed vessel. In the early stage of phthisis, tubercle will be found in the walls of the small vessels. If the blood has been forced from the bronchi into the air-cells, small, red, dense nodules will be found scattered through the lung-substance, very closely resembling, in their gross appearance, pulmonary infarction.

Etiology.—Ulceration of the bronchial mucous membrane is rarely a source of bronchial hemorrhage; and seldom does an aneurism open into a bronchus. The two prominent causes of bronchial hemorrhage are: *first,* over-distention of the capillaries of the bronchial mucous membrane; *secondly,* weakness of the capillary walls of the bronchial membrane. Such weakness of the walls of the capillaries may be an hereditary or an acquired condition. In both cases there is probably rupture, which may escape detection at the autopsy. The tendency to bronchial hemorrhage from weakened capillaries is much stronger between the ages of fourteen and thirty, especially in young, delicate persons born of phthisical parents, than at any other time. There is also a strong disposition to this form of hemorrhage in those who are already suffering from developed phthisis, or who have an acquired phthisical diathesis. Usually in these cases the direct cause of the hemorrhage is a sudden distention of the weakened bronchial capillaries from violent physical exertion, or from certain peculiar atmospheric influences. In rare instances it occurs without any appreciable cause. That form of bronchial catarrh which precedes or attends the development of phthisis is very frequently preceded or attended by bronchial hemorrhage. Here, probably, the exciting cause of the hemorrhage is active hyperæmia of the bronchial membrane. Bronchial hemorrhage may be induced by the inhalation of irritating gases or vapors and by the rarefied air of high elevations; in both of these instances the hemorrhage follows over-distention of the capillary vessels of the bronchial membrane.

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[1] Rindfleisch thinks that when phthisis follows hæmoptysis the hemorrhage is due to "vascular tubercular infiltration."

Any form of obstructive heart disease that leads to *passive* hyperæmia of the lungs will predispose to bronchial hemorrhage. Intense *active* hyperæmia will also cause it. The violent coughing of bronchitis, pertussis and pneumonia oftentimes induces it. It may follow suppression of any habitual discharge, and is then called "vicarious bronchorrhagia."

Symptoms.—The symptoms which attend a bronchial hemorrhage vary with the profuseness of the hemorrhage. If the quantity of blood expectorated is very small no symptoms will be developed except the spitting of the blood, which is of a bright red color. It is not often, however, that the symptoms that attend a bronchial hemorrhage are so trivial, for these hemorrhages are usually profuse. All bronchial hemorrhages are attended by the spitting of bright red, frothy, arterial blood. A very profuse bronchial hemorrhage may come on suddenly without any warning, but usually the patient has had some previous indication of its occurrence, such as a sense of constriction at the upper portion of the chest, or a sense of uneasiness during inspiration, which he cannot account for. Those who *have* had hæmoptysis may suffer prodromata, such as headache, dizziness, palpitation, increased pulse-tension and a general constriction about the chest. Cough may or may not precede the hemorrhage. Usually the patient feels as if some fluid had suddenly commenced trickling under the sternum, and he notices an unusual sweetish taste in the mouth. He spits and finds that the fluid is blood, although there may have been no cough previous to the hemorrhage. Now he feels more or less bronchial irritation, which is followed by a cough. Loud moist râles are heard, more or less blood is expectorated, short intervals occur between the fits of coughing, and in this way blood may continue to be expectorated for several days, or the expectoration may continue only for a few hours.

The amount of blood expectorated varies; sometimes, when the hemorrhage is profuse, the whole quantity may reach a pound or more; at other times not more than an ounce or two is expectorated. During the occurrence of the hemorrhage the countenance of the patient assumes an anxious expression; he becomes tremulous and often faints. This, however, is not owing wholly to the loss of blood, but is probably due to the shock to the nervous system from the sight of blood and knowledge of the fact that a hemorrhage from the lungs has taken place. The pulse becomes rapid and tense, and, unless the bleeding is profuse, the face is red. The temperature during the bleeding is usually depressed, but soon returns to the normal. If the temperature rises to 103° or 104° F. during or immediately following, it is a very certain indication that the hæmoptysis is the ushering-in symptom of acute tubercular pneumonia. All these symptoms may be present when the individual has not lost more than half a pound of blood. Hemorrhage from the lungs weakens a patient more than an equal amount of hemorrhage from any other organ of the body. After the profuse expectoration of blood has ceased, the patient goes on coughing for a few days, expectorating small, dark, coagulated masses of blood or blood-streaked sputa. Sometimes bronchial hemorrhage is so profuse that the blood spouts out of the mouth and nose. This is followed by nausea and vomiting of blood, but it is worthy of notice that the nausea and bloody vomiting follow and do not precede the hemorrhage. Death may occur from suffocation in these cases. Rapid and profuse (but *non*-phthisical) hæmoptysis should lead to the suspicion of the rupture of an aneurism. Attacks of bronchial hemorrhage are rarely single; usually for a week or two they recur at intervals. At length the patient becomes pale and feeble, then recovery gradually takes place, so that in a few weeks he may feel better than before the hemorrhage. This is the most favorable termination that can be hoped for, except in those cases in which the hemorrhage is comparatively insignificant. It is important to remember that attacks of bronchial hemorrhage, however profuse, are generally recovered from in spite of extreme prostration and tendency to syncope which sometimes attend their occurrence. When the recovery from a bronchial hemorrhage is not speedy it is quite likely to be followed by more or less febrile excitement, the temperature rising, perhaps, to 101° F., the pulse becoming accelerated and feeble. The patient becomes paler and weaker, has almost complete loss of appetite, and is troubled with a hacking cough, almost constant, which is accompanied by a tenacious, scanty, muco-purulent expectoration. The respiration is hurried, and there is dyspnœa on slight exertion. Under these circumstances the bronchial hemorrhage is followed by broncho-pneumonia, which, in the majority of cases, within a few weeks goes on to more or less complete resolution, and the patient, by means of change of air and proper hygiene, may finally recover. There is another class of cases in which the hemorrhage is followed by still more active febrile symptoms, the temperature rises higher, the pulse is more rapid and feeble, emaciation follows, usually accompanied by profuse night sweats, and the patient dies of *acute phthisis* within a few months after the first hemorrhage. Previous to the hemorrhage he has good health, and there were no physical evidences of disease of the lungs or bronchi. (This subject is more fully considered under the head of Acute Phthisis.)

A physical examination of the chest during a bronchial hemorrhage usually gives negative results. On auscultation nothing abnormal is found, with the exception of a few large and small moist bronchial râles. It is not well to disturb the patient by frequent examinations of the chest.

Differential Diagnosis.—There are four conditions which may be confounded with bronchial hemorrhage, namely, *epistaxis, pulmonary apoplexy, hæmatemesis, and aneurisms* rupturing into the air-passages.

Epistaxis is very easily distinguished from bronchial hemorrhage, for the nose-bleed occurs before the apparent bronchial hemorrhage, and the blood is always coagulated and dark-colored. It is not attended or followed by a cough, and blood can always be detected in the nostrils, posterior nares, or pharynx. The characteristics of the hæmoptysis which occurs in connection with pulmonary apoplexy will be considered under that head. The diagnosis between these two forms of hæmoptysis rests upon the character and quantity of the blood expectorated, and the existence or non-existence of cardiac disease or pyæmia; and here a physical examination is of great importance.

Hæmatemesis is to be distinguished from bronchial hemorrhage by the fact that the blood in hæmatemesis is always coagulated and grumous, of a dark red color, and vomiting precedes or accompanies the hemorrhage.

In bronchial hemorrhage if nausea and vomiting are present they *follow* the spitting of arterial blood; and hæmatemesis is not accompanied or followed by a cough. The gurgling in the bronchi, loose cough, and bright frothy appearance of the blood are never met with in hæmatemesis. Blood is alkaline when from the lungs, and acid from the stomach. "*Spurious hæmoptysis*" (bleeding from gums, pharynx, etc., as from bad teeth) may be confounded with bronchorrhagia, but an examination of the mouth soon reveals the true state of affairs. When an *aneurism* ruptures into a bronchial tube, the hemorrhage is generally profuse, and it is soon followed by death. The long history of aneurism which precedes the rupture, as well as the physical signs, which at least will have led to the suspicion of aneurism, are in most instances sufficient to enable one to determine the nature of the hemorrhage.

Prognosis.—Bronchial hemorrhage rarely proves immediately fatal, or directly endangers life. The prognosis as to final result is always unfavorable; it is in a large proportion of cases either the precursor of phthisis, or a sign that phthisis already exists. It certainly always demands serious consideration. The prognosis is much more favorable when the hemorrhage is due to the excessive action of the heart, or bronchial hyperæmia induced by the inhalation of irritating substances or gases than when it occurs without any apparent exciting cause. Should hæmoptysis be sufficient to induce anæmia, the latter condition is very obstinate and persistent, more so than when it follows other hemorrhages.

Treatment.—*Absolute rest* in a cool room is of the greatest importance. The patient should be placed in bed and not allowed to sit up, turn over, or even speak above a whisper. If the cough continues and is constant, or induces the hemorrhage, it must be quieted by full doses of opium. Ergot, tannin, gallic acid, acetate of lead, spirits of turpentine, persulphate of iron, or common salt may be administered; the balsams, copaiba or sweet spirits of nitre, may be given, if their administration will quiet the anxiety of the patient or friends. It has never seemed to me that styptics or astringents have any control over bronchial hemorrhages, but aconite and opium are the two remedies upon which I rely. When the pulse is full and strong, I use the aconite; when it is weak, I employ morphia hypodermically. The application of ice bags to the surface of the chest may be resorted to in extreme cases, but it must be carefully done, for the reason that patients to whom ice bags are applied are exceedingly liable to have broncho-pneumonia follow their attacks of bronchial hemorrhage. Dry cupping over the surface of the chest is of service whenever the hemorrhage is preceded or attended by marked pulmonary hyperæmia. Patients with hæmoptysis should be urged to eat ice and drink freely of cold drinks. In all cases it is important to keep the patient under observation until all bronchial irritation produced by the presence of blood in the bronchial tubes has subsided. If there is tendency to a return of the hemorrhage, everything likely to bring on an attack must be carefully avoided, and the nutrition of the patient must be improved by the administration of *iron* combined with a most nutritious but non-stimulating diet. Moderate exercise should be taken daily in the open air, and all mental and physical exertion should be avoided.

DISEASES OF THE LUNGS AND PLEURA.

I shall consider the diseases of the lungs and pleura under the following heads :

I. *Acute Lobar Pneumonia.*
II. *Lobular Pneumonia.*
III. *Interstitial Pneumonia.*
IV. *Pulmonary Hyperæmia.*
V. *Pulmonary Œdema.*
VI. *Pulmonary Infarction.*
VII. *Pulmonary Apoplexy.*
VIII. *Pulmonary Gangrene.*
IX. *Pulmonary Anæmia.*
X. *Pulmonary Collapse.*
XI. *Pulmonary Emphysema.*
XII. *Morbid growths in the Lung and Pleura.*
XIII. *Parasitic Diseases.* (*Hydatids.*)
XIV. *Pleurisy.*
XV. *Pulmonary Tuberculosis.*

ACUTE LOBAR PNEUMONIA.

Acute Lobar or *croupous pneumonia* or *pneumonitis* is an acute general disease characterized by an inflammation of the vesicular structure of the lungs, with an exudation into the alveoli which renders them impermeable to air : a condition called "hepatization." A single lobe, the whole of a lung or both lungs may be simultaneously involved.

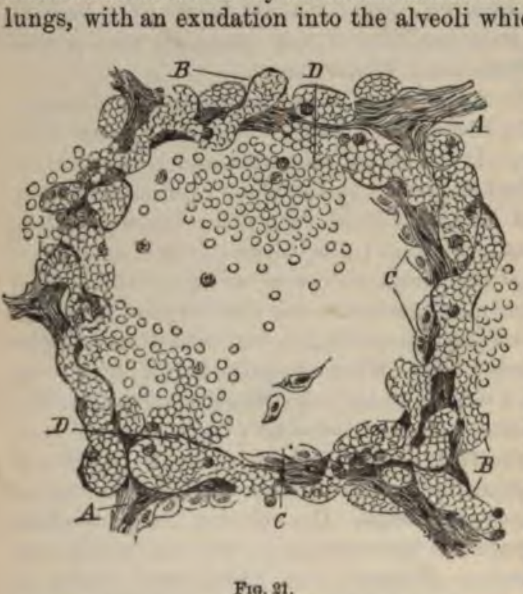

Fig. 21.

Section of Lung showing a single Alveolus in the first Stage of Lobar Pneumonia.

A, A. Wall of alveolus.
B, B. Distended, varicose and tortuous capillaries.
C, C. Alveolar epithelial cells.
D, D. Blood globules in cavity of the alveolus. × 300.

Morbid Anatomy. — Anatomically as well as clinically, lobar pneumonia may be divided into three stages. *First:* a stage of congestion or *engorgement*. *Second:* a stage of consolidation or *red hepatization*. *Third:* a stage of *gray hepatization*. Arterial injection preceding engorgement cannot be demonstrated. Some have called this injection the "*dry stage*" of pneumonia.

Stage of Congestion or Engorgement.—In this stage the portion of lung involved in the pneumonic process does not collapse when the thoracic cavity is opened ; it has a firmer feel than normal, and is more or less distended ; its resiliency is lost ; it crepitates less than normal ;

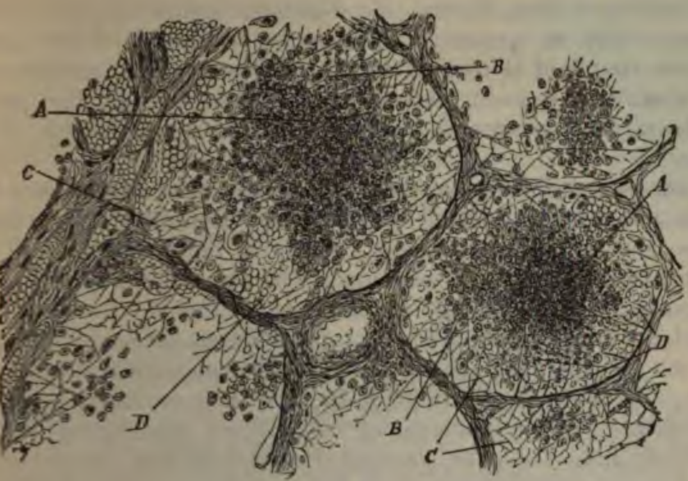

Fig. 22.

Section of Lung in the second Stage of Lobar Pneumonia.

The pulmonary alveoli are seen filled with Pus corpuscles (A, A), changed Epithelial cells (B, B), fibrillated Fibrin (C, C), and red Blood globules (D, D). × 250.

and often pits on pressure. It is not wholly airless, for air can be pressed from one part to another. It is somewhat friable; its color is a dark brownish red, often purple; and its weight and specific gravity are increased.

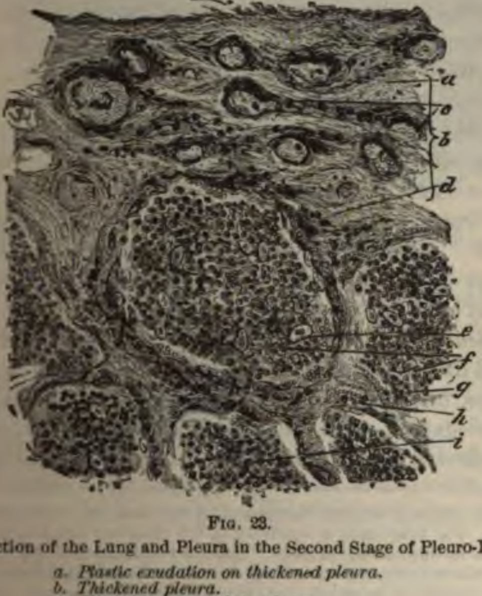

Fig. 23.

Vertical Section of the Lung and Pleura in the Second Stage of Pleuro-Pneumonia.

a. Plastic exudation on thickened pleura.
b. Thickened pleura.
c. Dilated blood-vessels in pleura.
d. Loose epithelial cells.

The alveoli contain red blood corpuscles (e), pus cells (g), changed epithelial cells (f), and fibrillated fibrin (i). The blood-vessels (h) in the alveolar walls are seen filled with red corpuscles. × 250.

On section a thin, frothy, blood-stained serum exudes. Sometimes it flows freely on pressure; it may be tenacious. When alcohol is added to the fluid it coagulates into a granular and amorphous mass. Dark blood flows from the distended capillaries. Occasionally, just under the pleura and between the air sacs, there are small spots of extravasation.

On *microscopical examination* of a thin section of the affected lung tissue the lumen of the alveoli will be found diminished by the distended, varicose and tortuous capillaries. Early in the disease some of the air-sacs may be collapsed from pressure. The alveolar epithelium is swollen and granular. In the air-cells are exfoliated epithelial cells, a few pus cells, red globules and serum.[1] At the autopsy it is sometimes difficult to distinguish the stage of pneumonic congestion from pulmonary œdema and congestion. In the latter the fluid in the alveoli is serum and contains no cell elements. When a stream of water flows over the cut surface of a pneumonic lung the color remains; in œdema and congestion it disappears.

Stage of Red Hepatization.—This stage receives its name from the *color* of the lung and its resemblance, when cut, to *liver-tissue*. The affected portion has a dark liver or mahogany color, and is mottled, the mottling becoming more marked as the stage advances. The volume of the affected lung is increased; so much so that it often bears the impress of the ribs. It is solid, firmer, and heavier than normal. Pressure does not indent, but tears it; it is friable, easily torn, and its torn surfaces have a granular appearance. Its specific gravity is increased. It is *airless*, and there is entire absence of crepitation. Artificial inflation is impossible.

On section the cut surface has a granular appearance; the granules correspond to plugs of inflammatory exudation, which fill the air-cells and the small bronchi. *Torn* surfaces show the granulations better than *cut* surfaces. The granules can be lifted out by means of a fine needle. When cut a dirty red viscid fluid slowly oozes from the surface, or it may only appear after twelve or twenty-four hours' exposure to the air. This may be scraped from the cut surface. A piece of the inflamed lung quickly sinks in water. Small spots of extravasation are sometimes seen. When a stream of water is poured over the cut surface the color changes from a maroon to a gray or a yellow-gray.

On microscopical examination the alveoli are found filled with a solid exudation composed of a network of fibrillated fibrin, in whose meshes are pus-cells, red globules, micrococci, and changed epithelial cells. The latter are in various forms—round, oval, quadrangular, triangular, or irregular—and have received different names, according to the views entertained by different observers in regard to their origin. At first these cells contain fibrinous material, but later they become granular, and then fat globules accumulate in them. They may become discolored from imbibition of hæmatin. The whole contents of an alveolus now present a more or less round form. The interstitial connective-tissue between the lobules may be infiltrated with pus and fibrin; the pulmonary pleura is always coated with fibrin if the surface of the lung is involved; and if the pleurisy precedes the pneumonia, or if it is extensive and an abundant plastic exudation covers the pleura over the inflamed portion of the lung, it receives the name of *pleuro-pneumonia*. The anatomical changes within the lung are, however, unmodified by the more extensive pleurisy, although it undoubtedly delays the processes of pneumonic resolution in the third stage. The red blood globules give the color to the lung. This stage may last from twenty-four hours to several days.

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[1] It is still a disputed question whether the bronchial or pulmonary vessels are the chief source of the pneumonic exudation. The lung-tissue is nourished by the bronchial arteries, while the pulmonary vessels are the medium for the interchange of gases. Hence it is claimed that only the bronchial vessels are implicated. Virchow has shown that pneumonic processes can be established when large branches of the pulmonary artery are plugged; yet he admits that the pulmonary capillaries have, secondarily, much to do with the exudation. Again, it is claimed that early in the disease the parts supplied by the bronchial vessels are not injected as they would be were they alone at fault. Probably both sets of vessels are involved.

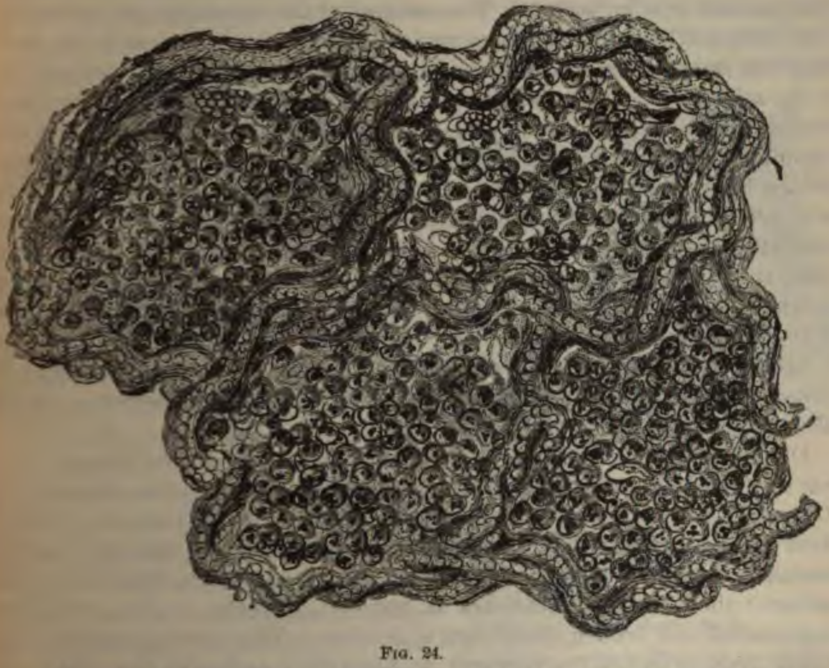

FIG. 24.

Section of Lung in Third Stage of Lobar Pneumonia, showing the Alveoli filled with Granular Matter and Cells, principally mono-nucleated cells.

The blood-vessels of the alveolar wall are much less distended than in the preceding stages. × 250. (*Drawn by camera lucida.*)

Gray Hepatization.—In the early part of this stage the lung remains of the same consistency as in the second stage. There is no sharp transition from red to gray hepatization. The mottling gradually becomes more marked, so that the affected portion becomes "marbled," or has a "granite" look. The surface is gray. The consistency becomes less and less until the tissue is a mere pulp, readily breaking down on pressure. The change in color is due to pressure on the blood-vessels, to the decoloration of the red blood globules, and to the fatty and granular change in the inflammatory products. The weight, friability, and density of the lung are increased.

On section the surface presents a uniformly dirty gray appearance. A reddish gray or dirty white puriform fluid flows either spontaneously or on slight pressure from the cut surface. The "granular" look of the second stage has disappeared or is indistinct. The amount of the accompanying œdema varies; when it is excessive a large quantity of serum exudes, and the tissue does not break down so readily as in other forms of gray hepatization.

On microscopical examination the alveoli are found filled with numerous round, mono-nucleated cells and micrococci; the intercellular fibres that bound the elements together have become granular. The alveoli are filled with a fluid or semi-fluid mass, in which numbers of discrete oil globules and protein granules are freely mingled. The contents of the alveoli are shrunken, and between them and the alveolar wall is a layer of fluid, so that, in a thin section, the contents of an air-sac are readily lifted out by a camel's-hair brush. The pleura over the affected portion is covered with a thin plastic exudation.

Lobar pneumonia may terminate: (1), in *resolution* (recovery); (2), *suppuration* (purulent infiltration); (3), *abscess;* (4), *gangrene;* or (5), *chronic pneumonia.*

During *resolution* the lung is moist, lighter than during hepatization, has a yellow or a yellow-green color, and shows a marked loss of elasticity. *On section* it is now granular, of a yellow-gray hue, and a tenacious puriform fluid readily escapes when the section is pressed. Some œdema may still remain. *Microscopically* the vessels are seen to have returned to their normal calibre; the alveolar epithelium is restored, the cells in the alveoli are degenerated and broken down into a detritus. The coloring matter of the blood gives origin to the pigment so plentifully scattered throughout the liquefied mass. The contents are either expectorated or absorbed; and the lung returns to its normal condition.

When *purulent infiltration or suppuration* occurs, the surface of the lung becomes yellow, its substance is soft, friable, moist, and it feels "miry," as if an abscess were being pressed. *On section* a diffluent purulent fluid exudes from the cut surface. The yellow color is due to the cells that are undergoing fatty change and to the anæmia resulting from over-distention of aveoli with pus. *Microscopically* the pus cells are seen to crowd the alveoli and to infiltrate the inter-alveolar tissue. This infiltration may, by its presence, interfere with the nutrition of the lung tissue, and the alveolar walls may become thin, indistinct and rupture.

Abscess may follow purulent infiltration, a small anfractuous cavity being formed by the rupture of several alveolar septa. These abscesses vary in size from that of a pea to one which may occupy an entire lobe. They may have a thick well-defined wall. Their interior is crossed by shreds of broken-down tissue. They increase either by peripheral growth or by fusion of several small abscesses. Their most common seat is in the lower lobes. These abscesses may be obliterated by a process of granulation and cicatrization. In such cases the abscesses are small, and communicate with a bronchus which allows a free discharge of their contents; or they may be encapsulated in firm cicatricial tissue, their contents subsequently undergoing cheesy and then calcareous changes. They may open into the pleural cavity (causing pyo-pneumothorax), or into the pericardium. External fistulous openings have occurred.

Gangrene occurs in about two per cent. of all cases. It is liable to occur when there is great constitutional weakness, and in chronic alcoholismus or in septicæmia. It may be circumscribed or diffused. The gangrenous portion consists of a dirty pulpy *débris*, sometimes without the "gangrenous fetor." When the part becomes diffluent a cavity is formed and shreds of gangrenous lung tissue are found in a fetid fluid. About this there is a zone of gray hepatized friable tissue, which in turn is bounded by normal lung tissue. In *diffused gangrene*, the cavities are large and shreds of tissue and vascular bands cross from side to side, and the cavity swarms with bacteria. Sloughing of the pleura may follow such a process.

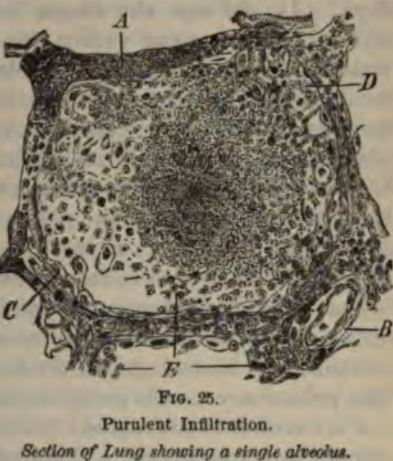

FIG. 25.

Purulent Infiltration.

Section of Lung showing a single alveolus.

A. Alveolar wall, largely infiltrated.
B. Transverse section of a small artery with infiltration of its walls.
C. Epithelial cells of alveolar wall.
D. Capillaries of wall of the air-vesicle.
E. Pus corpuscles.

The alveolar cavity is filled with pus corpuscles, granular fibrin, and a few large nucleated cells. × 250.

Chronic pneumonia may be a result of lobar pneumonia, when resolution is delayed and an interstitial inflammatory process is established during the stage of gray hepatization. The peculiarly hard and œdematous condition that sometimes marks gray hepatization is, by some, regarded as an intermediate stage between croupous and interstitial pneumonia. Finally, the alveolar contents in the third stage may undergo subsequent cheesy changes. Whether this occurs independent of tubercle is doubtful. This is sometimes called *cheesy infiltration* as opposed to *tubercular infiltration.*

In *childhood,* except before the second year, croupous pneumonia is rare. Double pneumonia is, however, more frequent than in adult life. The morbid appearances are the same as in adults. In *old age* the changes are somewhat different; the process usually begins in the upper lobes. In the stage of engorgement crepitation is absent; and in the second stage the lung is blue or nearly black. A *section* shows granules that are much larger than in adult life. "Granulations" are very often absent in senile pneumonia. *Gangrene* is far more frequently a termination of lobar pneumonia in old age than at any other period. The highly rarefied state of the lungs at this time of life seems to favor the development of the small abscesses so common in the aged.

The most frequent seat of lobar pneumonia is the lower lobe of the right lung; the next most frequent seat is the lower lobe of the left lung; then the upper lobe of the right, the middle lobe of this lung being least frequently involved.

Double pneumonia has been variously estimated as occurring in from 5 to 15 per cent. of cases. Even in epidemics it rarely ranges above 12 to 15 per cent. Double pneumonia is more frequent in the senile than in the adult period of life.

The stage of congestion lasts from one to three days; red hepatization from three to seven days; and gray hepatization from two to thirteen days. In old age the stages merge rapidly into each other; abscess of the lung may occur within 36 or 48 hours after the onset. Red hepatization is not infrequently reached within the first six or eight hours in the aged. The changes in the pulmonary *pleura* over a pneumonic lung are quite characteristic. An uneven, thin, downy-looking layer of plastic exudation covers its surface. This plastic layer may conceal the liver-brown color of the pneumonic lung. As the third stage is reached the opposing surfaces of the pleura may become agglutinated. The pleuritic changes follow very closely those which occur within the lung. The cells in the pleuritic exudation are mainly pus. The pleuritic membrane is opaque, congested and ecchymotic. It may become so thick as to give a dull note on percussion after resolution is reached. The *right heart* is dilated; and immediately after death both ventricles may contain clots. The pulmonary vessels going to the involved part may contain thrombi.

Pericarditis is so frequent that it must be regarded as more than a coincidence or complication. The *liver* and *spleen* are congested. The splenic changes resemble those which occur in fevers. The lymphatics of the lung are choked with fibrin and blood corpuscles. The deeper lymphatics contain products identical with those in the pulmonary alveoli. In the lymphatic vessels and in the bronchial glands there is always some evidence of inflammation.

Gastro-intestinal catarrh is sometimes present; and may be attended by hemorrhage. The vessels of the brain are more or less engorged. Meningitis is a not infrequent complication of pneumonia.

Etiology.—Among the predisposing causes *age* ranks first. There are three periods in life in which the liability to pneumonia is greatest: early childhood; 20 to 40; and after 60. Though catarrhal pneumonia is *very* frequent in children,[1] the statement that lobar pneumonia is rare at that period is not correct. From reliable data it appears that lobar pneumonia is five times more frequent in the first two years of life than in the whole succeeding eighteen. Nine-tenths of all deaths after the sixty-fifth year are caused by lobar pneumonia.

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[1] *Vogel.* Kinderkr. s. 222.

Sex. In early life (before the third year) both sexes are equally attacked. Between twenty and forty, when the condition of the sexes is most diverse, the proportion of males to females attacked is 3 or 2 to 1. After sixty, when the condition of the sexes again is similar, there is little disproportion; but always in favor of males. Whenever women work, or are exposed, as men, the disease makes no discrimination as to sex. The *puerperal state* does not seem to increase the predisposition to pneumonia; but it is more apt to occur at the time of the catamenia.

The *general bodily condition* at and before the pneumonic seizure has but little predisposing influence. It is a question whether the strong or the weak are oftenest attacked. Convalescents from acute and severe illness, habitual alcohol drinkers and those who are "malarious" are far more liable to pneumonia than those who are free from such conditions. Enervating habits, poverty, dyscrasia (cancer and chronic nervous diseases especially) and antihygienic surroundings are predisposing causes. Diphtheria, measles, erysipelas, small-pox and the other acute infectious diseases must be regarded as predisposing causes. Chronic and acute uræmia and all diseases which arise from the retention of excrementitious products are powerful predisposing causes. Chronic blood diseases act in like manner. Long-continued passive pulmonary hyperæmia—*e. g.*, from heart disease or from hypostasis—leads to pneumonia. The pneumonia that frequently occurs during acute articular rheumatism has been regarded by some as "metastatic from the joints." A more rational view is that it is due to the blood changes which are part of the rheumatic fever. One attack of pneumonia predisposes to others; twenty-eight attacks have been noted in one individual. When pneumonia follows a severe blow or injury to the chest or shock from any traumatic cause, the injury or shock must be looked on as a predisposing cause. In the *aged* lobar pneumonia has developed as soon as four hours after fracture in the hip joint. *Cold* does not affect the pneumonia rate except in the old. March and April statistics usually exhibit the highest pneumonia rate. A continuously low or high temperature has much less influence than a changeable temperature. Its etiology shows that it is a disease predisposed to by all things that *depress* the vital powers. Children and the aged are greatly depressed by the intense cold of winter and the chilling winds of March and April. In Europe it is often called the "May epidemic."

Pneumonia is unknown in the Polar regions; it is common along the coast of the Mediterranean Sea. Elevation above the sea seems to predispose to it both in hot and cold climates. North and east winds favor its development. Rainy seasons do not influence the pneumonia rate to any appreciable degree; nor do damp or marshy districts. But both have a *marked* influence over bronchitis and other *local* pulmonary diseases. It is a well-established fact that pneumonia occurs oftener among the poor than among the rich, the private soldiers than their officers, the sailor on shore oftener than on ship, the soldier oftener than the civilian at the same military post. All this is explained by the better hygienic surroundings of the one class as compared with the other. The less the resistance capable of being opposed to some (unknown) pneumonic influence, the more strongly predisposed is the individual. Every increase in population in a district increases the pneumonia rate.[1] In New York City from 1840 to 1858 the mortality rate of pneumonia was 5.85 per cent. From 1859 to 1877 it was 6.2 per cent.

The question now meets us: is pneumonia a specific constitutional disease, an acute infectious disease, or a local inflammation ?

The following facts tend to prove that it is *not a local malady*. All kinds of solid and gaseous inhalations and traumatism have failed to produce lobar pneumonia.[2] They always induced *lobular* and not lobar pneumonia. Section of the vagi produces hepatization, but not croupous pneumonic consolidation. Cold does not influence the prevalence of pneumonia as it would were it a local disease (*e. g.*, bronchitis). Wet and cold increase a bronchitis but not a pneumonia rate. Lobar pneumonia is more prevalent in our Southern than in our Northern States. Epidemics in the West Indies were more devastating than those in Iceland. On our continent the prevalence of pneumonia increases from pole to equator. All acute general diseases increase with the population; pneumonia does this. Statistics show pneumonia to be more frequent in New York City now than twenty years ago.[3] While cold has something to do with its development, the *exciting* effect of cold cannot be accepted. Again, there is *no relation* between the amount of lung involved and the intensity of the symptoms.[4] In local inflammations the reverse of this is true. No second chill occurs when another lobe, part, or the other lung is attacked.[5] Prodromata sometimes occur in pneumonia. But the absence of regular and constant prodromata, the absence of a (known) period of incubation, of a typical temperature range, and of characteristic surface phenomena, the fact that the disease is not contagious—these are the reasons advanced by those who regard it as a local, not a general disease. The *resemblances* of pneumonia to acute general diseases are: distinct initiatory chill, an orderly pyrexia, a rather typical course, *i. e.*, a day of abrupt crisis, a definite duration, and the symptoms following in regular sequence. There is a peculiar facies; an occasional herpetic eruption; nephritis is not rare; the cerebral symptoms resemble those of the exanthems; there are sweats and sudamina; and its mode of commencement—coma in the old and convulsions in the young—indicate that it is an acute general disease. Etiologically it is often developed under conditions similar to those which attend the development of diphtheria and cerebro-spinal meningitis; atmospheric conditions are acknowledged factors in its causation. English writers describe a "sewer-gas pneumonia." There have been epidemics of pneumonia in garrisons and aboard ship where there was overcrowding, bad hygiene, etc., etc.[1] There is a "pythogenic" pneumonia arising under miasmatic influences which is contagious.[2] The epidemic form of pneumonia at certain times bears the distinct characteristics of a specific infectious disease.[3] Miasmatic and zymotic pneumonia are names indicative of a supposed origin. We have abortive cases of pneumonia, just as we have abortive typhoid and cerebro-spinal meningitis. Again, the names sthenic, asthenic, malignant, icteric, etc., etc., indicate varieties similar to those found in fevers and acute general diseases. Pneumonia is allied to acute general diseases by the fact that certain complications occur with more or less regularity—lesions in the peri- and endocardium and albuminuria—and that abortion is usually induced when it attacks pregnant women. It has visceral and blood changes very like those of fevers. Pneumonia is sometimes a disease of intra-uterine life. No local disease occurs in the fœtus, but fevers frequently do.

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[1] Hirsch says: "The amount of mean fluctuation in the mortality from pneumonia is in inverse ratio to the density of the population."

[2] *Virchow's Archiv*, Bd. LXXX. *Heidenhain*, *Sityl.* K. K. Akad zu Wien, 867. *Reitz*, *Gendrin*, Hist. Anat. des Inflam

[3] *N. Y. Med. Record:* Article on Causes of Death in Acute Pneumonia.—*Loomis.*

[4] "The local inflammation * * * offers no sort of parallelism to the accompanying fever."—*Sturges.*

[5] "Small consolidations with high fever and severe constitutional symptoms, and extensive infiltrations with a comparatively slight fever—this is the rule, not the exception."—Ziemssen's Cycl. Vol. 5, p. 146.

The success of modern methods of treatment based on this belief bears evidence to its being a general (self-limiting) acute febrile disease. The nature and action of the poison that may be supposed to cause pneumonia are indicated by the following facts:—hyperinosis does not seem capable of causing pneumonia: fibrin increases as hepatization advances and does not ante-date it or the pyrexia.[4] Its resemblance to the acute general diseases is mainly in its nervous phenomena, and the complications which render pneumonia dangerous are those which diminish the *nerve supply* or weaken the muscle-power of the *heart*.

While pneumonia is thus admitted to be a general constitutional disease with local manifestations, undoubtedly depending upon a specific cause, that specific element is at present not satisfactorily determined. Various micrococci have been described as the exciting cause of the disease. To my mind the proofs of the invariable etiological relation of any one are insufficient. It seems more probable that under certain predisposing conditions several micro-organisms may become the exciting cause of a pneumonia.

Symptoms.—*Subjective or rational symptoms.* The invasion, in about one fourth of the cases, is preceded by prodromata.[5] In old age they are more frequent than in adult life (60 per cent.). They rarely occur in children. For a day or longer there may be *malaise*, anorexia, headache, dull pains in the limbs, back, and lumbar region, vertigo, epistaxis, and slight diarrhœa, or there may be slight jaundice, flashes of heat, and rigors. Flying pains in the limbs and chest are common in old age. Rise in temperature is sometimes a prodrome. In Bellevue Hospital, in 1877, a patient for two or three days preceding the initial chill had a temperature of 102°–103°

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[1] In the *U. S. Sanitary Commission Memoirs* Dr. Russel reports: "The surgeons on duty with the regiments in the barracks (Benton, Mo.) report that men occupying the same bunks with those affected were very much more liable to be attacked than those more remote. Some of the most intelligent surgeons believed that it was actually contagious."

[2] *Dublin Med. Journal*, Vol. I. 1874.

[3] *Berliner Klinische Wochenschrift*, 1879, No. 378.—*Kuhn.*

[4] Pneumonia resembles quinsy and acute articular rheumatism. Trousseau finds a resemblance between erysipelas and pneumonia. Sturges places it in a "middle class" between specific diseases and local inflammations. Cohnheim calls it a miasmatic contagious disease. The idea of its being a specific general disease dates from the eighteenth century. *Nov. Theo. Morg.* 1786, *Strackins.*

[5] Grisolle found them in about 25 per cent. of his cases. Fox found them in 28 per cent.

F. In epidemics, febrile symptoms and diarrhœa are common.[1] In most cases the invasion is sudden, and the disease is ushered in by a distinct chill.[2]

Generally the patient is seized with a chill in the night. This chill is intense and prolonged, more so than in any other disease except pyæmia and malarial fever. It lasts from one half an hour to three hours. Its abruptness and violence are characteristic. In children, headache, nausea, vomiting, delirium and convulsions may usher in the disease, its onset resembling that of an exanthem; when these do not occur in all their intensity, the child is restless or stupid, and there are thirst and anorexia, increasing towards night. Again, a child may awake in the middle of the night with a burning skin, bounding pulse, flushed face and hacking cough. When, in children, the pneumonia is ushered in by convulsions followed by a loss of consciousness, the consolidation is usually at the apex.

A distinct chill is less frequent in the pneumonia of *old age*; yet when an old person has a marked chill pneumonia may always be suspected; although less frequent it is more diagnostic than in adults. A protracted fit of shivering and pain in the side are the two initial symptoms in about 50 per cent. of the cases of acute sthenic senile pneumonia. In the other half of the cases the onset is attended by slight increase in the frequency, and irregularity of the respirations, slight pyrexia, short hacking cough, and a feeling of great exhaustion. Intense weakness may be the only symptom. Nausea, vomiting, diarrhœa and collapse, or a semi comatose condition, not infrequently usher in a senile lobar pneumonia. In a very few cases, stupor, coma, and disturbance of intellect may be the only early noticeable symptoms, and they may persist during the whole course of the disease. The initial *chill* (whenever occurring) is rarely repeated.

With the initial symptoms there is a *rapid rise in temperature* accompanied by pain in the side, which is aggravated by coughing and by deep inspiration. The breathing is accelerated, there is dyspnœa, cough, expectoration, the countenance is flushed and anxious, there is headache, loss of appetite, and intense thirst. The urine is scanty and dark. The bowels are constipated. The tongue is heavily coated. The symptoms increase until the day of crisis, when they either suddenly remit and the patient breaks out in a profuse sweat, or they subside by lysis. The defervescence is usually reached between the fifth and ninth day.

The following is an analysis of the prominent objective symptoms of pneumonia:

The *respiration* is more constantly increased in frequency in pneumonia than in any other acute disease, and varies from 30 to 80 per minute. Usually, in acute diseases, the respirations increase with the pulse rate; in lobar pneumonia the ratio between pulse and respiration is early perverted. The respiration may be 80 per minute and the pulse rate not more than 100. The acceleration is not in proportion to the amount of lung involved, and it does not depend on the pain in the chest or the pyrexia. It is *panting*, not "catching," in character. It may or may not be accompanied by dyspnœa. In children accelerated breathing is more marked than in adults. The discrepancy between the pulse and respiration is not as marked as in adults; in the former the pulse may range between 150 and 160, and in the latter between 80 and 90. Expansion of the nostrils is an early symptom in the pneumonia of children. In *old age* expiration is sudden, the whole act is "panting." The average number of respirations per minute is 22, and the duration of inspiration is to that of expiration as 6 to 9. It is rarely accompanied by dyspnœa. An exaggeration of (normal) senile "catching breathing" is one of the most frequent forms of abnormal respiration in senile pneumonia.

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[1] *London Lancet*, 1878, Vol. II.

[2] 77-80-92 per cent. are the figures given by Fox, Louis, Huss, Grisolle and Lebert as representing the frequency of the initiatory chill.

Dyspnœa, although frequent, is by no means constant. It does not depend upon the amount of lung involved, since double pneumonia may be accompanied by less dyspnœa than when only a single lobe is involved. It is often so great that the patient is unable to lie down. The greatest dyspnœa occurs where there is marked nervous prostration. In "secondary" and complicated pneumonia the dyspnœa is greater than in primary uncomplicated pneumonia; it is panting, not labored. In *children*, dyspnœa is most marked when the apex of the lung is involved. In *old age* dyspnœa is so infrequent that even with respiration at 70 they do not complain of difficult breathing. When a patient over seventy years who is asthmatic, or who has chronic bronchitis, develops a pneumonia, the dyspnœa that may have accompanied the previous condition *diminishes*. He simply feels exhausted, and usually dies suddenly.

Pain follows the chill; it is situated underneath the nipple of the affected side. It is sharp and stabbing, often located over the pneumonic spot, and is intensified by coughing, sneezing and deep inspirations. In central pneumonia there is no pain; it is the pleurisy that causes it. Pneumonia itself is a painless disease. Pain in the affected side rarely continues beyond the third or fourth day. If it continues beyond the eighth day it is evidence of pleuro-pneumonia. It is present in 85 per cent. of all cases. In *old age* pain is *never* severe. It is rather a dull, uneasy feeling referred to the whole chest or to the abdomen.

Cough is present in over 90 per cent. of the cases. It comes on within twenty-four hours after the advent of the disease. At first it is short, "hacking" in character. It may entirely cease just before a fatal issue. It is more constant in children than in adults; it is sometimes paroxysmal. Old people with pneumonia often have no cough. When present it may be so slight as to escape the notice of both patient and physician. Should bronchitis or asthma have preceded the pneumonia, the cough diminishes, and may wholly disappear on the advent of the latter. The *expectoration* is characteristic. In the first forty-eight hours of the disease it is simply frothy mucus. Then it becomes semi-transparent, viscid, gelatinous and tenacious, but *never* opaque. So tenacious is it that the cup containing it may be inverted without spilling the mass. It can be drawn out between the thumb and finger into thin strings. This tenacity in great part causes the difficulty of expectoration. Its color varies. About the second day the "brick-dust" or "rusty" sputa may be observed. This color is due to the presence of blood. The sputa may be creamy and yellow, or of a very dark or prune-juice color; the latter is indicative of a depraved blood state, and occurs especially in alcoholic subjects. As death approaches, the sputa become scanty, less tenacious, more diffluent, and often of a greenish hue. Greenish sputa may occur in the middle of the pneumonia and during resolution, and in "bilious pneumonia." When resolution occurs the sputum becomes abundant, and of a yellow, creamy color. There may be no sputum throughout; or it may not appear until the sixth or even the twelfth day. The sputum may remain brick-dust till the ninth or tenth day. In pneumonia of the apex and in that complicating acute articular rheumatism the sputa are often entirely wanting. In *children* sputa are usually absent; but brick-dust masses may be detected in the matters vomited. In *senile pneumonia* expectoration is never an early symptom, and is liable to cease suddenly during any period of the disease. Rusty sputa are present in about 33 per cent. only of such cases; frothy or "catarrhal" sputa are the rule. A chocolate-looking serous sputum, appearing soon after the onset of a pneumonia, shows a depraved condition and indicates "typhoid pneumonia." Examined under the microscope the sputum is found to contain swollen spheroidal red and white blood discs, minute fat spherules and the other elements described under morbid anatomy.[1] In about 75 per cent. of cases there will be found in the sputa (when floated in water) casts of the alveoli and bronchioles. The chemical constituents of the sputa are albumen and mucin. Tyrosin and sugar are sometimes found in it. There are two explanations of the acid reaction of the sputa: Verdeil thought it due to excess of *pneumic* acid; Bamberger claims that it is due to deficiency in alkaline phosphates.[2] Early in pneumonia there is an increase in the chloride of sodium in the serum, and it has been thought that, from the rapid and excessive cell-transformation in the lung, chloride of sodium is attracted to that organ.[3] The expired air in pneumonia is colder than normal, and there is a diminution in the amount of carbonic acid excreted.

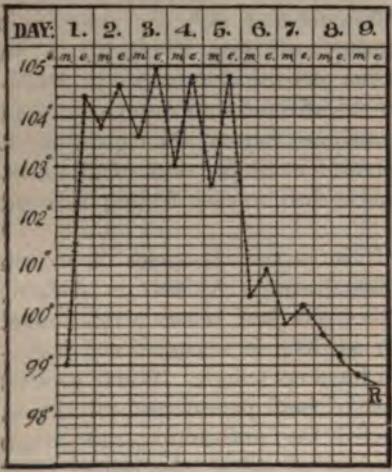

FIG. 26.

Temperature Record in a case of Acute Lobar Pneumonia, ending in Recovery.

The *temperature-range* of a typical case of lobar pneumonia indicates that it belongs to a remittent or sub-remittent type, rather than to the class of febrile disorders marked by a continuously high temperature. Rarely, it is intermittent. The temperature rises suddenly during the initial chill, and in two to three hours after it may range from 102° to 105° F. After the first twenty-four hours the temperature is subject to morning and evening exacerbations and remissions; but the morning temperature is rarely 2° F. lower than the evening—the difference in the *sub*-remittent type may amount to only ½° or 1° F. At midnight a second exacerbation may occur, but not so marked as that occurring early in the evening. Rarely, remissions occur in the evening and exacerbations in the morning. The temperature is usually highest on the evening of the third day. In some cases the maximum is not reached till a few hours before the crisis. Just before death the temperature may rise very high, even to 109½° F. If, after the fourth day, a marked remission is followed by a high temperature, it indicates either an extension of the pneumonia, or the occurrence of some active complication. If, in a mild pneumonia, the temperature suddenly rises, it indicates a grave complication. The sudden fall of temperature on the fifth or sixth day indicates a *crisis*, and the beginning of convalescence: it may occur in the morning or after the evening exacerbation. In a typical case it is usual to find the temperature on the morning of the fifth, sixth, or seventh day two or more degrees lower than on the preceding night. Then it falls gradually until a normal, often a subnormal temperature is reached. The crisis may show itself by successively increasing remissions, while the temperature during the exacerbations rises to the same height as before. It is usual for the remission to be exaggerated just before the crisis; again, the fever may reach its highest point just before the final fall. When the temperature declines gradually ("*lysis*"), a normal point is usually reached by the ninth day, sometimes not until the twelfth or fourteenth. A protracted, slow fall is met with oftenest in the weak, debilitated, and in those who have been bled or depressed by treatment. A continuously high temperature after the tenth day indicates purulent infiltration. (See Fig. 25.) Pneumonia *at the apex* has the highest temperature range. The fifth and seventh are the days of crisis in the majority of uncomplicated cases. Of 867 cases, 677 ended before the eighth day. Neither the height of the fever nor the amount of lung involved influences the day of crisis. In *bilious* pneumonia occurring in miasmatic regions, the temperature is paroxysmal. *In children* the temperature rises very rapidly, sometimes reaching 106° in the first twelve hours. The highest recorded temperatures are in the pneumonias of children. The critical fall is remarkable, the temperature quite often reaching 2° to 2½°

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[1] Dr. Walshe affirms that pus cells are not found in the brick-dust sputum.

[2] Catarrhal sputa contain 10 to 14 per cent. of alkaline phosphates: pneumonic sputa none. In catarrh, soda is to potash as 31 to 20: in pneumonia, 15 to 41. There is 5 per cent. more sulphuric acid in pneumonic sputa than in catarrhal.

[3] In one case where there was no chloride of sodium in the urine, 10 per cent. of the solid material of the sputa consisted of that salt.

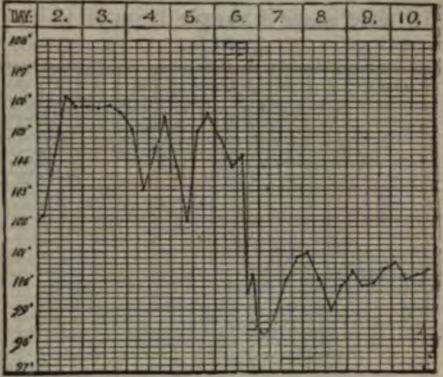

FIG. 27.

Temperature Record in a case of Acute Lobar Pneumonia, with observations every six hours. Recovery.

below normal. This low temperature may continue two or three days. In *old age* it is mainly by the temperature that the exact time of invasion is determined. The rectal temperature may be 103° to 104° on the first days, and then continue at the initial point for three or four days, with morning and evening oscillations of a degree or 1½°. The temperature does not begin to rise until several hours after the chill, if the chill occur. (See Fig. 21.

The *pulse* varies with the severity, extent and stage of the pneumonia. In mild cases it ranges from 90 to 120; if it continues above 120 the case is severe. The pulse is soft and full at the onset. Later it becomes small and feeble. In severe cases, and when the nervous system is markedly implicated, it is rapid, small and feeble, and may be 130 to 140 or 160 at the onset. High temperature is usually accompanied by a rapid pulse, and *vice versâ*. When the critical fall of temperature occurs, the pulse falls correspondingly. After the third or fourth day the pulse exhibits *dicrotism* in many cases; it may be jerky, very compressible and intermittent. Just before death, it becomes markedly slow in many instances. It is not the most extensive pneumonia that is accompanied by the greatest flagging of the heart. Heart-failure may exist before, or just as hepatization is commencing. The pneumonia with the highest temperature is *not* the one where heart failure is most marked or occurs earliest. When the heart is failing, the pulse shows that the artery is unequally filled by each beat. First, the *force* varies; then waves occur, and finally true intermission. I have been able to detect heart-insufficiency by these variations in the pulse within twenty-four hours after the onset of the pneumonia, and occasionally during the initial chill. In *children* the pulse-rate is greatly increased; it may be 200 per minute. It is small, unequal and irregular, but never intermittent. In *senile* pneumonia the pulse is not a reliable indication. Its average rate is 73 to 78; rarely does it reach 120. In old age, both in health and disease, the pulse has a fictitious hardness, on account of arterial rigidity. It may not be irregular or intermittent when the heart is, and *vice versâ*. Intermittence of the pulse in senile pneumonia is common, independent of any cardiac disturbance. *In all cases of senile pneumonia, the pulse should be counted at the heart.*

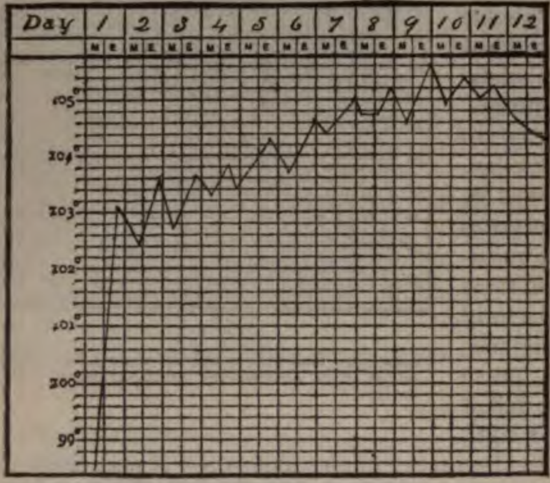

Fig. 28.

Temperature Record in a case of Acute Lobar Pneumonia passing into purulent infiltration and ending in Death on the 12th day.

The *skin* is often hot and dry until the crisis; but it may be bathed in perspiration from the onset. A moist surface is regarded as a favorable sign, but when, at the acme of the disease, the parched skin becomes moist and the patient is not relieved, it is an unfavorable symptom. In most cases, the *expression of the countenance* is characteristic. The face is anxious, and over the malar bones is a mahogany flush, not diffused as in typhus fever, but well defined and circumscribed; it is called the "*pneumonic spot.*" The rest of the face is pale.[1] Usually, one cheek is more flushed than the other; this is due to disturbance of the vaso-motor system. When there is a great disturbance of the circulation, or when vaso-motor disturbance is excessive, the lips become cyanosed. At the time of the crisis the lips become pale. In about 50 per cent. of cases, pneumonia is attended by an *herpetic eruption* upon the cheeks, nose, lips or eyelids. It is rare before the second or third day, and it may not occur until the crisis is reached. Herpes occurs with varying frequency in different years, and is more commonly met with in pneumonia than in any other febrile disease. One winter, nearly every case of pneumonia in Bellevue Hospital was accompanied by "herpes labialis." Sudamina may accompany profuse sweatings. In *children*, while the surface of the body is hot and dry, the extremities are cool, and the pneumonic flush is bluish or violet-colored. Cyanosis of the extremities is more frequent than in adults, and herpes labialis is more constant. All the cutaneous symptoms are exaggerated in children. *In old age* the pneumonic flush is often the first objective sign of pneumonia. The eyelids alone are cyanotic. If the face is at first dusky, it later assumes a sallow hue, and the surface heat is succeeded by a cold, clammy perspiration.

The cerebral symptoms are not very significant in the early stages of pneumonia. Headache is the first to occur, and may continue throughout the entire course of the disease. It usually diminishes after the third day. When severe in the evening there will be slight delirium at night, so slight as often to escape notice. Delirium and convulsions rarely occur except in the debilitated and in those of dissipated habits. It is most frequently met with in drunkards, and then assumes the character of delirium tremens. Sometimes in non-alcoholic pneumonia the delirium assumes an active, violent character. Whenever delirium is present it is important to make diligent search into the former habits of the patient. Pneumonia of the apex is oftenest accompanied by severe cerebral symptoms. The delirium may pass into coma. When delirium and headache are marked symptoms, muscular tremors ("subsultus tendinum") are very apt to occur with insomnia and frightful hallucinations. These cerebral symptoms occur so early and are so marked in alcohol drinkers that they mask the pneumonia; a physical exploration alone reveals the disease. When delirium is present in the weak and feeble it is of the low, muttering, "typhoid" type, and soon passes into a state of stupor. Photophobia, disturbances of vision, and deafness are rare. In *children* the cerebral symptoms are more prominent than in adults. Stupor and restlessness on the one hand, or headache, delirium and convulsions on the other, may usher in pneumonia in children, and they may rapidly pass into a semi-comatose condition. Convulsions are as common in children as they are rare in adults. They may be general, resembling those of epilepsy ("Eclamptic Pneumonia"), or they may attack single muscles or groups. Tetanus and opisthotonos are uncommon. Delirium and coma occurring late are usually followed by fatal coma. The cerebral disturbances often strikingly resemble those of acute meningitis. In *senile pneumonia* headache may persist throughout the disease. It is usually accompanied by mild delirium, especially when the pneumonia is at the apex. It is a busy, active delirium, and the patient has a constant desire to get out of bed.

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[1] Bouillaud regards the flush as best marked in pneumonia of the apex. Some regard the flush as best marked, or existing solely, on the cheek corresponding to the affected side; others as on the opposite side.—*Jaccoud.*

The symptoms referable to the *digestive tract* are not important. Nausea and vomiting are among the initial symptoms, and occur in about 75 per cent. of cases. At first the tongue is covered with a white fur; later it becomes dry. Anorexia is marked, and thirst is intense. The lips and tongue may become brown, dry and cracked, and sordes collect on the teeth. Diarrhœa may be an initial symptom; it usually accompanies nausea and vomiting. The bowels are usually constipated. In *children* nausea and vomiting are not only common, but in 25 per cent. usher in the pneumonia. They usually cease by the second day. Persistent diarrhœa often precedes death. In *senile pneumonia* the tongue early becomes dry, brown and shrivelled, and is protruded with difficulty. Although these patients may not complain of thirst, they drink with avidity when fluid is placed to their lips. Dysphagia is frequent. At the crisis critical diarrhœa is more frequent than critical sweats. Loss of strength occurs early, and is more marked in pneumonia than in any other acute disease except typhus fever. Recovery is rapid when convalescence begins.

The *urine* in pneumonia is scanty, high colored and of high specific gravity. The amount of urea and uric acid excreted is two or three times more than normal; it increases until the crisis, and then suddenly diminishes, falling below normal. Inorganic salts, chloride of sodium especially, are constantly diminished and may be wholly absent. Reappearance of the chlorides marks the approach of convalescence. At the crisis they are present in excess. Urea and uric acid are also sometimes retained in the system; and at the crisis there will then be a critical diarrhœa followed by prolonged convalescence. Bile pigment and sometimes the bile acids appear in the urine. Slight albuminuria is present in 35 per cent. of the cases. The severer the pneumonia the more marked the albuminuria.

Epistaxis may occur at any time, but is most frequent at the onset and at the crisis. Swelling of the veins of the hands in children is an unfavorable symptom. When pneumonia is to terminate fatally dyspnœa greatly increases, the patient suddenly "sinks," the pulse becomes small, rapid, intermittent and dicrotic; moist râles are heard in the larger bronchi or trachea, and there are physical evidences of pulmonary œdema. The sputa become frothy, liquid, and blood-stained ; they may be entirely suppressed. The respirations are more and more hurried, and the radial pulse becomes imperceptible. The face is sunken and livid ; the extremities become cold, and the capillary circulation more and more imperfect. The body is bathed in a profuse cold sweat. Death is usually preceded by a semi-comatose state. The temperature may steadily rise up to the time of death, or there may be "defervescence." Death may occur at any period of the disease. In alcoholic pneumonia death is preceded by active brain symptoms. In *children* death is often preceded by convulsions or coma ; sometimes exhaustion or collapse is most marked. Cyanosis and extreme rapidity of pulse are common in children before death.

Senile pneumonia may end fatally within a few hours after the onset in a most unexpected and quiet manner. In other cases sallowness of the skin, cold, clammy sweat, working of the auxiliary muscles of respiration, a feeble, rapid, irregular and intermittent pulse, and a sudden rise or fall of the temperature may precede the fatal issue.

Abscess.—Acute pneumonia terminates in abscess in 1½ to 2 per cent. of all cases. It is met with oftenest in debilitated weak subjects. The sputa are copious and fetid, yellowish in color, consisting almost wholly of pus. The fever is of the hectic type, and is accompanied by rigors and sweats. The patient grows weak and emaciated, death resulting from exhaustion, from asphyxia, or from discharge of the abscess into some neighboring cavity or organ.[1] The physical signs of the cavity are the most reliable evidences of an abscess. Abscess is rare in *children*. In *old age* there are no well-marked signs.

Gangrene as a termination of pneumonia has been found in about 14 per cent. of cases. This, however, is an exceptionally high percentage. Its occurrence is marked by signs of sudden collapse. The pulse is rapid, feeble and intermittent ; the face is pale and "death-like ;" there is profuse expectoration of blackish-green masses containing shreds of decomposed lung substance having a gangrenous odor. The breath is offensive and the body has a cadaverous smell. The sickening odor of pulmonary gangrene is most perceptible after coughing. Gangrene has its seat in the lower lobes of the lung, and it is here we must search for its ill-defined physical signs. In *old age* when pneumonia is to terminate in gangrene typhoid symptoms are present early and death occurs in collapse, usually within five days from the onset.

Purulent infiltration has symptoms that differ but slightly from those of the third stage of pneumonia. When resolution does not take place at the period of crisis and the temperature remains high, accompanied by symptoms of prostration and profuse purulent expectoration, purulent infiltration may be suspected. Somnolence and mild delirium are quite frequent during "purulent infiltration." The sputum contains a large number of cells in various stages of fatty degeneration. The fever has regular evening exacerbations, and it may range higher than at any other period in the disease. The tongue becomes brown and dry, and sordes collect on the teeth and mouth. Recovery is slow and convalescence tedious. Death results from exhaustion.

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[1] Fox and Green state that abscess is located preferably at the apex ; Da Costa says at the base.—Guy's Hospital Reports. Ser. VII. 1848.

Typhoid pneumonia is a term that has been applied to a pneumonia attended by typhoid symptoms. It has also been called "asthenic," "low," or "nervous" pneumonia. It is marked by extreme prostration that may exist from the onset. In the majority of cases, well-marked pneumonic symptoms, after having been present for a short time, soon give place to intense nervous prostration and adynamic symptoms. There is no sputa, no dyspnœa, no pain, no cough. Sordes collect on the teeth and gums. The tongue is thickly coated, and later, covered with black crusts. There is incontinence or retention of urine. The pulse is small and rapid. There is stupor, somnolence, and continual low, muttering delirium. This form is common in the *aged*. In some cases there is marked disturbance of the special senses—the speech being most affected. Tremor and subsultus tendinum are frequent. Typhoid pneumonia may be accompanied by glandular swellings, sharp and darting muscular pains, arthritic symptoms or vomiting. It is not infrequent in epidemics, and it may follow Bright's disease, erysipelas, alcoholismus, or phlebitis. Recovery is always possible, but is slow and tedious, and may not begin until the twelfth or fourteenth day. A modification of typhoid pneumonia sometimes accompanies dysentery, intestinal catarrh or phlegmonous gastritis. There is great sweating, profuse colliquative diarrhœa and high fever.

Bilious, or *gastric pneumonia,* is lobar pneumonia occurring in malarial districts, and accompanied by gastro-enteritis with hepatic symptoms. It is sometimes called "malarial pneumonia." It has the characteristics of a severe pneumonia, but the fever is paroxysmal. The tongue is heavily coated; nausea and vomiting are common and may be persistent. The epigastrium is distended and tender, the skin more or less jaundiced; the liver is enlarged, and there is constipation or exhausting diarrhœa; the latter is accompanied by greenish-black, viscid and inodorous discharges. "Bilious" pneumonia may be sthenic or asthenic; but prostration is apt to be nearly as marked as in the typhoid variety. The symptoms of bilious pneumonia have frequently led to the diagnosis of "*typhoid gastric fever*." It runs a much more protracted course and has a much longer period of convalescence than the typhoid variety; vomiting is "bilious," and somnolence and stupor may indicate a fatal issue.

Latent pneumonia seldom occurs in adults unless it complicates some disease whose symptoms are so severe that the pneumonia is obscured. Inter-current *senile* pneumonia is always latent; and Grisolle states that a physical exploration gives negative results in the majority of instances. Senile pneumonia may run its course without expectoration, dyspnœa, flushed face or physical signs. Its diagnosis is then difficult. It is to be remembered that of all phlegmasiæ of advanced life, pneumonia is the most frequent; and of all the acute diseases of advanced life it causes the highest temperature range and the greatest prostration. When an old person has a slight rigor, followed by a febrile movement attended by great prostration for which there is no explanation, pneumonia may be suspected even though all its usual signs are absent.

Intermittent pneumonia, which is by some described as a distinct type, is a form of acute pneumonia in which a malarial element is so pronounced that all the subjective and even the physical signs undergo distinct intermissions, returning each day with increased severity. It may assume the quotidian or the tertian type. During the intermission the temperature may fall to the normal. Recurring chills and sweats are often present; and the pneumonia is not infrequently double. By some it is regarded as peculiar to old age; it is very rare at any other period. Those malarial influences that give rise to this type of pneumonia are more frequently met with in our Southern and Western States than in any other part of the world.

Physical Signs.—*First Stage, or Stage of Congestion.*—The physical signs indicative of the first stage of lobar pneumonia are usually present within twenty-four hours after its invasion. If the pneumonia commences in the central portion of the lung their appearance may be delayed till the third day. By studying these signs in connection with the anatomical stages of the disease their importance in diagnosis and prognosis can best be appreciated.

Inspection.—The movements of the affected side are more or less restricted. The unaffected side moves as in health. In double pneumonia the respiratory movements will assume the *costal* type, attended by increase in the abdominal breathing.

Palpation.—There is more or less marked increase in the vocal fremitus over the affected lung; the degree of increase corresponding to the extent of the congestion.

Percussion.—There is slight dulness over that portion of the chest which corresponds to the affected portion of the lung. It is not well marked until the end of this stage, although the pulmonary capillaries are engorged with blood from the very first. Even at the end of this stage there sometimes remains a slight tympanitic note. Very extensive central pneumonia may fail to give any signs until the second stage is reached. Absolute dulness in this stage is very rare.

Auscultation.—During the "dry" stage—which, according to some, precedes the exudation—there will be noticed a feebleness and unnatural dryness of the respiratory murmur. This murmur is sometimes harsh, sometimes weaker than normal, losing the "breezy," rustling quality of normal breathing. Elsewhere it is exaggerated. As soon as the congestion is well marked, fine crackling sounds are heard at the end of inspiration—"*crepitant râles*"—which have been regarded as characteristic of this first stage, but which are usually pleuritic crepitation. These sounds resemble those produced by throwing salt on hot coals or rubbing the hair between the fingers. They are as numerous as they are minute, are unaffected by coughing, and remain audible for from twelve to twenty-four hours. This râle is of an unvarying character, and continues, *i. e.*, is not inter- or remittent. If the pneumonic stages succeed each other in rapid succession, the crepitant râle may not be heard. They are rare in pneumonia developed with acute articular rheumatism. The respiratory murmur is feeble or assumes a broncho-vesicular character. Bronchial breathing *may* be heard in this stage (*Traube*). The voice sounds are slightly increased in intensity over the engorged spot. In *children* the "pneumonic crepitation" is usually absent; and though it may be heard at the end of a full inspiration after crying, it is never as fine or as distinct as in adults. There will be no marked increase in vocal fremitus. In *old age* the physical signs are modified by a more complete bony union of the chest walls, by curvature of the spine, rigidity of the bronchial tubes, by the rounded form of the chest, and by senile rarefaction of the lungs.

Second, or Stage of Red Hepatization.—The physical signs of this stage are more diagnostic than those of either of the other stages.

Inspection shows the expansive movements of the affected side more markedly diminished than in the first stage; while those of the other side are increased. There may be absolute loss of motion over the affected lung.

Palpation.—There is usually marked increase in the vocal fremitus over the consolidation. In some instances this is so slight that no difference can be detected. Very rarely it is *less* than on the normal side. The heart may be slightly displaced. Rarely can pulsation be felt over the inflamed lung. The majority of authorities regard this pulsation as due to increased pulsation in the arteries of the inflamed spot, but there is no reason to doubt that the cardiac impulse itself can be transmitted through the solidified lung as well as the arterial impulse or the vibrations from the *chordæ vocales.* In central pneumonia, vocal fremitus may be normal. Pleuritic effusions mask the signs.

Percussion.—There is marked dulness over that portion of the lung which is the seat of the pneumonia. Over the unaffected lung there is exaggerated resonance. The nearer the hepatization to the surface the more marked the dulness. There is a sense of resistance accompanying the percussion. A pneumonic lung is more resistant than any other form of consolidated lung. When an entire lobe is consolidated its exact outlines can be defined. The percussion may have a tympanitic quality anteriorly, but there will always be dulness posteriorly. There may be slight tympanitis just around the pneumonic spot. When an entire upper lobe is consolidated a tympanitic percussion sound may be caused by vibration of the air in a large bronchus. The "cracked-pot sound" is occasionally met with in pneumonia over the relaxed and permeable parts of the lung in the immediate vicinity of the consolidation. When heard over the condensed portion it is caused by the sudden expulsion of air from the large bronchus. This occurs most frequently in the young with thin chest walls. In basic pneumonia the percussion note under the clavicle of the affected side may be amphoric.

Auscultation.—As soon as the air-cells are completely filled with the pneumonic exudation the crepitant râles cease, and bronchial respiration is heard over the affected lung. It often has a metallic character; or it may sound like tearing a piece of linen. Bronchial respiration is more intense in pneumonia than in any other disease.[1] At the commencement of the second stage tubular breathing attends expiration only. Later, it accompanies both acts. Pleuritic exudation may mask the auscultatory signs. Plugging of a large bronchus will prevent tubular breathing; a violent fit of coughing may allow it to occur when the mucus is dislodged. The voice sounds are increased in intensity, and bronchophony is heard over the whole of the consolidated lung. Bronchophony has the same diagnostic significance as bronchial respiration because it is produced by the same physical condition of the lung. When the pleural cavity is partly filled with fluid, bronchophony is indistinct or absent below the level of the fluid; while at the level the voice sounds may be ægophonic. Pectoriloquy may be heard independent of fluid in the pleural cavity. The heart sounds are abnormally intense. In *children* dulness is especially marked in the infraclavicular region. Some speak of a feeling of greater solidity below than above the scapula which can be perceived before the ear can detect dulness on percussion. Vocal fremitus *may* be increased, but this is not always to be expected. In *old age, inspection* and *palpation* give negative results. What is dull on percussion in old age might be regarded as resonant in

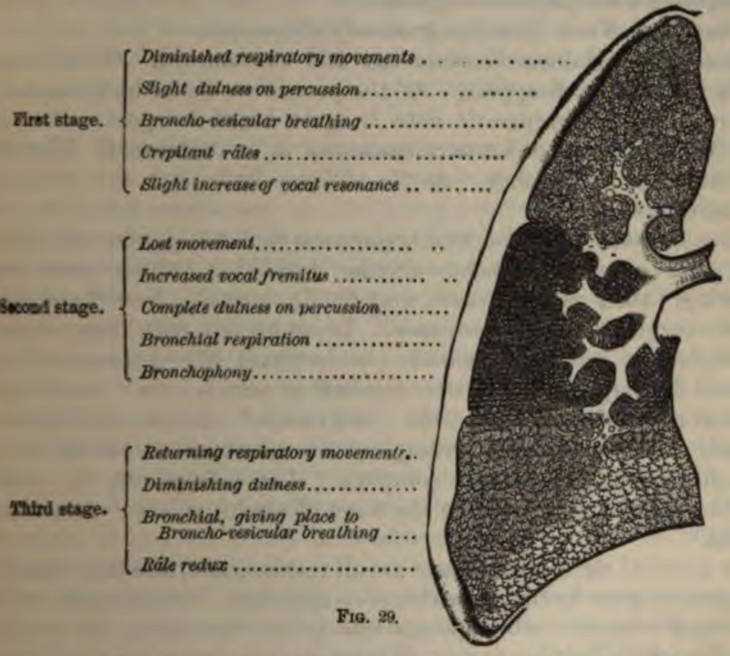
First stage.
Diminished respiratory movements
Slight dulness on percussion
Broncho-vesicular breathing
Crepitant râles
Slight increase of vocal resonance
Second stage.
Lost movement
Increased vocal fremitus
Complete dulness on percussion
Bronchial respiration
Bronchophony
Third stage.
Returning respiratory movements
Diminishing dulness
Bronchial, giving place to Broncho-vesicular breathing
Râle redux

FIG. 29.

Diagram Illustrating the Physical Signs in the Three Stages of Lobar Pneumonia.

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[1] Laennec taught that bronchial respiration was due to the superior conducting power of condensed lung. Skoda combats this view, and says that bronchial respiration is generated or magnified in caverns and in the bronchi of condensed lung substance by the air in these cavities and bronchi vibrating in consonance with that of the trachea; the condition necessary for this consonance is provided in the circumstance that the air is pent up in confined spaces whose solid walls reflect the sonorous undulations.

adults. Hence dulness on percussion is a relative term in senile pneumonia. When the pneumonia is superficial there is actual dulness. Tubular or bronchial breathing marks the second stage, and is even more intense than in adult life. Small gurgles or mucous râles are heard in this stage. Bronchophony is not very common, and never distinct. Ægophony is frequent. By causing the aged patient to cough and expire violently tubular breathing may be heard.

Third, or Stage of Gray Hepatization.—There is no abrupt transition from the second to the third stage; the physical signs of early gray hepatization are the same as in the second stage.

Inspection.—As resolution progresses, expansive motion on the affected side becomes more and more apparent.

Palpation.—Vocal fremitus gradually diminishes.

Percussion.—Dulness becomes less and less marked. Of all the signs this is the last to disappear. As the percussion sound becomes more and more resonant the tympanitic note is again heard in spots. It is a long while before normal pulmonary resonance is re-established. The dulness may disappear in patches. As the dulness diminishes the pitch of the percussion note rises.

Auscultation.—The bronchial respiration that was present in the second stage gives place to broncho-vesicular breathing, which soon becomes "blowing," then *indeterminate,* and finally normal. Bronchophony gives way to exaggerated vocal resonance. In connection with these changes in the respiratory and vocal sounds the crepitant râle returns, but is soon obscured by larger and moister crepitating sounds—the "resolving sub-crepitant râle" of pneumonia—the "*râle redux.*" Large and small mucous, sibilant, and sonorous râles accompany the sub-crepitant sounds, to disappear only when resolution is complete. Not infrequently the bronchial râles that are developed during the stage of resolution are "consonant" or ringing.[1]

The physical signs of this stage are all retrogressive, and they disappear in the opposite order to that in which they appeared. In rare cases resolution is so rapid that the sub-crepitant râle is not developed. In this class of cases bronchial breathing and dulness on percussion continue for some time after the crisis.

If the consolidated lung becomes the seat of *purulent infiltration,* the temperature remains high and symptoms of great prostration are developed. Bronchial breathing continues, and becomes more intense, dulness persists, and when râles occur they are high-pitched, sharp, and resemble fine gurgles. The occurrence of *abscess* and *gangrene* is indicated by the physical signs which attend the formation of a cavity in consolidated lung substance. No one of the physical signs present during a pneumonia is sufficient for a diagnosis; but the manner and order of their occurrence and their relation to the subjective symptoms enable one to reach a positive diagnosis in all typical cases. The only diagnostic symptom is the *sputum.*

In *children* bronchial breathing rarely disappears before the seventh day;

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[1] Skoda and Traube.

it is often accompanied by the sub-crepitant râle. When resolution takes place, bronchial breathing and the sub-crepitant râle will disappear simultaneously. If *purulent infiltration* occurs, large gurgling crepitation will be heard. Vesicular breathing is rarely heard before the eighth or ninth day.

In *old age*, inspection, palpation and percussion give similar results to those in adult life. *Auscultation* shows the crepitating sounds to be louder, and gurgles large and loud are often heard at a distance from the chest. The *râle redux* is not distinctive of, or peculiar to the third stage of senile pneumonia. The sound heard at this stage is a muco-crepitating sound, *i. e.*, a sound produced in bronchi of medium size. The physical signs of pulmonary abscess in the aged are very generally wanting. Distinctly localized gurgling and cavernous respiration may, with the rational signs of abscess, suffice for a diagnosis. The sputa will also aid, but the diagnosis is only approximate. In old age the physical signs are subject to greater variations than in adult life.

Differential Diagnosis.—Lobar pneumonia may be confounded with *pulmonary congestion and œdema, capillary bronchitis, pleurisy, hypostatic congestion, catarrhal pneumonia* (in children), *pulmonary infarction, acute phthisis* (especially in children), *meningitis* and *typhoid fever*.

Pneumonia begins with a chill, followed by a rapid rise in temperature and pain in the side; in pulmonary *congestion and œdema*, there is no chill or rise in temperature, and no pain. The sputum of pneumonia is viscid, rusty and (microscopically) diagnostic; in pulmonary congestion and œdema there is profuse watery, blood-stained expectoration. Pneumonia is commonly unilateral, and may occur in any portion of the lung; pulmonary œdema is bilateral, and usually occurs in the most dependent portions of the lungs. In pneumonia there is complete dulness on percussion, crepitant râles and bronchial respiration; in pulmonary œdema the dulness is not complete, there is no bronchial breathing, and there occur numerous large, liquid, sub-crepitant râles.

The resolving stage of pneumonia may be mistaken for *acute capillary bronchitis;* but in the latter the sub-crepitant râle is heard all over the chest; while in pneumonia it is usually limited to a small area. The expectoration is muco-purulent in bronchitis, and the temperature range is lower than in pneumonia. There is no dulness on percussion, no bronchial breathing in capillary bronchitis; the vesicular murmur is feeble, and cyanosis is more marked. The breathing is labored in bronchitis, and panting in pneumonia.

Pneumonia is ushered in by a distinct chill followed by fever; *acute pleurisy* begins with chilliness or several rigors, and the temperature rarely rises above 100° F. The dry, hacking cough of pleurisy is accompanied by slight mucous expectoration, and the characteristic pneumonic sputum is absent. In pleurisy the face is pale and anxious, and the pulse is firm, small, tense and wiry; in pneumonia the face has a mahogany flush, and the pulse is full and compressible. The breathing in pleurisy is "catching;" in pneumonia it is "panting." There are no critical days in pleurisy. Vocal fremitus is diminished or absent in pleurisy with effusion, there is flatness on percussion, and the sound of the percussion changes with a change in position of the patient. In pneumonia vocal fremitus is increased, and there is dulness—not flatness—on percussion. In pleurisy the respiratory sounds are feeble, and a grazing, rubbing or sticky friction-sound is heard; in pneumonia there are crepitant râles and bronchial breathing. Bronchophony and bronchial breathing may exist in pleurisy, but they are always diffuse—never sharp and tubular as in pneumonia.

Hypostatic congestion is accompanied by copious, *watery*, blood-stained expectoration; it occurs in the most dependent portions of the lungs, disappears when the patient sits up, and is accompanied by no rational symptoms except dyspnœa and expectoration.

Lobular pneumonia in children is always secondary; it is not ushered in by a chill, usually follows a bronchitis, and is developed in both lungs. There are no days of crisis, and the physical signs of pneumonia are limited to circumscribed spots. The range of temperature in the two forms of pneumonia differ; the two curves represented by Figs. 20 and 25 show the differences.

Pulmonary infarction is rarely met with independent of cardiac disease or pyæmia. It is a non-febrile disease, and intense dyspnœa, coming on abruptly, is its prominent symptom. In pneumonia dyspnœa comes on slowly. The expectoration in infarction consists of small black coagula; in pneumonia it is viscid and contains few blood-globules. The dulness of an infarction is circumscribed, and around it moist râles are heard; in pneumonia the area of dulness is extensive, and there are no moist râles. There is a peculiar garlic-like odor to the breath, in pulmonary infarction, never present in pneumonia.

When lobar pneumonia has its seat at the *apex*, it may be confounded with *acute phthisis*. But the history of a well-marked chill, followed by the characteristic pneumonic symptoms, will enable one to exclude phthisis. Moreover, the fever in phthisis is subject to irregular exacerbations and remissions. If the signs of consolidation persist with little or no change, if the temperature at no time falls to normal, if there are night sweats, if emaciation is progressive—then the case is to be regarded as one of phthisis, even though pneumonia may have complicated it.

In children pneumonia is so often accompanied by cerebral symptoms that it may be mistaken for *meningitis*. Meningitis comes on insidiously, the temperature rarely rises above 103° F., the pulse is often lower than normal, there are no thoracic symptoms, no dyspnœa, the face is pale and anxious, and the physical signs of pneumonia are absent.

Sometimes latent pneumonia may be mistaken for *typhus fever*, especially when typhus is prevailing. While in charge of the typhus fever patients on Blackwell's Island, I frequently saw cases where such a mistake had been made during a typhus epidemic. In these cases there will be dry tongue, delirium, and high temperature. The countenance resembles that of pneumonia, but the presence of the typhus eruption and the absence of the physical signs of pneumonia will establish the diagnosis.

Pneumonia with typhoid symptoms is sometimes mistaken for typhoid fever. The differential diagnosis is not difficult, if one remembers that the pneumonia which complicates typhoid fever does not come on until late in the fever, and the regular history of typhoid fever precedes its development. On the other hand, when the typhoid symptoms are present from the beginning, or come on at the end of the second stage of pneumonia, the physical signs of pneumonia will precede the typhoid symptoms. If a patient over sixty years of age, with this type of pneumonia, is not seen until the second or third week of his sickness, although evidences of lung consolidation may be found, it will be very difficult to decide whether the pneumonia is or is not complicating a typhoid fever, and under these circumstances the diagnosis will be difficult if not impossible.

Prognosis.—The phenomena of the crisis of pneumonia are a sudden fall of temperature followed by profuse sweats and a diminution in frequency of respirations and pulse. The cough becomes loose, the dyspnœa abates, the flush disappears from the face, the sputum is more copious, loses its rusty hue, diminishes in viscidity and becomes "creamy," thin and watery. Thirst decreases, the appetite returns, pain ceases and the patient falls into a quiet sleep, waking extremely exhausted. Epistaxis, hæmaturia and hemorrhage from the bowels may occur at the crisis. After the crisis the amount of *urea* in the urine, which was augmented before, becomes normal and the chloride of sodium reappears. The crisis in *children* is marked by a greater fall in temperature and by a more profuse sweat. When children have been restless or delirious the crisis is marked by a state of stupor. In *old age* the crisis is marked by a critical diarrhœa rather than by a sweat.

The fatality of pneumonia is shown by the following statistics: of 12,421 cases treated in the hospitals at Stockholm, 11 per cent. died. In the Vienna hospitals 24 per cent. died. The Basle Hospital Reports for thirty-two years give 23 per cent. of deaths. Grisolle reports 59 per cent. of deaths in those over sixty. In the "U. S. Medical Reports," May 1st, 1861, to July, 1866, of 61,202 cases which occurred among the white troops, 14,738 died—more than 24 per cent.; and of 16,133 among colored troops, nearly 33 per cent. died. The deaths from all other inflammatory diseases of the respiratory system for the same time were only one-seventh as many as from pneumonia.[1] Of 255 cases treated in Bellevue Hospital during a period of four years the rate of mortality was 34 per cent. The statistics of private practice are very different: of Lebert's 205 cases, only $7\frac{3}{10}$ per cent. died. Ziemssen lost only $3\frac{1}{3}$ per cent. of his cases. Bennet lost none of his 105 cases. (He says, however, that no complications existed.) Brundes, of Copenhagen, lost over 21 per cent. of his 142 cases. Fox gives to pneumonia the fifth, and Walshe the third place among fatal diseases. The average mortality-rate from all the published reports to which I have had access gives $20\frac{1}{10}$ per cent. of deaths. But the rate varies in different years.

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[1] The Confederate Hospitals' Reports give over 30 per cent. of deaths from pneumonia for the same period.

The prognosis depends more on the age than on any other single element. In infancy the mortality is greater than in early childhood. Between the ages of forty and sixty the death-rate is from 10 to 25 per cent., while from ten to thirty years almost all of the uncomplicated cases recover. After sixty the prognosis is always unfavorable. Pneumonia is the most fatal of all acute diseases at this period of life; most "sudden deaths" in the old are from acute lobar pneumonia. Some of the most reliable modern authorities state that nine-tenths of deaths after the seventy-fifth year are from acute pneumonia. It is more fatal in females than in males. In some years the proportion of deaths is far greater in summer than either in the spring or winter; and certain—as yet unknown—atmospheric influences are of the utmost importance in determining the death-rate. The extent of lung involved influences the prognosis; double pneumonia is rarely recovered from. When an entire lung is involved, the prognosis is not as good as when only a single lobe is involved. Apical pneumonia—especially in the old and very young—is more often fatal than basic. The feebler the patient the more unfavorable the prognosis.

Complications render the prognosis unfavorable: of 225 of my own cases, 87 were fatal and 168 recovered. Of these, 124 were complicated and 131 uncomplicated. Of the complicated cases, 75 died; of the uncomplicated, 12 died.[1] The most dangerous complications are those which exert a direct influence on the heart, diminishing its power and obstructing the flow of blood from the right ventricle. Acute infectious diseases are dangerous complications because they hasten heart failure.

Pneumonia may be regarded as mild when the temperature is below 104°. When the fever ranges above 106° for two days, the case is unfavorable. A gradual rise in temperature after the fourth day is always an unfavorable sign. A low temperature is dangerous only when the respirations are greatly accelerated. When the pulse is 120 to 130 for two or three days, the prognosis is bad. If the pulse reach 150 per minute, or if it becomes irregular, intermitting, or dicrotic, the patient rarely recovers. In children a rapid pulse is of less significance, and in old age the pulse is never a reliable guide. Prune-juice expectoration is an unfavorable sign, indicating extensive blood changes. When expectoration is absent in the second or third stage, or if it become scanty and difficult, the prognosis is unfavorable. Sudden suppression of the sputa, with coincident tracheal râles, indicates impending death. Delirium coming on after the sixth day, convulsions in *children*, with jactitation and subsultus, or, in the *aged*, a tendency to coma, are unfavorable signs. Exhaustion and prostration, accompanied by a sunken pallid face and cold, clammy sweat, are always dangerous. In children, bronchial breathing, after the seventh day, numerous subcrepitant râles, copious and persistent diarrhœa, and swelling of the veins of the hands are bad symptoms. In old age a sudden rise or fall in temperature, apathy, somnolence, and a sallow, anxious countenance are dangerous symptoms. Pulmonary congestion and œdema in the unaffected part of the lung often precede a fatal issue. The occurrence of purulent infiltration, abscess, or gangrene renders the prognosis unfavorable.

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[1] Lebert states that he lost only 5⅙ per cent. of his uncomplicated, and *all* of his complicated cases. Huss lost 6 per cent. of uncomplicated and 20 per cent. of complicated cases. Fox states that pneumonia complicated by endocarditis is fatal in 75 per cent. of cases; by pericarditis, in 54 per cent.; by Bright's disease, in 50 per cent.; and by alcoholismus, in 25 per cent. Brundes (of Copenhagen), in 120 uncomplicated cases, lost 6⅗ per cent.; of 22 complicated, he lost *all*. The danger of complications is markedly shown by these statistics.

In pneumonia the fibrin-factors of the blood are increased (often 400 per cent. more than normal), the heart-power is diminished, so that the ventricles cannot empty themselves, the columns and cords whip up the residual blood (already prepared for clotting), and "heart clots" always form when the death struggle is prolonged and cardiac contractions feeble. The "heart failure" is the beginning of death. Post-mortem results can never give all, or the true causes of death, but only the modes of death. If, on account of heart failure, pulmonary œdema and congestion occur and heart clots form, these clots cannot be called causes of death. Jürgensen states that in fatal cases of pneumonia œdema of the lungs is probably *always* present, and heart clots are frequent. Death may occur, then, from heart-insufficiency, from complications (cardiac especially), or from asphyxia. Fatal collapse may follow an apparently regular and well-marked crisis.

Treatment.—If we regard pneumonia as a *general* disease with a characteristic local lesion, the treatment must be modified by the constitutional condition of each patient and by the type of the pneumonia. If it is uncomplicated and occurs at certain periods of life, it will terminate spontaneously in recovery by crisis ; but when certain complications exist, when certain conditions are present, and at certain ages, it is almost necessarily fatal.

Any plan of treatment in such a disease, if resorted to indiscriminately, will prove unsatisfactory. Although a large proportion of cases will recover without treatment, yet well-directed therapeusis will save lives and render convalescence less tedious. The pneumonic lung no more requires treatment than the intestinal ulcers of typhoid fever. It is the *general* condition of the patient, not the *local* changes, which is to govern us in the management of each case. Agents for the arrest of local inflammation have no place here ; hence *venesection*, once generally practised, has been almost entirely abandoned. A careful study of the pathology of pneumonia not only leads to the conclusion that bleeding does harm, but it strongly contra-indicates the use of all those agents which have been employed for the arrest of simple pulmonary inflammation. Hence veratrum viride, aconite, antimony, calomel, the tartrate of potash and antimony, iodide of potassium, and all so-called "cardiac sedatives" have been discarded, for it is evident that they add a new load to an already overburdened heart. They may, for a time, lower temperature and pulse-rate, but this will be accomplished at the expense of heart-power. Cardiac insufficiency will therefore appear earlier and be more profound.

Counter-irritation by blisters, or other irritants, to the chest (in the early stages) is apt to do harm ; but blisters may be applied during the third stage, to hasten resolution. The application of leeches, followed by a linseed-meal poultice or other soothing fomentation, will relieve the pain in the side, which is often so urgent at the onset, and, if the condition of the patient will allow, may be of service. If extensive pulmonary œdema occurs, dry cups applied to the chest will relieve the dyspnœa, and for a time dispel the œdema. It has come to be a rule to incase the chest in a cotton-batting or flannel jacket, covered with oiled silk. This has no influence over the course of the pneumonia, but it promotes diaphoresis, protects the surface from sudden changes of temperature, and it is always grateful to the patient. The "jacket" is especially beneficial in children.

Absolute rest is important; the patient should be moved as little as possible, and should not be kept in a constrained posture. If signs of heart failure occur, he should not be allowed to sit up or talk. The sick room should be large, cheerful and well ventilated, and its temperature should range between 65° and 70° Fahr. A most important adjuvant is a carefully-regulated diet. The food should be fluid or semi-fluid, and highly nutritious, *e. g.*, milk, eggs, beef-tea, and concentrated broths. Milk is preferable to all other nourishment.

The *nervous shock* which attends the onset of acute lobar pneumonia is greater than in any other acute disease, except, perhaps, acute peritonitis, and the important question presents itself: what measures shall be employed to counteract, or mitigate, the impression made on the nerve centres by the morbific agent which is operating to produce the pneumonia? The experience of the last five years leads me to the conclusion that during the developing period of the disease, when the pneumonic blow is first struck, and until the infiltration is complete (usually for the first four days), the patient is to be brought under the full influence of *opium* and held in a state of comparative comfort, by hypodermic injections of morphia, repeated at regular intervals; and that by this course a pneumonic patient is placed in the best condition, not only for sustaining the primary shock, but for resisting the pneumonia. Thus given, opium does not interfere with the employment of any stimulating or anti-pyretic measures which may be demanded. And not only does it diminish the chances of the occurrence of heart failure, but the great relief and comfort which it gives to the sufferer in the first four days of his struggles are sufficient to commend its use. After the pneumonic infiltration is completed, opium should be discontinued, for paralysis of, and a consequent accumulation of secretion in the bronchi may greatly increase the already deficient respirations.

In all severe types of pneumonia there are two prominent sources of danger—*heart-insufficiency* and *high temperature*. The two prominent indications for treatment are, therefore, to *sustain the heart* and to *reduce the temperature*. A large proportion of deaths in pneumonia directly result from heart failure; alcohol, judiciously used, is the most efficient means for preventing or overcoming it, but its indiscriminate use is more dangerous than indiscriminate bleeding. Only a few ounces of brandy may be required to carry a pneumonic patient through a critical period; or its free administration may be demanded to save life. In the old and feeble, and in those who have been accustomed to the use of alcohol, stimulation may be necessary from the very onset. The indications in each case demand careful study; in no disease is so much discretion required in the administration of stimulants. The pulse is the indicator of the heart's condition. A frequent, feeble, irregular or intermitting pulse calls for stimulants. The quantity required in any case is to be determined by its effects on the pulse. It is best to begin with small quantities, and carefully note the effect of the first few doses. If it acts beneficially, a favorable effect will be seen in a few hours; and then the quantity administered must be varied to suit each case. It is seldom necessary to use more than six or eight ounces of brandy in twenty-four hours; but, when demanded, it is to be unsparingly given. A dicrotic pulse is always an indication for its use. The period immediately following the crisis is the time when stimulants are usually most required. Delirium, muscular tremor and subsultus are indications for their use. Critical collapse in the aged must be combated by a very free use of stimulants. Carbonate of ammonia is extensively employed as a stimulant in pneumonia;—it is claimed that its use diminishes the danger of heart clot, but there is no evidence in support of this statement;—and if given in sufficiently large doses to act as a stimulant it irritates the stomach. It is unquestionably inferior to alcohol as a cardiac stimulant. Camphor and musk are also inferior to alcohol, and digitalis is only of service when there are evidences of extensive renal congestion.

There are two plans of treatment advocated for reducing temperature in pneumonia: (1) the application of *cold* in various ways to the surface of the body; and (2) the internal administration of some one of the antipyretics. It is claimed that the temperature can be reduced by applying cold compresses to the chest; a cloth of some thickness is to be wrung from cold water and applied every ten or fifteen minutes to the affected side. This not only relieves the local symptoms, but it lowers the body temperature and hastens the day of crisis. Some prefer the "Esmarch ice-bag" to the cold compress. When cold is used at all, I prefer the coil. There is no doubt but that the pain in the side and fever will be relieved by this means, but the relief is only temporary; and my own experience leads to the belief that pneumonia treated in this way is more likely to extend, and that there is great danger of chilling the patient. The other methods of applying cold to the surface for the reduction of temperature in pneumonia are the cold bath, the cold pack, and cold sponging.

The experience of American practitioners is against the cold bath and the cold pack. The shock of cold causes depression, which the feeble or old cannot rally from. And though fever is lessened, heart-failure more rapidly follows, and is more difficult to overcome. My own experience is decidedly against the use of cold on the surface for the reduction of temperature in pneumonia. Cold "sponging" may be practised when it is grateful to the patient.

Of the internal antipyretics, antipyrin and antifebrin are largely employed. Of these, antifebrin is to be preferred. In many instances it will effect a reduction of temperature not obtainable by other antipyretics. In the majority of cases of pneumonia, however, sulphate of quinine is more efficacious than either of the newer drugs. To act antipyretically it must be given in doses of from gr. x. to gr. xx. within a period of not more than two hours.[1] The very large antipyretic doses of quinine which have been recommended seem to me to be attended with danger, for in such large doses it appears to act as a cardiac depressant, and I believe that with gr. x.–xv. given in one dose we obtain as certain an antipyretic result as with much larger doses. No one of the antipyretics should be long continued or given in large doses.

If there is great restlessness or wakefulness during the third stage, hydrate of chloral, or, better, small hypodermatics of morphia can be given. If there is even slight evidence of cyanosis, these remedies should be used with great care. When the pupils are small, belladonna or hyoscyamus may be given. For the relief of the distressing cough which is sometimes present, five grains of hydrate of chloral combined with one-twentieth of a grain of morphia, or twenty-five drops of chlorodyne every two hours, may be given. If expectoration is difficult from loss of muscular power, stimulating "expectorants," such as senega and turpentine, are useful. But if this difficulty arises from great viscidity of the sputum, alkalies will be found of service, and, as alkalies and neutral salines also have a diuretic and diaphoretic action, they are especially indicated just before the crisis. For the relief of the delirium of chronic alcoholism, tartar emetic and digitalis are highly recommended by English authorities.

In the first stage of senile pneumonia an emetic, when not specially contra-indicated, is given in the "Salpêtrière Hospital." Ipecacuanha is regarded as especially indicated. The nitrate of potash and the hydrochlorate of ammonia are also highly recommended in *senile pneumonia.*

In children the chest should be thoroughly protected, the diet carefully regulated. Leeching and blistering are both harmful, and should never be employed. Stimulating expectorants are often indicated, and the moderate use of stimulants in feeble children is always required. During convalescence, iron, quinine, mineral acids, cod-liver oil, and blood-making wines, should be given.

Very recently there has been advocated an ***antiseptic treatment*** of pneumonia, based upon the recognition of the specific character of the disease. Such treatment cannot be supposed to affect the local etiological element in the lung, but may have the effect of neutralizing poison elements in the blood and thus reducing the constitutional manifestations. It cannot be denied that a septic element exists in some if not in all cases;

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[1] Liebermeister gives *quinine* until the temperature has been reduced by it to within ½° of the normal. Few American practitioners carry the antipyretic effects of quinine so far. In Ringer and Gill's experiments upon "*The Influence of Quinine on Temperature*" in health, it took at least gr. xx. to produce a fall of 1°. From fifty to eighty minutes elapsed before the fall occurred, and the effects lasted from forty-five minutes to three hours. Ringer states that in pneumonia (and some other diseases) quinine does not readily pass out with the urine, but is delayed in the system for considerable time, and its antipyretic effects are continued longer than in other diseases. Prof. Flint states that he has seen pneumonia rendered *abortive* in a certain proportion of cases by xx. to xl. grains of quinine daily, and even when this result has not followed, the disease has often been favorably modified in a greater degree than by smaller doses.

hence the sulphites and hyposulphites (20 grain doses every three hours) are recommended. Carbolic acid, from 1 to 5 grains, sulpho-carbolate of soda (5 to 20 grains every two hours), have both been used quite extensively as antiseptics in the treatment of pneumonia. Thymol and salicylic acid have risen into favor because they are powerfully antiseptic and are almost physiologically inert. Quinine has also been advocated for its antiseptic power. The antiseptic treatment of pneumonia has not yet assumed a definite aspect or been sufficiently tried for any definite statements to be made concerning it.

LOBULAR PNEUMONIA.

Lobular, *catarrhal*, or *broncho-pneumonia*, is always secondary, being preceded by, or associated with, inflammation and obstruction of the smaller bronchi, which lead to the consolidated lobules. It may run an acute, subacute or chronic course, and differs very decidedly both in its clinical and pathological history from acute lobar pneumonia.

Morbid Anatomy.—The anatomical changes in lobular pneumonia are confined to scattered groups of air-vesicles, hence the gross appearance of the portion of lung involved will vary with the duration and extent of the pneumonic process. In well-marked cases there will be found scattered throughout one or both lungs, small, circumscribed nodules of a light, deep-red, or bluish color, which do not inflate when the lung is inflated. If they are situated near the surface of the lung, they cause small, rounded elevations. When they are of considerable size, a reddish fluid oozes from their cut surfaces on section, and a small quantity of dark blood can be pressed from them. They are less tough than healthy lung substance, and break down easily on pressure. These nodules shade off into the surrounding zones of lung-tissue, which may be the seat of œdema, congestion, or emphysema. The nodules vary in size from that of a pea to that of a hazel nut, and are very rarely granular.[1] When the lung is inflated these spots of consolidation are rendered more prominent, so that they stand out sharply defined from the adjacent lung-tissue. In some instances these isolated spots of consolidation become confluent and involve a large portion of lung—perhaps a whole lobe—and become pale, firm, and dry, resembling in color the gray hepatization of lobar pneumonia. The smaller bronchi are congested ; their walls are often thickened, and they may contain a thick, tenacious, puriform secretion which, later, may become dry and inspissated. When a *section* of the lung is made they often stand out prominently or even rise a little above the level of the cut surface. A *peri-bronchitis* is very often associated with these changes. Cylindrical and fusiform dilatations of the tubes are not infrequent. Often, when a small patch of consolidation is cut across, there will be found at its centre a dilated bronchiole filled with pus. Discoloration begins at this point and extends toward the periphery. The connective-tissue of the portion of lung involved is increased, and this, in long-standing cases, is often pigmented. Bronchiectasis may occur at various points.

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[1] Jürgensen says : "Granulation is *never* observed."

A *microscopical* examination of an affected lobule may distinguish three stages in the inflammatory process. First, the air vesicles may be more or less completely filled with pus and serum, containing swollen and granular epithelium. The capillaries in the walls of the air vesicles are usually elongated, and red globules may escape into the air-sacs. In the unaffected portions of lung-tissue the epithelial cells appear large and more distinct than in healthy lungs. In the *second* stage the affected lobules become solid and *airless*. Their color changes to a pinkish gray. The other changes are similar to those that take place in the stage of red hepatization in lobar pneumonia, except that less fibrillated fibrin is found in the exudation, the pus and epithelial cells are more abundant, and there are fewer red blood globules.[1] The anatomical differences between the second stages of acute lobar and acute lobular pneumonia are as follows: acute lobar pneumonia involves a whole lobe; acute lobular only portions of a lobe. Lobar pneumonia advances steadily and uninterruptedly from one point, usually from the base upwards, until the whole lobe is involved; while lobular begins simultaneously in several lobules remote from one another. Moreover, these lobules are in different stages of the inflammatory process, *e. g.*, one is dark, red and moist; another is grayish and quite firm. In lobular pneumonia the small bronchi are more or less filled with catarrhal products; while in lobar pneumonia the exudation is fibrinous and does not extend beyond the infundibula and minute bronchioles.

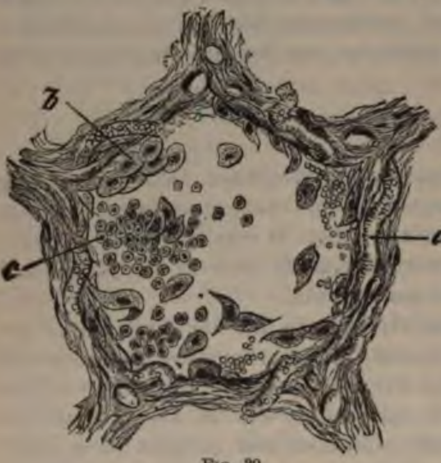

FIG. 30.

Alveolus from a Lung in Lobular Pneumonia.

The capillaries (a) are distended with blood, and within the alveolus are seen swollen epithelia (b) and pus cells (c). × 250.

The *third* stage is the stage in which occurs either *resolution, cheesy degeneration,* or *purulent infiltration. Abscess* and *gangrene* may both occur, but they are *very* rare. When *resolution* occurs the contents of the alveoli become fatty and granular and are absorbed, and the pulmonary epithelium is restored. Large confluent spots of catarrhal pneumonia may undergo *cheesy degeneration.* And even solitary lobules may remain pale and yellow, looking like so-called cheesy tubercles; when cut into, a fluid escapes from their centres. Some lobules that look like cheesy masses are soft, never granular, and a *puriform* fluid flows from their cut surfaces. While the contents of the alveoli are undergoing cheesy changes, hyperplasia of the interstitial connective-tissue is taking place, which leads to more or less *fibroid induration* or "*sclerosis*" of the lung. On the pleura covering the superficial nodules an exudation of plastic lymph occurs; the bronchial glands are swollen and hyperæmic. Catarrhal pneumonia in adults occurs independently of lobular collapse or *atelectasis*.

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[1] Many pathologists claim that the pulmonary alveolar epithelium takes no active part in the processes that result in the above-described consolidation. Rindfleisch asserts an active proliferation.

Etiology.—Lobular pneumonia is always secondary to obstruction in the air passages, especially those of capillary size. It may be excited either by the gradual extension of inflammatory processes from the tubes to the air-cells, or by the entrance of inflammatory products from the tubes into the air-cells. It is most frequently met with between the ages of one and three. The senile period also seems to predispose to it. The more imperfectly nourished the child, the more anti-hygienic the air, surroundings and food, the more liable is it to develop lobular pneumonia. Debility and a long-continued recumbent posture predispose to it. Indirectly, any cause of bronchial irritation is a predisposing cause. The bronchitis of measles, whooping-cough, influenza, and that which accompanies the acute infectious diseases, often leads to lobular pneumonia. It occurs in lung-tissue adjacent to spots of hemorrhage, or pyæmic infarctions; traumatism may induce it. It is intimately associated with all varieties of acute and chronic phthisis. It is claimed by some authorities that caseous changes—whether primary, constituting caseous pneumonia, or secondary to a catarrhal process—are always dependent upon the presence of tubercle bacilli.

Symptoms.—The phenomena which attend this form of pneumonia are always more or less obscured by those of the disease by which it has been preceded. It has no early distinctive symptoms. From an anatomical standpoint it is evident that its symptoms should resemble those of capillary bronchitis. It rarely runs a regular course,—terminating after a definite period in either death or resolution,—but may be protracted for weeks or months. A large number of cases occur in the course of whooping-cough and measles or other diseases complicated by bronchitis. The acute form is met with almost exclusively in children. In adults the disease usually runs a sub-acute or chronic course.

After a diffuse capillary bronchitis has existed for a variable period, attended by its ordinary symptoms, such as a cough with muco-purulent expectoration, slight rise in temperature and labored breathing, if lobular pneumonia is developed the labored breathing becomes panting and accelerated; the respiration may be 100 per minute. Dyspnœa is greatly increased. It is rarely ushered in by a rigor or a distinct chill. The temperature will gradually rise to 104°–105°, *unlike* the sudden rise of lobar pneumonia. It runs no typical course; though exacerbations and remissions are marked, they have no regularity; sometimes the morning, sometimes the evening temperature is the higher. It varies with the extent of lung involved, and also with the rapidity with which consolidation is developed. This steady rise in temperature, occurring in any disease in which lobular pneumonia is liable to be developed, is one of its most valuable diagnostic suggestive symptoms. When death occurs early the temperature may rise to 108°. The pulse rate often reaches 140 to 150.[1] In twenty-four to forty-eight hours the pulse becomes small, compressible, and feeble, though at first it is full and hard. The cough, which during the bronchitis was loose, "bronchial," and paroxysmal, now becomes dry, hacking, and non-paroxysmal. It is usually very painful. The expectoration is seldom seen in children; but we may find in the matters vomited clumps of tenacious, often blood-streaked muco-pus.

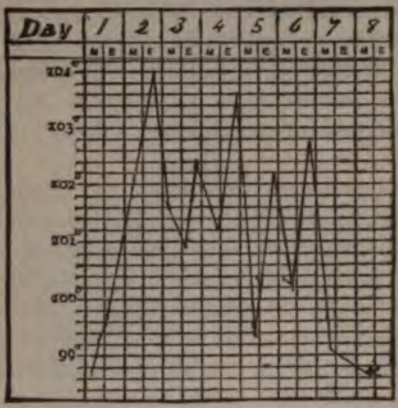

FIG. 31.

Temperature Record in a case of Acute Lobular Pneumonia in a child. (Recovery.)

After a time the dyspnœa becomes constant. The breathing is shallow, inspiration is short, the chest expanding but very slightly. The auxiliary muscles are called into play, and there is marked expansion of the nares during inspiration. The pale and anxious face becomes cyanotic, and the restlessness and jactitation give place to a lethargic semi-comatose state, interrupted by occasional but ineffectual attempts to cough. Towards the end the cough almost entirely ceases. Diarrhœa frequently increases the exhaustion; and vomiting which may accompany or follow the cough, while rare at the onset of the disease, is frequent in its advanced stages. Anorexia is an early symptom, though young children will take the breast, while older children cannot be made to swallow even the blandest liquids. The tongue may become dry; sordes collect on the lips and teeth; aphthous stomatitis is common. Emaciation is rapid. The sub-acute form often occurs in the bronchitis of strumous children, and in that which accompanies measles and whooping-cough. Its occurrence is marked by an elevation of temperature, but the rise is not so great nor so sudden as in acute cases; it rises gradually until it reaches 103° F. or 104° F. The cough becomes more severe and metallic in character, and the respiration changes from the labored respiration of bronchitis, to the rapid panting respiration of pneumonia. The patient begins to lose flesh, becomes pale, has profuse sweatings and fits of exhaustion; the appetite becomes capricious or is entirely lost; loss of strength and emaciation are progressive; the face appears bloated, small indolent abscesses appear on the nates and back, the patient assumes the appearance of extreme anæmia, and finally death slowly comes from wasting and exhaustion.

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[1] Jürgensen states that he has found the pulse *often* over 200 a minute.

Perhaps, when hope of recovery has been abandoned after a prolonged illness, resolution of the consolidated lung takes place, and a slow though complete recovery is reached. When the disease is to end fatally the temperature rises rapidly, cyanosis increases, the respiration becomes irregular in rhythm, and the comatose state is interrupted by convulsions in which death occurs. Death may occur suddenly in the midst of a violent fit of coughing. The disease may terminate with symptoms which resemble those of well-marked tubercular meningitis. When recovery occurs, it is very slow, pulse-rate, temperature, cough, and dyspnœa all imperceptibly diminishing. There is no rapid fall in temperature, such as occurs in croupous pneumonia. In a few cases, especially in older children, slight delirium will occur at night. The *urine* contains chlorides and slight traces of albumen.

Chronic lobular pneumonia differs from acute in the severity rather than the character of its symptoms. When it supervenes upon some catarrhal affection of the bronchi of moderate severity—a whooping-cough or an attack of measles—the temperature gradually rises until it reaches 102° or 103° Fahr. Exacerbations and remissions then occur which are more irregular than in the acute or sub-acute variety. The respirations increase in frequency. The increase in the pulse-rate, the dyspnœa, the anorexia, the loss of flesh and strength—all are more marked than in the acute form and much more persistent. The interference with respiration is greater than in lobar pneumonia. As muscular weakness increases the auxiliary and normal respiratory muscles become more and more enfeebled, and the supply of oxygen becomes, in some cases, so much diminished as to cause complete muscular paralysis. From all of these causes, and perhaps from the prolonged fever, the heart becomes feeble. Should recovery occur, the fall of temperature and the decline of the other symptoms are gradual, and there is great liability to a second attack during the protracted convalescence. A spot of consolidation often remains after recovery is apparently complete. Bronchiectasis, fibroid induration of the lung and emphysema are frequent sequelæ; and in children as well as in adults, phthisis is a not infrequent sequela. The pneumonic symptoms are much less pronounced in adults than in children, except when, in the latter, the disease supervenes upon diphtheria. In the old and feeble, especially when they have lain in one position for a long time, lobular pneumonia occurs as the result of hypostasis, independent of bronchial catarrh. In senile bronchial catarrhs gravitation determines the lobular pneumonia, and it is not infrequently unilateral. If epidemic influenza is complicated by lobular pneumonia the sputa, in adults, may be quite free and blood-streaked, but never rusty.

Physical Signs.—*Inspection.* In well-marked cases the expansive movement of the chest is diminished; the diaphragmatic depression is deepened and the lower ribs may appear retracted. Should there be extensive pulmonary collapse the chest walls will be retracted.

Palpation.—Slight increase in vocal fremitus may exist over isolated spots, if the pneumonic nodules are large and near the surface.

Percussion.—There is slight dulness over the consolidated spots. The diminished pulmonary resonance in lobular pneumonia is not easily distinguished from lobular collapse, and since collapse is symmetrical, usually involving both posterior bases, the difficulty is increased. One should therefore percuss, in succession, over similar areas on either side of the chest, for if a spot of dulness on one side has no counterpart on the other, one may be sure that it corresponds to a spot of pneumonic consolidation, and not to collapse. The upper part of the chest may be extra resonant and even tympanitic if there is much emphysema.

Auscultation.—On auscultation, small mucous râles resembling sub-crepitant râles, having a fine, crackling and metallic character, are heard over the spots where there is dulness. These râles are audible both with inspiration and expiration. They are not as fine as, and are more superficial than ordinary pneumonic crepitation. In children, during and after a fit of crying, fine crepitating sounds may be heard which were not audible during quiet respiration. The breathing is less vesicular and may even be broncho-vesicular. There is increased vocal resonance and perhaps bronchophony. The respiration in unaffected portions of the lung is more or less exaggerated. When there is an extensive bronchitis with the pneumonia, moist, dry and bubbling râles, varying from the finest to the coarsest, may be heard all over the chest.

Differential Diagnosis.—Lobular pneumonia may be confounded with *croupous pneumonia, capillary bronchitis, acute tuberculosis* and *pulmonary collapse.*

The differentiation between lobar and lobular pneumonia has been given.

Capillary bronchitis may be primary; lobular pneumonia is always secondary. The range of temperature in capillary bronchitis is lower than in lobular pneumonia. The breathing is labored in capillary bronchitis and *panting* in pneumonia. Capillary bronchitis is marked by exaggerated resonance on percussion; lobular pneumonia by dulness. In capillary bronchitis râles are heard all over the chest and there is no bronchial character to the breathing; while in pneumonia fine râles are heard over the dull spots accompanied by broncho-vesicular respiration.

Acute tuberculosis is accompanied by a higher temperature than lobular pneumonia and the pyrexia precedes the physical signs of bronchitis; whereas in lobular pneumonia the signs of a bronchitis precede the physical evidences of consolidation. Lobular pneumonia occurs oftenest in the child (1 to 4 years); tuberculosis occurs in early adult life. Acute tuberculosis is attended by more rapid exhaustion and emaciation, and hæmoptysis is not infrequent. The presence of brain symptoms is in favor of tuberculosis. The history of an inherited tendency to tubercular disease is rarely absent in tuberculosis.

In *collapse of the lung* there is a tympanitic quality to the percussion note over the unaffected portions. The affected side is more retracted, and sinking of the ribs and elevation of the diaphragm are more marked than in pneumonia. In collapse the respiratory murmur is feeble or absent and has no bronchial character. Râles are rare in a collapsed portion of lung. Vocal resonance and fremitus are diminished.[1]

Prognosis.—The prognosis in lobular pneumonia is determined almost entirely by the conditions which precede and attend its development. In children under five the average mortality is one in five. The younger and feebler the subject, the more unfavorable the prognosis. The prognosis is better when it follows measles than when it occurs after whooping-cough and scarlatina or when it complicates renal or heart disease. When the initial bronchitis is severe and extensive, when the temperature rises rapidly to 105° to 106° Fahr., when there is cyanosis, muscular paralysis, and the pulse is feeble and frequent, the prognosis is very unfavorable. Lobular pneumonia occurring in a rachitic subject is always grave. The greater the extent of lung involved the more unfavorable the prognosis. The more *abrupt* the onset the *better* the prognosis. The average duration of acute lobular pneumonia is from ten to fourteen days; death may occur within the first week. Chronic lobular pneumonia may be complicated by capillary bronchitis, fibroid induration of the lung, tuberculosis, pleurisy, emphysema and pneumothorax. A very frequent complication is acute intestinal catarrh. It may be complicated by pyæmia and pulmonary infarcts *non-pyæmic* in origin. Death may result from asthenia, asphyxia, or from complications.

Treatment.—It must be borne in mind in the treatment of this affection, that it is a secondary disease and that its occurrence indicates that the patient is in an enfeebled condition. All depressing remedies must be avoided; even when the disease assumes a very active form depletion is not allowable. When the bronchitis is extensive, vapor inhalations and the internal use of muriate of ammonia are to be employed in accordance with the plan proposed in the treatment of capillary bronchitis in children. The patient should be kept in a warm room, the temperature of which should never fall below 60° F. The ventilation should be as thorough as possible, but all draughts and sudden changes of temperature should be avoided. The air should be kept moist and the body should be covered with flannel. Some recommend cold compresses to the chest; the cold pack is likewise advocated. Cold baths or baths from 77° to 86°, which are subsequently lowered to 60° Fahr., are also advocated even for young children. Jürgensen recommends that a small stream of cold water be thrown just over the upper part of the back of the neck, the irrigation of which produces the most violent respiratory efforts. My experience has been altogether against cold applications; and I regard the application of counter-irritants, blisters, etc., especially to young children, as productive of more harm than good. I prefer that the chest should be enveloped in linseed or mild mustard poultices. Or, in very young children, it may be rubbed two or three times a day with a stimulating liniment and wrapped in cotton-batting covered with oiled silk.

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[1] *Jürgensen* states that the differential diagnosis between collapse and catarrhal pneumonia can be determined only in two ways: the diminution in volume of a certain portion of the lung, if distinctly demonstrated, is evidence of simple collapse, an increase in volume is evidence of infiltration. In pulmonary collapse there is not the rise of temperature which always attends broncho-pneumonia.

During the whole course of the disease the food should be fluid, nutritious, and administered in small quantities and at short intervals. Brandy or gin in milk, ten to twenty drops every three or four hours, may be given to a very young infant, and the quantity may be increased until the pulse is increased in force, the respirations become less frequent, and the distress and cyanosis diminish. As a rule stimulants must be commenced at the very onset of the disease and continued throughout its entire course; the quantity to be administered is to be determined by the necessities of each case. The drug which has most power in reducing temperature and combating asthenia is the sulphate of quinine, which may be given in full doses during the period of fever; and as an aid to resolution it is most serviceable in small doses during the active period of the disease. If the attendant bronchitis is extensive, and the accumulation in the tubes obstructs the entrance of air into the lungs, emetic doses of ipecacuanha will often afford great relief. Apomorphia is advocated by some, but the danger of its producing collapse is very great in young children. Under no consideration is opium to be given. Oil of turpentine, five drops every four hours, is often beneficial in chronic cases. While a patient with acute lobular pneumonia should always be kept in bed, it cannot be too constantly borne in mind that he should not constantly lie upon his back, for collapse and hypostasis are apt to occur in a lung whose power of resistance is diminished. Convalescence should be managed with the greatest care, for fatigue and exposure may induce bronchitis, a second attack of lobular pneumonia, and quite probably the advent of phthisis will be hastened, if a tendency to that disease exists. If the disease is prolonged and emaciation is marked, cod-liver oil, iron by hydrogen, or the syrup of the iodide of iron should be given, with a change of air.

INTERSTITIAL PNEUMONIA.

Interstitial pneumonia is a fibroid induration of the lung due to chronic inflammation involving its fibrous framework. Multiplication of the connective-tissue elements in the pulmonary septa takes place, which leads to progressive obliteration of the alveolar cavities and conversion of portions of the lung into callous fibrous masses. It has been called chronic fibroid, and chronic interstitial pneumonia; also scirrhus and cirrhosis of the lung.[1]

Morbid Anatomy.—The new tissue formation in this variety of pneumonia may involve the walls of the air-vesicles, the bronchi, the blood-vessels, and the pleura. It may be arranged in the form of nodules, bands, or irregular patches, and it may involve an entire lobe. The lung is sometimes shrunken from one-third to one-quarter its normal size. The first change is hyperæmia of the intercellular and interlobular tissue, followed by the development of fibro-nucleated tissue from the alveolar and bronchial walls, and from the interlobular connective-tissue. At the same time the alveolar epithelium undergoes more or less proliferation. As the new tissue contracts, it slowly replaces and obliterates the alveolar structure. As a consequence, the calibre of the air-cells is diminished. The new tissue development may reach such an extent that all of the air-cells of that portion of the lung which is the seat of the process may be obliterated, and no trace of lung-tissue remain. This is not infrequently observed in the subpleural tissue, and at the apex of the lung in chronic pleuritis. The alveolar cavities, when not obliterated, are empty or contain exudation products. Frequently, dense fibrous bands pass in from a thickened pleura, and the changes are more localized. The nuclei of the capillaries participate in these fibroid changes.[1] In a few instances the process begins, and is chiefly located, in the tissue about the bronchi and the blood-vessels.

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[1] Chomel and Grisolle state that it is very rare. Fox (Reynolds' Practice, p. 245) says it is rare except in connection with tubercles; but Niemeyer (Practice Medicine, vol. i. p. 195) states that it is one of the most frequent pulmonary diseases.

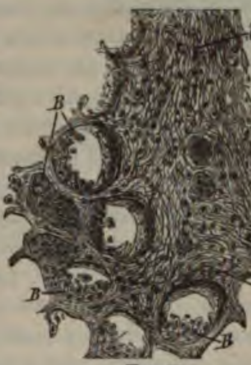

FIG. 32.
Interstitial Pneumonia.

Section of Lung through one of the interlobular septa with the contiguous vesicular structure.

A A. Band of new connective-tissue from an interlobular septum—much thickened and containing many lymphoid and large nucleated cells.

B B B. Pulmonary alveoli on one side of the lobular septum with walls thickened and infiltrated. Changed epithelia are seen crowding the air-vesicles. × 200.

On section of a lung or portion of a lung that is the seat of interstitial pneumonia, there is a creaking sound with the movement of the knife, accompanied by a sense of resistance that normal lung never offers. The lung substance tears with difficulty. The cut portion is firm, dry, hard, solid and shining, its color varying: at times it is of a dull, glistening, slaty blue; at others whitish, resembling an amyloid organ; or it may be yellowish red.[2] The bluish-colored lung is called by some "gray black-fibroid induration."[3] Sometimes the cut surface presents a marbled appearance, due to the irregular pigmentation of the interstitial tissue. The bronchi are usually dilated in those portions where the fibroid changes are well marked. The dilated tubes may form cavities of considerable size, and their mucous surfaces are often ulcerated, or the seat of intense passive hyperæmia. Bronchial dilatations rarely occur unless there has been a preceding peri-bronchitis, which has diminished the elasticity of the bronchial tubes. As the new connective-tissue develops at different points, the weakened bronchi become constricted, and as a result of this constriction bronchiectases form, the dilatation being increased by the violent inspiratory efforts that attend the fits of coughing which are so common in interstitial pneumonia. It has been maintained that with the diminution in size of the lung, there is compensatory retraction of the chest-walls; but that this compensation being inadequate to the loss in size, dilatation of the *bronch* occurs, to restore the equilibrium.[1]

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[1] Fox in "Reynolds' System."

[2] "Red induration of chronic pneumonia."—*Fox.*

[3] Reynolds' System. Art. *Pneumonia.*

Microscopically, the new tissue is seen as a dense homogeneous, or obscurely fibrillated mass, containing comparatively few cells. Pigmentation is especially marked about the vessels, and shades off gradually into the new tissue. The arteries are not obliterated, but when cut across stand gaping in the section.[2] The pleura of the affected lung is thickened and adherent. The bronchial lymphatic glands may be hypertrophied, indurated, or cheesy. The heart is somewhat hypertrophied, and nearly always displaced when there is considerable retraction. Granular kidney and granular or cirrhotic liver are frequent accompaniments; and all these changes are ascribed to the existence of a "fibroid diathesis."[3]

Etiology.—Interstitial pneumonia is always secondary—a conservative process in many instances. All protracted inflammatory processes in the lungs are attended by more or less interstitial pneumonia. All pulmonary phthisis, unless it run a very acute course, is attended by it. It is the limit of progression in many cases of peri-bronchitis, and occurs in all lung tissue which is the seat of neoplasms, infarctions, encapsulated abscesses, etc. Chronic bronchitis inducing a dry pleurisy, with the gradual development of thickenings and adhesions, is a frequent determining cause of interstitial pneumonia; it progresses more rapidly when starting from the pleura, than from any other centre. Interstitial pneumonia is more apt to develop in the gouty and rheumatic, after pleurisy or bronchitis, than in any other class. Niemeyer states that it may result from simple "collapse."

Symptoms.—The subjective symptoms of interstitial pneumonia are at no time well defined; and it is impossible to determine the exact period of its commencement, for in the majority of cases its symptoms are continuous with those of the pre-existing disease. If, after a lobular or pleuro-pneumonia, the dulness on percussion, bronchial breathing, absence of the vesicular respiration, slight elevation of temperature, cough and dyspnœa continue beyond the period at which resolution should occur, the development of interstitial pneumonia may be suspected. Dyspnœa is a constant symptom, and is increased by active exercise, and by lying on the unaffected side. Cough is rarely absent, and, as the disease progresses, it becomes paroxysmal. It may be accompanied by a copious muco-purulent or gray-black fetid expectoration. Sometimes it will separate, on standing, into three layers:—the lowest contains the solid matter, and is yellow in color; the middle is a greenish fluid; and the surface is frothy, containing mucus and fat-granules. If there are deep-seated bronchiectases, the cough is harassing and painful; when ulceration of the bronchial mucous membrane exists, hæmoptysis is not infrequent. The pyrexia has no regular or typical course; the temperature is often highest in the evening, rarely rising above 102° F. The morning temperature may be normal. The respirations and pulse are but slightly accelerated, there will be gradual loss of flesh and strength, night-sweats, dyspeptic symptoms, and not infrequently diarrhœa. The fever assumes a hectic type, and the consequent anæmia and interference with the pulmonary circulation lead to general dropsy. In some instances a large portion of lung may remain indurated for a long time, without giving rise to any symptoms except dyspnœa. The patient always lies on the affected side; any other position increases dyspnœa and cough.

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[1] Sir D. Corrigan. Ziemssen records a case where dilatation of the bronchi occurred in both lungs, while induration was found only in *one*. Secondary inflammation of the indurated parts is regarded by Traube as one of the most common causes of gangrene of the lung. In 100 cases of bronchiectasis gangrene occurred in eight.

[2] Cornil and Ranvier describe a *senile* (? physiological) condition of lung called *slaty induration of the apex*. The tissue is hard, elastic, non-crepitant and black; upon the surface it sometimes presents depressed cicatrices of the pleura and dense fibrous adhesions. Upon section there is seen a dense tissue formed of thickened septa, limiting retracted alveoli; or there are emphysematous dilatations surrounded by a dense fibrous-tissue which is infiltrated with black pigment. Caseous or calcareous nodules lodged in minute cystic cavities are also formed in the midst of this fibrous-tissue. The nodules are altered pus. Here we also find spiculæ of bone at the apex in some cases.

[3] Rokitansky, in his Pathological Anatomy, thus describes a bronchiectatic cavity: "We find a bronchial tube widened into a fusiform or rounded pouch: in the latter case the dilatation often being greater on one side than another, so that a greater part of the bronchial sac lies out of the axis of the bronchial tube. In rare cases the size of such a pouch may equal that of a hen's egg. They will often contain a bean, a hazel-nut, or a walnut. We further find, either that any one of the bronchial tubes may become expanded into a pouch of this kind, the tube retaining its normal calibre on either side of the dilatation; or else quite a large tract of the bronchial ramifications may undergo enlargement. Then many such sacs of different size are so grouped together that they form, as it were, a vast sinuous case with many branches, whose individual pouches are bounded and separated from one another by ledges or valvular folds of the bronchial wall."

Physical Signs.—*Inspection.* After retraction of the lung has occurred, inspection will show retraction of the chest-walls over the indurated portion of lung, with marked loss of expansion on the affected side. Without these signs the diagnosis of interstitial pneumonia cannot be made. The younger the patient, the more the chest-walls are retracted. There will be bulging of the healthy side, and increased respiratory movement. The apex-beat will be displaced to the right or left, according as the right or left lung is the seat of induration. In some rare instances it is so displaced as to be seen under the *right nipple* or under the *left clavicle.*[1]

Palpation.—Vocal fremitus is usually increased over the affected portion of the lung, but it may be diminished.

Percussion elicits a dull note, "toneless" or "wooden" in character. The note is high pitched, and somewhat tubular. A sense of resistance is imparted to the hand in percussing. The normal lung may overlap its shrunken, indurated fellow; hence, dulness will not be as well marked anteriorly as posteriorly. It may even be *normal* in front and dull and wooden behind. The percussion note over the opposite lung is extra-resonant.

Auscultation.—There is a loss of vesicular quality in the respiration over the affected portions of lung, and the breathing is more or less bronchial in character. The bronchial sounds are loud and sometimes amphoric. Bronchophony and pectoriloquy are not infrequently present. Râles are heard, varying from the large, moist, metallic, to the high-pitched bubbling. The râles may only be audible after coughing. The respiration in the unaffected side will be exaggerated.

Differential Diagnosis.—Interstitial pneumonia may be confounded with

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[1] Niemeyer states that the "depression of the infra- and supra-clavicular regions which accompanies pulmonary consumption is due to interstitial pneumonic induration."

pleurisy with retraction, *cancer of the lung, collapse of one lung,* and *pulmonary phthisis.* It is often difficult to distinguish fibroid pneumonia from *pleurisy* with retraction, and without the aid of an intelligent history it is impossible. There will be retraction of the chest-walls in both. The retraction from pleurisy is uneven, the ribs are twisted, and the spine more or less curved. In interstitial pneumonia the retraction is uniform, and there is a general diminution in size of the affected side. Hæmoptysis, fetid expectoration, and pyrexia may be present in induration of the lung, but not in pleurisy with retraction. Bronchial respiration is usually present in pneumonia, while the respiratory sounds are feeble or entirely absent over the affected side in pleurisy.

In distinguishing fibroid induration of the lung from *cancer* of the lung, although the physical signs of the two conditions are similar, the history of the two diseases is so different that by it alone a differential diagnosis can be made. Then the existence or non-existence of cancer in other parts of the body is important, for primary cancer of the lung or pleura is rarely met with. After the cancer has become sufficiently extensive to simulate the physical signs of pneumonia, there can be but little difficulty in the diagnosis. For, at that late period, the constitutional disturbances attending the development of a cancer, as manifested by the cachectic look and the glandular enlargements, will make the way to a correct diagnosis quite plain. If the disease has lasted two years or over, cancer is excluded.

Collapse of an entire lung is exceedingly rare, and can only be caused by a tumor pressing on a main bronchus. In collapse the respiratory sounds would be feeble or absent, and there would be no cough, expectoration, or constitutional symptoms. If pressure on the trachea or a main bronchus is long continued, the lung supplied by the compressed bronchus will be studded with hepatized lobules, collapsed lobules, and interstitial pneumonia ; the diagnosis will be determined, not by the physical signs, but by the presence of the tumor.

Prognosis.—Interstitial pneumonia is never a direct cause of death. Such patients live for years, and suffer only from dyspnœa. As it is a secondary disease, the prognosis will be determined by the primary disease. Extensive induration of the lung, following a slowly resolving croupous pneumonia and accompanying a chronic bronchitis, may continue for years after bronchial dilatations have occurred. The dangers and causes of death are intercurrent pulmonary affections, marasmus, hæmoptysis, and secondary right heart dilatation, accompanied by tricuspid regurgitation. If gangrene occurs in indurated tissue, it rapidly extends, and causes death by exhaustion or septicæmia. Some cases of sudden and unexpected death occur from thrombosis of the pulmonary artery.

Treatment.—Advanced interstitial pneumonia is incurable. Cicatricial tissue, once formed in the lungs, remains during the life of the patient. Something may be done to prevent its further development ; if it is developed from a bronchitis or a pleurisy, it is important to guard against the recurrence of the bronchitis or pleurisy. Under these circumstances, the hygienic and climatic conditions are all important, and the individual should live in the climate best suited to his condition. High altitudes are always indicated.

Besides residence in a proper climate, the patient should be warmly clad, and efforts should be made to improve the general health. Cod-liver oil and iron are serviceable in most cases.

PNEUMONOKONIOSIS.

Pneumonokoniosis is a general term implying deposit of dust in the lungs. The specific term *anthracosis* is usually employed in the general sense, and the secondary inflammatory changes are included in the disease. The more frequent forms are, *anthracosis,* from coal and carbons; *siderosis,* including all metal dusts; *chalicosis,* covering mineral dusts; *tabacosis,* from tobacco; and *byssinosis,* induced by any vegetable fibre; commonly these diseases are known as stonecutter's phthisis, miller's lung, potter's asthma, and "scooper's" pneumonia.

Morbid Anatomy.—Under conditions of gradual inhalation, all the mineral and insoluble forms of dust slowly infiltrate the lung until the bronchial walls, interlobular septa, lymph channels, and lymphatic glands are more or less completely filled. Inflammatory changes are induced which are largely fibroid in character, and which can be differentiated anatomically from fibroid pneumonia or fibroid tuberculosis only by the presence of the exciting dust elements and the absence of tubercular tissue. Tubercular changes may supervene, but are not frequent.

The morbid appearances of the lungs are the same as in fibroid pneumonia, with the addition of grades of pigmentation ranging from a light brown or bluish black mottling, and striation, to a uniform black, with the pigment so abundant as to stain the fingers and fill the secretions with pigment particles. The bronchial glands are always more or less affected, and may cut like lumps of coal dust. Acute inflammatory processes may be induced by long exposure to peculiarly irritating particles. The changes are those of an acute pleuro-pneumonia. The pleura is red, thickened, and covered with plastic exudation; the consolidated lung is soft, pulpy, and deeply congested; the alveoli are filled with exudation products which extend to the smaller bronchi; and elements of the exciting irritant may be found with the microscope.

Etiology.—All forms of dust not soluble in the bronchial secretion may induce pneumonokoniosis. Mineral and metal dusts are usually inhaled slowly and intermittingly, and induce fibroid conditions. Vegetable elements cause bronchitis primarily; tobacco induces constitutional rather than local symptoms; and the dust from grain, more particularly oats, is the most common cause of acute pneumonic conditions, owing to its peculiar properties and the unexampled duration of single exposures. Grain "scoopers" work in confined places, as the holds of vessels, often for thirty-six hours, and at times for three or four days, with intermissions of only a few minutes for food and rest.

Symptoms.—The symptoms all depend upon the form of secondary inflammation. In the acute cases an acute muco-purulent bronchitis is followed in one or two days by the local evidences of pulmonary consolidation in both lungs, attended by asthenic fever of 103°–105° F. Delirium is a prominent symptom. The pneumonic sputa are at first "rusty" or hemorrhagic, then purulent and exceedingly offensive.

The disease lasts from ten days to two weeks, and recovery is prolonged over one or two months.

Cough and dyspnœa are the only symptoms of simple dust deposits. Later, both the physical and rational signs of fibroid pneumonia, compensatory emphysema, and bronchiectasis will develop. Tubercular complications are rare, but may be present.

Diagnosis.—This can be made only from the history, as the physical signs of the pneumonic and fibroid conditions are not modified by the etiology.

Prognosis.—The prognosis depends upon removal of the cause and the extent of changes already present.

Recovery from the acute condition is the rule, while death is unfortunately long delayed, even in the most distressing forms of fibroid pneumonia.

Treatment.—Prophylaxis is of paramount importance. Free ventilation must be secured so far as possible, and further inhalation of dust prevented by respirators. A large, fine, flat sponge forms one of the best, as it can be frequently removed and cleansed. Treatment of the acute pneumonia is tonic and symptomatic; for the chronic condition it is the same as for bronchitis, emphysema, and fibroid pneumonia, when united from other causes.

HYPERÆMIA OF THE LUNGS.

Hyperæmia of the lungs is a condition in which there is an excess of blood in the lungs; it may be local or general, active or passive. Active hyperæmia, or fluxion, is not of so frequent occurrence as passive hyperæmia or congestion. The former is due to increased afflux of blood; the latter to obstructive causes which slow the current and favor accumulation of the blood in the pulmonary capillaries. *Active* pulmonary hyperæmia may be associated with violent and accelerated action of the heart. It may be developed in young persons with contracted chest by violent exercise, like running or jumping. It may follow sudden checking of an habitual flow, mental excitement, or the drinking of large quantities of alcohol. Sudden diminution of the atmospheric pressure (as during a violent inspiration), *e. g.*, in croup, laryngitis, or whooping-cough, may cause active hyperæmia. It may be developed by the inhalation of stimulating gases or a highly rarefied atmosphere such as is met with at high elevations.

Passive hyperæmia, or *pulmonary congestion*, depends upon an obstruction to the return circulation. It occurs with varying appearances and anatomical characteristics that have led to its subdivision into *splenization, brown induration*, and *hypostatic congestion*. A form of *active* hyperæmia has, because of its physiological cause and situation, been called *compensatory hyperæmia*. Other divisions are sometimes made, but all the varieties can properly be classified under these heads.

Morbid Anatomy.—In active hyperæmia the lungs contain more blood than normal. *On section* a bright red frothy fluid flows, and if the active hyperæmia be local and compensatory, extensive œdema may result. The mucous membrane of the bronchial tubes may be minutely injected. The alveolar epithelium undergoes nutritive changes, and becomes swollen and granular. The pulmonary capillaries stand out turgid and distinct in the alveolar wall.

In passive hyperæmia the lungs, wholly or in part, are distended, of a dark blue or dark red color, crepitating little, and are heavier and less elastic than normal. The process begins in the lower lobes and then becomes general. *On section*, dark blood, often in considerable quantity, flows freely from the cut surface, but the lung-tissue retains its dark color because much blood still remains in the capillary vessels. The interstitial tissue is often œdematous and studded with points of extravasation. The bronchial tubes and pleuræ show *post-mortem* staining. Epithelial changes are very common, and the alveoli may contain many swollen and granular cells that have become detached. Even fibrin and leucocytes are found in the air-cells in simple congestion.[1]

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[1] Cornil and Ranvier.

Splenization is a form of congestion which has received its name from the close resemblance which the affected portion of lung-tissue bears to the spleen. The portion of lung which is the seat of this form of congestion is of a darker color than normal, and scattered throughout its substance will be seen little red or yellowish-white points; these little points are simply blood extravasations. Lung-tissue, in a condition of splenization, is of a dark reddish-blue, brown, or black color, airless, firmer than normal, crepitates less freely, has a more uniform homogeneous appearance upon its cut surface, and is less moist than normal lung-tissue; a dark fluid will sometimes ooze from its cut surface, but not so freely as in the other forms of hyperæmia, and the fluid is more watery in appearance. In splenization there is swelling of the alveolar walls, dilatation and tortuosity of the vessels, and a more or less collapsed state of the lung; and when a congested lung is also deprived of air it looks like muscle; and the condition is then called "carnification of the lung." In the development of this condition hyperæmia occurs and is followed by interstitial œdema; it is this interstitial œdema that distinguishes splenization from hyperæmia. It occurs in connection with typhoid and typhus fever, measles or any disease in which there are certain blood changes, and it is always developed slowly.

Hypostatic congestion is a term applied to that form of hyperæmia which occurs in the most dependent parts of the lungs; it is usually bilateral in those dying of diseases which have confined them in bed for a long time. It very closely resembles splenization, but the lung-tissue is very friable instead of doughy, and the little whitish or reddish points which are seen in splenization are absent in hypostatic congestion. The lung texture itself is but little altered. Low forms of pneumonia are liable to occur in hypostatic congested parts of the lung, and hence some call it "hypostatic pneumonia," and others again call it splenification (differing from the above described splenization).

Compensatory, or collateral, congestion, is that form of congestion which occurs in one portion of the lungs, because of obstructed circulation in some other portion. The pulmonary congestion in unaffected portions of the lung in pneumonia and pleurisy is an example of this form. The same kind occurs in collapsed lung-tissue, and about points of venous obstruction, tumors, emphysema, etc., etc.

Brown, or *pigment, induration*[1], by some called the *pneumonia of heart disease*, is a form of congestion especially connected with obstruction or regurgitation at the mitral orifice. The lung is distended, firm, heavy, seldom very moist, of a dark brown or red color, and usually contains only a moderate amount of air. It is dotted with yellowish or brownish spots, usually of small size, while its own color is generally red. The capillaries of the lung are exceedingly enlarged, both in width and length; sometimes they are three times as long as normal, and encroach on the lumen of the alveoli. The brown or yellow spots are due to old blood extravasations which have undergone granular and pigment degenerations; red spots from recent extravasations are often found beside the old ones. Some parts of the lung may present these changes very markedly, while others are but little affected. Within the air-cells are usually found large cells which have undergone more or less pigmentation. In and around the connective-tissue cells are seen these pigment granules in great number. All of these changes result from prolonged interference with the return circulation. Interstitial connective-tissue thickenings occur in this form. The pigmentation which is present is the result of the long-continued retention of blood in the parts, and the consequent changes in the blood itself. Minute blood extravasations, or even diffuse pulmonary hemorrhage, may occur with brown induration. The pulmonary arteries and veins are enlarged and congested, and the smooth muscular fibres of the parenchyma of the lung may undergo hypertrophy. A brownish fluid sometimes flows from the cut surface (Virchow's "brown œdema").

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[1] First described by Virchow, in 1847.

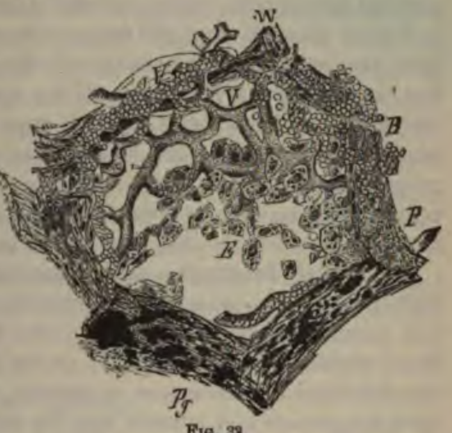

FIG. 33.

Section of Lung showing a single Alveolus in Brown Induration.

W. Wall of alveolus thickened and containing masses of pigment. Pg.
VV. Capillaries, swollen, tortuous; at B, the vessel rupturing, blood escapes into the alveolus.
P. Infiltration of pus.
E. Epithelium, swollen, distorted and pigmented.
× 250.

Etiology.—The causes of the different varieties of pulmonary hyperæmia I have sufficiently considered in connection with their morbid anatomy, so that they do not require separate consideration.

Symptoms.—It is difficult to distinguish the symptoms of pulmonary congestion from those of pulmonary œdema, and also from those of diseases in which it is liable to occur as a complication. If the congestion is considerable, there is more or less dyspnœa, cough, and expectoration. Blood-stained, watery expectoration is the prominent objective symptom of pulmonary congestion. The advent of *active hyperæmia* is usually very sudden. Dyspnœa, more or less marked, is present in both active and passive hyperæmia; it arises from decreased alveolar capacity and consequent diminished supply of oxygenated blood to the system. The dyspnœa is often urgent if the congestion is extensive, but in many cases of passive hyperæmia there will be little change in the respiration; the patient becomes accustomed to habitual dyspnœa and suffers no special inconvenience except moderate shortness of breath on physical exertion; especially is this the case in brown induration of the lungs;—a feeling of tightness or oppression is often experienced in the chest, but pain is rarely present. In extreme cases of passive hyperæmia there is usually lividity of the lips and extremities, and a sense of prostration; all the symptoms which attend imperfect aëration of the blood are developed, and the patient dies as in pulmonary œdema. The temperature may be elevated; in pigment induration it may be 100° to 102°, according to Fox, and even in other simple forms slight pyrexia is not uncommon.

The **Physical Signs** of pulmonary hyperæmia are not well marked; the movements of the chest are increased and the respiration more or less labored in character. The *percussion* sound is at first abnormally resonant, perhaps tympanitic, but as œdema is developed in the congested portion and in brown induration of the lung, it becomes somewhat dull. *On auscultation*, the respiratory murmur is feeble or harsh; in brown induration of the lung there is a feebleness of the normal vesicular murmur, the inspiratory sound becomes harsh, and the expiratory becomes prolonged. Abundant small, bubbling râles are heard when œdema accompanies the hyperæmia, as is almost always the case when it is active.[1] The *physical signs* in hypostatic congestion depend on the position of the patient, but are best marked, as a rule, behind and in the infrascapular spaces.

Differential Diagnosis.—The diagnosis of pulmonary congestion is not difficult if one considers the circumstances under which it occurs, and the two prominent symptoms, viz.: the dyspnœa, and the copious, watery, blood-stained expectoration. If, in the progress of acute pneumonia, watery, blood-stained expectoration is present, and the dyspnœa is severe, pulmonary congestion and œdema may be recognized as having occurred in that portion of lung not involved by the pneumonia. There is a blood-stained expectoration present in pneumonia, but it is of a tenacious character, and entirely unlike the copious, watery, blood-stained sputum of pulmonary congestion and œdema. The existence of pulmonary œdema being established, it is impossible to determine, either by the rational or physical signs, whether the attendant hyperæmia is active or passive in character; but in the majority of cases the circumstances under which it occurs will decide the question. Pulmonary congestion is readily distinguished from spasmodic asthma by the absence of the characteristic râles of asthma.

Prognosis.—Active pulmonary hyperæmia is usually rapid in its course, and either terminates in complete recovery, in pneumonia and pulmonary hemorrhage, or destroys life in a few hours. Recovery from acute hyperæmia is attended by sero-frothy sputa. Patients suffering from the disease can generally be relieved at its onset from the dangers which attend it. The prognosis in passive hyperæmia depends altogether upon the condition with which it occurs. When it occurs with heart disease the prognosis will vary according to the exact condition of the heart, it may then last for years, or if the patient is in the advanced stages of heart disease, and an intense pulmonary congestion comes on suddenly, the prognosis is unfavorable; in brown induration, the prognosis is uncertain. Extensive pulmonary congestion in the form of splenization leads to unfavorable results. As a rule, pulmonary congestion and œdema are very serious affections, because they complicate already existing dangerous conditions.

Treatment.—In cases of active hyperæmia coming on abruptly, and rapidly assuming a threatening aspect, an effort must be made to lessen the quantity of blood in the pulmonary vessels. This is best accomplished by the application of wet or dry cups over the entire chest, or over the seat of the congestion, or, perhaps, in extreme cases, by opening a vein in the arm; these remedies to be followed by steam inhalations, poultices, and warmth to the extremities;—saline purges are beneficial. Digitalis may be given in *active* hyperæmia from alcohol. In congestion the attention must be turned to the condition in connection with which it occurs, and the pulmonary circulation must be regulated by overcoming or controlling the cause of the congestion. If it depends on feeble action of the heart, administer stimulants; as stimulants, ammonia, quinine and ether are especially valuable in addition to alcohol. Stimulants are especially demanded in hypostatic congestion, and the position of the patient must be constantly varied. Deep, full inspirations are of service. If the hyperæmia is caused by forcible heart's action (the organ being diseased), give aconite in full doses. Hydragogue cathartics are indicated when pulmonary œdema is also present. When dependent on valvular lesions, the treatment indicated for these lesions must be employed.

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[1] Powell says a fine inspiratory crepitant râle is heard in heart-disease congestion, and that there are repeated bronchial catarrhs.

PULMONARY ŒDEMA.

Pulmonary œdema is a secondary affection which may be complicated by pulmonary congestion or may occur independently of it.

Morbid Anatomy.—The anatomical lesion of pulmonary œdema consists in the presence of serum in the cavity of the alveoli and in the interstitial tissue of the lungs; if it is associated with pulmonary congestion the serum will be blood-stained; if there is no pulmonary congestion present, the serum in the cavity of the alveoli and interstitial tissue will be light colored. Lungs which are the seat of pulmonary œdema do not collapse when the thoracic cavity is opened. Unless congestion is present, that portion of the lung which is the seat of the œdema is paler than normal lung-tissue. When the œdematous portion is pressed upon with the finger the indentation remains. The weight of the lung is increased.

On section, serum exudes or can be easily expressed from the cut surface. The serum is usually frothy unless the air cells are filled with serum. By this means we are able to determine the amount of œdema present. Œdema may occur in any portion of the lung, but it is most frequently met with in the most dependent portion. The pleural surfaces are moist, and the pleural cavities may contain some serum. When œdema of the lungs is found at a post-mortem examination, it is impossible to decide, by simple inspection, whether it occurred before or after death. In order to determine its exact import it is necessary to know the physical signs and symptoms present previous to death.

Etiology.—Pulmonary œdema, as has already been stated, is a secondary affection. It may be caused by hydræmia resulting from general dropsy depending upon Bright's disease, scorbutus, purpura, anæmia, etc. It occurs in portions of lung which are the seat of pulmonary hyperæmia (active or passive), but especially when the hyperæmia is due to heart-failure. It may be found in lung-tissue which is adjacent to parts that are the seat of inflammatory or irritative processes, as pneumonia, capillary bronchitis, miliary tuberculosis, etc. When the circulation has been obstructed in one portion of the lung, œdema[1] may arise in another portion of the same lung; its occurrence in connection with pneumonia is not infrequent under such circumstances, and is often an early indication of the necessity of prompt and careful attention in order to avert its fatal tendencies. Want of "tone" in the vessels, from pressure on the vagus or the pulmonary plexus, may cause it. It occurs in the course of acute general diseases, such as typhoid, typhus, and scarlet fevers, with feeble heart action, especially in the aged and feeble. Under such circumstances the posterior portion of the lungs is usually the seat of the œdema, and its production is aided by gravitation.

Symptoms.—The prominent rational symptoms of œdema of the lungs are increased frequency in the respiration and dyspnœa. Frequently the dyspnœa is sudden in its advent and extreme, amounting to orthopnœa. The temperature remains normal. The pulse, if increased in frequency, is feeble. There is more or less cough attended by a frothy, watery expectoration, which is colorless unless pulmonary congestion is present; then it is more or less blood-stained. The cough often has a peculiar "retching" character. If the œdema is extensive, or if it complicates some pulmonary disease, the lips become blue, the extremities livid and cold, and the patient presents a more or less cyanosed appearance.

Physical Signs.—The signs furnished by *inspection* and *palpation* are negative. There is more or less dulness on *percussion* (never complete), over the seat of the œdema; usually the dulness is equally diffused over the posterior surface of the chest on both sides, and is best marked at the most dependent portion of the lungs. It is usually more extensive at one base than at the other. On *auscultation* the respiratory murmur is feeble, sometimes entirely absent, or harsh in character. With inspiration and the commencement of expiration, small-sized bubbling râles are heard over the seat of the œdema. Sometimes these râles greatly resemble pneumonic crepitation, but they may generally be distinguished from it by their liquid character. The absence of any bronchial character to the respiratory sound excludes the presence of pneumonic consolidation. Vocal fremitus and resonance may be increased or diminished; both are quite unreliable as a means of diagnosis.

Differential Diagnosis.—Œdema of the lungs may be confounded with the first stage of *pneumonia*, with *hydrothorax*, and with *capillary bronchitis*. It is distinguished from *pneumonia* by the absence of a chill followed by febrile symptoms, by the liquid character of the râles, and by its occurrence on both sides at the most dependent portion of the lungs. A patient in the last stage of Bright's disease may suddenly develop high temperature and a cough, but in such a case the absence of a chill, as well as the bubbling character of the râles, will enable one to recognize the condition as pulmonary œdema, not pneumonia. The expectoration in the two diseases is very dissimilar.

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[1] Cohnheim states that it is the inflammatory state of the vessels, rather than increase in blood pressure (compensatory), that causes œdema in this (third) class.

The physical signs of pulmonary œdema and *hydrothorax* are quite distinctive. Œdema may be distinguished from hydrothorax by the presence of râles, and by the fact that the level of dulness is *not* changed by a change in the position of the patient, while in hydrothorax the upper border of the area of dulness, recognized by percussion while the patient is sitting or standing, will immediately shift its position when the patient stoops forward.

The onset of the *capillary bronchitis,* from which pulmonary œdema is to be distinguished, is almost always accompanied by fever. The expectoration differs in character from that of pulmonary œdema. In capillary bronchitis it is at first scanty and tenacious, and even when the disease is fully established, although it may be abundant, it is still tenacious. In pulmonary œdema the expectoration is always frothy and watery in character and abundant. In œdema there is always some dulness on percussion, often it is well marked; in capillary bronchitis there is no percussion dulness, but, on the contrary, exaggerated resonance. In both affections the râles closely resemble each other; they are usually more abundant in capillary bronchitis than in œdema. The two diseases are liable to occur together; but the presence or absence of fever, and the character of the expectoration, are generally sufficient to enable one to make a correct diagnosis.

Prognosis.—This mainly depends upon the condition of the patient at the time of the occurrence of the œdema. A large number of persons die (often suddenly) from pulmonary œdema in connection with general dropsy; especially is there danger when it occurs with the general dropsy depending upon renal or cardiac disease. When one lung is the seat of pneumonic inflammation, not infrequently œdema is suddenly developed in the other lung and destroys life. In continued fevers, phthisis, and other exhausting diseases, pulmonary œdema due to cardiac insufficiency often occurs as the immediate cause of the fatal issue. Extensive pulmonary œdema, sufficient to give rise to extreme dyspnœa and a cyanosed condition of the face and extremities, is of serious import and should not be lightly regarded; it necessitates a very guarded prognosis.

Treatment.—The treatment of this affection will depend almost exclusively upon the condition with which it is associated. If it occurs in connection with Bright's disease, the excretory function of the kidneys must be increased, and the vicarious excretory power of the bowels and skin brought into active operation with hydragogue cathartics, diuretics and diaphoretics; all of these eliminating forces must be crowded to their utmost. Dry cups must be applied over the thoracic and lumbar regions as often as the patient will bear them, in numbers varying from twenty to fifty at each application. If it occurs in connection with typhus or typhoid fever, stimulants are indicated, for it does not generally make its appearance in connection with these diseases until symptoms of heart exhaustion are present. If the heart's action is feeble, its power must be increased; under such circumstances, the administration of digitalis will be of service. When the œdema occurs in connection with pulmonary congestion, counter-irritation, regulation of the heart's action, or any means which will have a tendency to relieve or arrest the congestion, should be employed. In those diseases in which there are feebleness of the circulation and depression of the vital powers, it is important that the patient should not remain constantly in one position. He should frequently be moved, in order to prevent gravitation of the blood to the most dependent portion of the lungs. Care must also be taken that the lungs are filled and emptied as frequently and fully as possible.

PULMONARY INFARCTION.

(*Embolic Pneumonia.*)

There are two well-defined varieties of hemorrhage or extravasation of blood into the lungs, the *circumscribed* and the *diffused.* The latter condition is more properly denominated pneumorrhagia. Circumscribed pulmonary hemorrhage is called *hemorrhagic nodular infarction, nodular pulmonary apoplexy,* and, recently, Jürgensen has given it the name of *embolic pneumonitis,* names that are certainly misleading. The lung-tissue is not torn or rent in circumscribed pulmonary hemorrhage. In describing this condition, I shall adopt the term "*pulmonary infarction.*"

Morbid Anatomy.—Lung-tissue, which is the seat of infarctions, is heavier than normal and has a tough feel; if the infarctions are near the surface of the lung, they can readily be felt. Their more frequent seat is at the centre of the inferior lobe, near the root of the lung and at its *periphery,* for at the surface the anastomoses are fewer and the circulation feebler than elsewhere in the lung. They often occupy the sharp border of the lung. The pleura over these spots is congested or covered by a fibrinous exudation. In extensive infarctions, a sero-fibrinous or a sero-hemorrhagic effusion takes place in the pleural cavity sufficient to somewhat compress the lung. The lung-tissue immediately surrounding the infarction may be normal, congested, œdematous or blood-stained.

On section of a fresh nodule it is seen to be wedge-shaped, the apex of the wedge looking toward the root, and the base toward the periphery of the lung. The nodules vary in size from that of a pin's head to an inch or more in diameter. There are usually several in each lung. The cut surface of a fresh infarction is firm, maroon-colored, moist, and airless; and from it flows a considerable quantity of bright blood. Older nodules cut with a cheese-like section and resemble in color a dark blood clot, they are distinctly granular and quite dry, firm pressure causing only a small quantity of blood to flow from their cut surfaces. They are readily broken down into small masses. The brownish colored triangular spots have a sharp line of demarcation which distinguishes them from the normal lung-tissue. An embolus will nearly always be found obstructing the artery leading to the infarcted portion of the lung.[1] These nodules may undergo a variety of changes. Resolution is the most frequent, and takes place as follows:—the alveoli and terminal bronchioles fill with blood and become completely airless; the blood rapidly coagulates, and its color changes from the maroon of a recent clot to a chocolate, yellow, red, or gray color. The fibrin, if present, becomes granular and fatty. The blood globules undergo disorganization, showing well-marked fatty changes, and all that remains of them are hæmatoidin and hæmatin crystals, the amount of which left after partial absorption, determines the color of the infarction. The infarction is now in a condition to be gradually and completely absorbed or expectorated. The restored lung may be but little damaged, a pigmented stain alone remaining to mark the site of the infarction. After a varying length of time air again enters the air-cells that were formerly filled with blood.[1]

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[1] Virchow and Cohnheim both state that a plug does not necessarily exist in all cases: that enfeebled capillaries may *alone* be the cause.

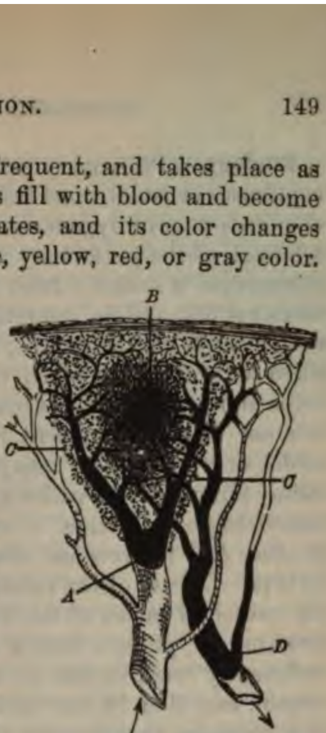

FIG. 34.

Diagram showing hemorrhagic infarct.

A. Embolus obliterating the artery.
B. Centre of infarct, disintegrating.
CC. Area of extravasation.
D. Vein filled with secondary thrombus. (*After Weber.*)

Microscopically the portion of the lung which is the seat of nodular infarction shows the capillaries distended with blood; and the arteries and veins adjoining are obstructed by coagula[2] which are red and soft in recent, and whitish and hard in old infarctions. Red, yellow, or brownish-black pigment granules are mingled with the granular and vesicular elements that fill the air cells, and the alveolar septa are thinned from pressure, and contain a varying number of red blood corpuscles "Coagulation-necrosis" is said to diminish the number of nuclei in the alveolar walls.[3]

When resolution does not occur, a cyst may form whose walls finally contract and form a dark pigmented cicatricial spot, in whose indurated tissue are found cheesy masses or calcareous plates. Again, infarctions may excite adjacent pneumonic inflammation, which in some instances may be so intense as to cause gangrene. Gangrene under such circumstances is rather a result of compression of the nutrient vessel (the bronchial artery). Usually gangrene only occurs when the embolus arrives from a gangrenous region.

When an embolism occurs in *pyæmia* or some allied state, and is stamped with pyæmic infection, the infarction will suppurate and an abscess will be formed whose anatomical characteristics do not differ from those of ordinary abscesses.[4]

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[1] Rokitansky claims that "the matters extravasated may become fluid again, and become partly absorbed and partly expelled through the bronchi. The parenchyma of the lung then gradually returns to its normal condition."

[2] Küttner states that "emboli do not *always* cause infarcts; for, although the pulmonary vessels are of the 'terminal' class, yet blood may reach the plugged vessels from bronchial vessels that enter the lung and from surrounding capillaries."

[3] Cohnheim and Weigert.

[4] Cohnheim states that "when we find in the lungs infarcts *and* abscesses an embolus has lodged on the hither side of the point of obstruction and has caused the abscess: while infarction depends purely

Etiology.—Pulmonary infarctions are either the result of rupture of the capillaries or small veins from augmented pressure following intense (mechanical) passive hyperæmia (as in mitral disease); or of the plugging of a branch of the pulmonary artery by an embolus. In the first variety the mechanism is simple: from too great pressure the capillaries are distended, stasis results, and an infarction is formed. In the second variety the plug causes arrest of the current, and the corresponding vascular area becomes filled with stagnant blood that is forced back into it from the adjoining veins and capillaries—"venous regurgitation." The arteries do not anastomose. The arrangement of the vascular distribution determines the pyramidal or "wedge-like" shape of the infarct. Specific infectious emboli induce pulmonary (so-called pyæmic) *abscesses;*—non-specific emboli produce hemorrhagic infarction. Cohnheim states that a specific plug can never produce *both* infarcts and abscesses. This view is not taken by other observers. Disease of the valves of the right heart and feeble heart power are the two chief causes of the formation of coagulum within the heart, which, breaking off, plugs a branch of the pulmonary artery. Any foreign body sufficiently small in size, or a clot from any part of the systemic venous system, may find its way to the right heart and thus cause an infarction.[2] It is supposed that thrombi may form in the pulmonary artery in cases of heart disease, *i. e.*, be of "independent" origin. Blood from the nasal or buccal cavities, or blood from the bronchial tubes, may make its way into the bronchioles and alveoli, and induce an appearance difficult to distinguish from embolic infarction. It is admitted as possible that thrombi formed in the left heart may break off, go the round of the circulation and finally lodge in some branch of the pulmonary artery.

Phthisis, scurvy, purpura hemorrhagica, gangrene of the lung, cholera, acute yellow atrophy, typhoid and yellow fever are not infrequently accompanied by pneumorrhagia of the circumscribed variety. Sometimes no cause can be found to account for a pulmonary infarction. Recent experiments have shown that *infarction does not take place unless arterial blood from some source is still sent into the part* after its *main* supply is cut off.[3]

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on the mechanical action of the simple embolus in the terminal artery." Harvard (in Quain's Dict.) thus describes the pyæmic—the metastatic abscess in the lungs: "Embolic passive hyperæmia is complicated by suppuration; but this suppuration is incomplete and consists rather in a rapid breaking down of the tissues, than in the formation of a large number of pus cells; while the characteristic deep purple congested zone around the affected spot is much intensified. Some describe this as a true sphacelus of the part; but there is no necrosis, and no foul decomposition of the patch affected in the suppurated form of embolic inflammation." When abscesses form, disintegration begins at the centre of the pyæmic infarction, and yellow croupous deposits form on the pleura over it, causing adhesions, and (*Rokitansky*) "a rounded nodular prominence like a boil projects."

[1] Rokitansky believes that an embolus always exists where there is a hemorrhagic pulmonary infarct. It may be difficult to find it, but it exists. But Cohnheim and Virchow state that emboli are *not always present*. Simple rupture of enfeebled capillaries may be the sole event. Stagnant venous blood is incapable of nourishing the walls of the tubes that hold it, and the effused corpuscles speedily fill the adjacent alveoli.

[2] Bed-sores, ulcerations, thrombosis of the femoral vein, phlegmasia alba dolens, wounds and marasmic thrombosis are common peripheral sources of emboli.

[3] FAT EMBOLISM IN THE LUNGS.—The discovery that the smaller vessels in the lungs were often plugged with fat granules, giving rise to fat embolism, was made by Wagner and Zenker. Fat emboli have been connected with the origin of metastatic abscesses by some who were inclined to regard fatal pulmonary œdema and congestion as direct results thereof. Of this, more proof is wanted. When the long bones are broken, some medullary fat gets into the circulation; but enough may enter that, when passing

Symptoms.—The subjective symptoms of pulmonary infarction are few and indefinite. A small infarction will only be attended by the expectoration of small blood clots. When one large or several small infarctions occur in the lungs of one who has had chronic heart disease, the "cardiac" dyspnœa will be increased; there will be a sense of constriction about the chest, attended by an ill-defined sense of the occurrence of some severe pulmonary lesion; irregular, intermittent and disturbed heart action nearly always precedes this occurrence. These symptoms are more apt to occur during or after some severe physical exertion or intense mental excitement, and are accompanied by the expectoration of small airless blood-coagula mixed with tenacious mucus. During the first three days the temperature sometimes rises to 101° and 102° F. Cerebral symptoms are frequently present. Syncope may occur in patients with weak hearts. If the infarction is large, or if a number occur at the same time, collapse, intense dyspnœa and convulsions may immediately follow its occurrence. If the infarction involves the surface of the lung and gives rise to local pleurisy, *pain* will be a prominent symptom. *Dark, scanty hæmoptysis* is the *pathognomonic* sign of pulmonary infarction. The rational symptoms of "pyæmic" infarctions are less marked than those of hemorrhagic. The cough and expectoration, the increase in the frequency of the respiration out of proportion to any rise in the pre-existing temperature, the constriction across the chest, and the dyspnœa taken in connection with the physical signs and the *history* will generally lead to the diagnosis of what some now call *embolic pneumonia*.[1]

Physical Signs.—*Inspection* and *palpation* give negative results, but there may be increased vocal fremitus over a large superficial infarction.

Percussion may—in the case of superficial and large infarctions—give localized dulness corresponding to the site of the infarction.

Auscultation may give bronchial breathing or bronchial expiration, especially in the mammary and mid-axillary regions; and sub-crepitant and crepitant râles may be heard in the immediate neighborhood of the infarctions. Co-existent peripheral œdema and pre-existing emphysema may, and frequently do, prevent any morbid physical phenomena from being appreciated.

Differential Diagnosis.—The etiology, the sputa, and the spots of localized dulness are the diagnostic points; of these the etiology is the most important. The sputa of *cancer* and of *echinococci* of the lung may be similar to that of infarction; but the long duration and the attendant signs of cancer, and the microscopical examination for hooklets, in the case of hydatids, will soon decide the question.

Prognosis.—In the slight circumscribed pulmonary infarction dependent

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through the lungs, it is deposited in the *small arteries* of this organ, and perhaps, subsequently, of other organs. This is all the more liable to occur when the heart action is feeble. The fat not only comes from fracture of bones, but puriform softening of right cardiac thrombi may cause it. Inflammation of bones, with or without operation, may induce it, and, also, osteo-myelitis, or softening of the marrow. The acetonæmia that has long been regarded as causing death in diabetes, is by many supposed to be inert, compared with fat embolism of the lungs, in hastening death in diabetes. Fat embolism may be the direct result of general lipæmia.

[1] Jürgensen, Ziem. Encyc., Vol. I.

upon or accompanying heart disease, or occurring with a condition that is not pyæmia, the prognosis is good. Non-embolic infarctions, and even small non-specific embolic infarcts may be absorbed. The larger and more numerous the infarctions the worse the prognosis. A large infarction may quickly terminate fatally by collapse.[1] In all pyæmic infarctions and in those occurring with cardiac thrombosis, the prognosis is bad.

Treatment.—The treatment of pulmonary infarction is, for the most part, expectant. When valvular lesions of the heart exist, the main thing is to regulate the heart's action and increase its power. Absolute rest in bed, and the administration of stimulants combined with small doses of digitalis,[2] are indicated, but they must be given with great care and their effects carefully watched. Stimulation may be made to the extremities, such as hot water or mustard sinapisms, and dry cups are to be freely applied over the chest. Venesection is contraindicated. But collateral hyperæmia and œdema, attended by great dyspnœa and lividity, may demand wet cups and even bleeding from the arm. Both give temporary relief and they avert danger. The constitutional condition of the patient is always to be considered. In pyæmic pulmonary infarction the treatment consists in supporting the patient by free administration of stimulants, quinine and iron. Dry cups may be frequently applied over the chest. If pleurisy and pneumonia occur they are to be treated as complications.

DIFFUSE PULMONARY APOPLEXY.[3]

In diffuse pulmonary hemorrhage or apoplexy the lung-tissue becomes torn and infiltrated with blood, which may be either fluid or coagulated. If situated near the surface of the lung the pleura may be lacerated. Generally the cavity made in the lung-tissue by the extravasation is of considerable size, and the coagulated or semi-coagulated blood in this cavity has all the characteristics of a blood clot. These apoplectic extravasations are never circumscribed, are usually of much larger size than infarctions, and greatly resemble apoplectic extravasations in the brain, being a mass of blood in shreddy œdematous and infiltrated parenchyma. They may prove immediately fatal, especially when the pleura is perforated. If the patient survives the shock of the accident, recovery usually takes place either by adhesion of the torn surfaces of the lung after absorption of the extravasated blood, or by the formation of a connective-tissue capsule around the clot, after which the latter undergoes a cheesy, cretaceous, or pigment degeneration, and remains permanently imbedded in the lung-tissue. It is rarely transformed into a serous cyst. This form of pulmonary apoplexy is much less frequently met with than the circumscribed form.

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[1] Jürgensen states that "embolic abscesses are not necessarily fatal to life;" and that "the prognosis always" depends more upon the primary disease than upon the accident which we call pneumonia by embolism.

[2] Gerhardt states that digitalis is *not* the drug for cardiac stimulation, but recommends large doses of morphia, hypodermatically, for the dyspnœa, and musk and alcohol to excite the heart.

[3] This use of the word apoplexy is unfortunate both for etymological reasons and because of its associations, but it has become general

Diffused pulmonary apoplexy may occur from a very large infarction, but this is comparatively rare. It generally occurs as a result of changes in the walls of the arteries. A branch of the pulmonary artery may be the seat of an aneurism, usually of small size ; or the hemorrhage may occur in connection with an aneurism of some other vessel, as the aorta, which has ruptured into the lung substance. It may occur as the result of a fall or shock ; it may also be of traumatic origin, resulting from fracture of the ribs, gunshot wounds, etc. Its most frequent cause is the rupture of thoracic aneurisms. Disease of the pulmonary artery other than aneurismal, has caused it. Erosions from cancer, gangrene or abscesses, may induce it. (Hertz.) It occurs oftenest in males (three to one), and after the twenty-first year.

Symptoms.—Profuse hæmoptysis, dyspnœa, lividity, or a sense of oppression, and often a condition bordering on collapse, are the chief symptoms of "diffuse pneumorrhagia." Convulsions occur, and the patient may suffocate from the bronchi becoming filled with blood.

Physical Signs.—The symptoms which mark the occurrence of diffuse pulmonary apoplexy are usually not well defined, and it may be difficult to positively determine its existence. There may be a profuse hemorrhage with the development of extensive pneumonic consolidation, but this will not distinguish it from other diffuse pulmonary hemorrhages. This form of apoplexy often goes unrecognized until the post-mortem examination.

Prognosis.—This is always grave. Recovery is only possible when the extravasation is of small size and the rent in the lung substance slight.

Treatment.—The diffuse variety of pulmonary apoplexy is not amenable to treatment ; in most cases the patient dies before he rallies from the shock of the hemorrhage. Cold internally and externally, ergot hypodermatically, and a solution of chloride of iron—all may be given if he rallies from the shock. During the collapse which follows the shock, alcohol and diffusible stimulants must be freely administered.

GANGRENE OF THE LUNGS.

There are two varieties of pulmonary gangrene : the *circumscribed* and the *diffused*. Circumscribed gangrene of the lungs is of much more frequent occurrence than the diffused variety. It usually involves the periphery of the lower lobes. If a bronchus opens into a gangrenous patch, inflammation of the bronchus results.

Morbid Anatomy.—In circumscribed gangrene, small isolated portions of lung-tissue, usually of a single lobe, become converted into bluish-green fetid sloughs, which at first are firm and surrounded by œdematous lung-tissue, but soon decompose into an ichorous fluid containing pus, pigment, crystals of ammonio-magnesian phosphate, tyrosin, margarin, leucin, vibriones, and bacteria, which may be discharged through a bronchus and leave a ragged, sloughy cavity surrounded by inflamed lung-tissue. Commonly, one gangrenous patch is solid, while another is becoming diffluent at its centre. A zone of catarrhal pneumonia nearly always surrounds a circumscribed patch. Vessels may traverse this cavity, but, as coagula rapidly form in them, hemorrhage rarely occurs. Sometimes, by the gangrenous process, an opening is formed into the pleural cavity and causes acute pleurisy or pyo-pneumothorax. Sometimes a spot of circumscribed gangrene becomes the centre of diffuse gangrene. In exceptional cases, the disorganized portion is expelled, a fibrous capsule forms, and healthy pus is produced. In such cases, the cavity may ultimately close up and cicatrize. Sometimes the pulmonary, but oftener the bronchial, arteries are plugged.

In *diffused gangrene of the lung*, an entire lobe is not infrequently involved, and sometimes an entire lung; unlike the preceding form, there *is no line of demarcation;* the gangrenous processes are not abruptly limited, but gradually merge into œdematous or hepatized lung-tissue. The affected pulmonary tissue is more or less decomposed, and converted into a putrid mass within an anfractuous cavity, containing, also, swarms of bacteria, floating in a grayish-black fluid; as the gangrenous process reaches the pleura this membrane becomes destroyed. Recovery under these circumstances rarely, if ever, takes place, the patient dying of septicæmia or pyæmia. Secondary gangrenous patches may be found in the same or opposite lung.

Etiology.—The conditions under which gangrene of lung-tissue may occur are numerous. Pulmonary gangrene *has* resulted from inhalation of noxious gases. In children it has followed *cancrum oris*. It may occur as the result of certain local pulmonary diseases, such as acute or chronic pneumonia, cancer, hydatids, bronchial dilatation, hemorrhagic infarctions, obstruction of the nutrient vessels leading to the gangrenous portions, or from the entrance of foreign particles, *e.g.*, bits of food swallowed by those with bulbar paralysis. It may result from erosive processes, *e. g.*, abscesses, ulcers or cancer. Putrefaction in bronchiectatic or phthisical cavities may lead to it. Traumatism not infrequently causes it. Pulmonary gangrene may occur in connection with blood-poisoning, such as is met with in low fevers, pyæmia, septicæmia, glanders, etc. Gangrene of the lungs sometimes occurs in certain nervous diseases, as dementia, softening of the brain, epilepsy and chronic alcoholismus. It is difficult to explain the occurrence of diffuse pulmonary gangrene in lunatics and drunkards.

Symptoms.—The symptoms of pulmonary gangrene, at its commencement, are often very obscure. When it develops from hemorrhagic infarction, its presence cannot generally be diagnosticated until the gangrenous process reaches a bronchial tube of considerable size. There may be dysp-

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[1] Cornil and Ranvier thus explain the loss of substance in *circumscribed gangrene;* "putrefaction and molecular destruction commence at the point where the gangrened inflammation comes in contact with the external air."

nœa, cough and pain. The two symptoms which most positively indicate the existence of pulmonary gangrene, are an extremely fetid breath, and the expectoration of gangrenous material; sometimes the fetid breath precedes the characteristic expectoration. The expectoration has usually a dirty black or brown color, and contains small black masses, and in rare instances wavy elastic fibres of lung-tissue are to be found in it; more or less blood is often present, and death may occur from hemorrhage. The sputa are yellow, or brown: *i. e.*, purulent or bloody; alkaline at first, but acid on standing; and in a test tube they form three layers: an upper of gray froth; a middle, clear and watery; and a lower containing shreds of lung-tissue. In some cases there is but slight constitutional disturbance, and the gangrenous process goes on for weeks before there are any general symptoms to indicate its presence. In other cases the greatest prostration is experienced from the beginning, the pulse becomes small and frequent, and the vital powers rapidly give way before the septic fever. Dyspnœa is in proportion to the prostration. Occasionally, death takes place from the exhaustion resulting from slow hectic fever. When diffuse gangrene of the lung occurs in connection with pneumonia, its occurrence is marked by a sudden prostration, accompanied by a small irregular pulse, a disturbed, anxious countenance, a fetid breath, and a black liquid expectoration having a gangrenous odor. If the gangrenous material is swallowed, as sometimes happens, severe diarrhœa and tympanitic distention of the abdomen occur. Gastritis sometimes results from swallowing putrid masses of sputa. In some cases of gangrene the temperature runs very high.

Physical Signs.—The physical signs of pulmonary gangrene are often obscure, and never distinctive. They are those of local consolidation followed by the evidences of breaking down of lung-tissue, and the formation of cavities in the lung substance. Percussion elicits a dull or tympanitic note; and after loose crepitation, gurgles and amphoric breathing are heard. There are no special signs indicating the nature of the disorganizing process; sometimes it is preceded by the signs of pneumonia, generally it is accompanied by signs of bronchitis, and in the later stages of the disease there are physical evidences of the formation of cavities in the lung-substance.

Differential Diagnosis.—The diagnosis of gangrene of the lungs rests almost entirely on the characteristic odor and appearance of the expectoration; prior to their occurrence the existence of gangrene cannot be determined. Gangrenous expectoration, accompanied by the physical evidences of softening and excavation of pulmonary substance, is sufficient for its diagnosis. Certain conditions may arise in which it will be difficult to make a differential diagnosis; for example, in some cases of *fetid bronchitis* there may be a profuse, greenish, sero-purulent expectoration, attended by an extremely fetid odor, not distinguishable from that of gangrene, and yet no true gangrene of the lung exists. But as bronchiectasis is nearly always present with fetid bronchitis, the physical signs of the latter would be very different from those of a gangrenous focus. (*Vide* p. 77.)

Again, gangrene of the lung may exist without any perceptible fetor to the breath or expectoration, or any of the other attendant symptoms of gangrene. Under such circumstances the gangrenous portion of the lung does not communicate with a patent bronchial tube. Again, local gangrene may occur in a phthisical cavity; when it does it is very difficult to distinguish it from true gangrene of the lung, especially if the patient is seen for the first time just as the gangrenous process is established. In this case the previous history would alone enable one to make a diagnosis. A *fetid abscess* is generally distinguished from true pulmonary gangrene not by the character of the fetor, but by the fact that the signs of excavation precede the occurrence of the fetor, while in true gangrene of the lung the signs of excavation follow the gangrenous expectoration. The sputa in abscess are decidedly purulent, and fetor does not usually occur until some time after they are expectorated. In all cases, in order to make a correct diagnosis, it is necessary to have found, in addition to the fetid breath and expectoration, decomposed pulmonary tissue in the expectorated matter.

Prognosis.—The prognosis is always unfavorable, although the circumscribed form is not regarded as absolutely fatal. Recovery can only take place in those cases where the gangrene is circumscribed and limited to a small portion of the lung-tissue. Under such circumstances it is possible for the slough to separate and be discharged, and induration and final cicatrization of lung-tissue to take place. Circumscribed gangrene may be latent, and it often progresses slowly, simulating anæmia. Diffuse pulmonary gangrene is always fatal. Sometimes death is the result of profuse hemorrhage; at other times it is due to perforation of the pleura; but more frequently the patient dies from the exhaustion which attends the septic infection. Gangrene may terminate by an external opening. It may be complicated by emphysema of the cellular-tissue, hemorrhage, pneumothorax, or peritonitis. Death often occurs within three days after the first gangrenous expectoration.

Treatment.—Under no circumstances are depressing remedies to be given. On the contrary, the vital powers of the patient must be sustained in every possible way by the administration of stimulants, tonics, and a most nutritious diet. Opium may be given in moderate doses to alleviate pain, allay the cough, and overcome constitutional irritation. Quinine is to be given for any fever that may exist. I have never found antiseptic inhalations to produce the beneficial effects claimed for them by some authorities, nor have I been able, by the internal administration of chloride of potash, to obtain satisfactory results. If antiseptic sprays are used, thymol and salicylic acid are the best. Traube gives acetate of lead and tannin preparations with opium. Charcoal, carbolic acid, creosote and chloride of sodium are recommended as deodorizers and internal disinfectants. Bromine, chlorine, oxygen, and permanganate of potash are similarly given. My own experience leads me to believe that all remedies of this class are powerless either to arrest the gangrenous processor even mitigate its unpleasant effects. It has been suggested that the lung-cavities should be tapped and washed out.

PULMONARY ANÆMIA.

Anæmia of the lungs may be due to local or general causes. In general anæmia from any cause, the lungs are paler and lighter than normal. Independent of senile atrophy, it is never met with except in conditions of extreme general anæmia. Local pulmonary anæmia may be caused by the compression of local emphysema; and by obstruction of the pulmonary artery or its branches.

Symptoms.—Dyspnœa and palpitation are its only signs.

ATELECTASIS.

(*Pulmonary Collapse.*)

Pulmonary atelectasis is a condition of the lungs where there is partial or total absence of air in the alveoli. When acquired, it is denominated *pulmonary collapse* or *compression* of the lung. Atelectasis is physiological in fœtal life, and may be described as absolute absence of air from the alveoli.

Morbid Anatomy.—In the new-born, atelectasis is usually lobular; rarely is more than one-half of a lobe involved. The lower lobes are oftenest the seat of atelectasis, then the tongue-like prolongations of the upper left lobe and the middle lobe of the right lung. The affected portions appear as sunken masses of violet or blue-red color; they do not crepitate, have a soft feel, but are tough, and resistant, and sink in water. In the atelectatic spots little yellow tubercle-like masses are found,—so-called "*bronchial abscesses,*" *vesicular bronchitis*, and *granulations purulentes.*

On section, the atelectatic part is brownish-red, smooth (*not* granular), airless, and in the earlier stages dilatable; later on, not. The walls of the alveoli are approximated, touch, and, according to some, grow together. Fatty degeneration and cell proliferation occur in the collapsed spots. A whole lung may be involved, but usually only a lobe or a portion of a lobe. The collapsed portions contrast strongly with the surrounding parts. Its seat is most often in the periphery and the lower lobes of the lung. The affected portion has the same tough, "liver-like" characteristics as in *congenital atelectasis*, the difference being that in acquired collapse the lobular points are disseminated. The collapsed portion may be engorged and œdematous, a condition sometimes called "splenization." The bronchi leading to the collapsed lobules are usually congested and plugged. When collapse occurs from pressure—*compression of the lung*—the part involved and its extent depend on the site and extent of the pressure. The air cells in the collapsed portion may or may not be wholly void of air. It is flesh-like; and for a time can be inflated and caused to return to its normal size and condition. If the inspiration is insufficient and the expiratory efforts normal in force, after a time all air will be expelled, and the dry, tough gray-red mass assumes a condition known as "carnification"; and in time only a fibrous or connective-tissue cicatrix remains. Small blood-clots may be found in the affected lobes, that are frequently decolorized and perhaps adherent to the walls of the vessels, whose calibres are impervious or obliterated.[1]

Etiology.—*Congenital* atelectasis occurs in feeble infants, in those born prematurely, and in those whose bronchi, nares or other parts accessory to respiration are plugged with mucus. *Pulmonary collapse* is rarer in adults than in young children. Any disease or condition that weakens or obstructs the power of inspiration may induce it. Brain diseases are sometimes accompanied by it. Too tight clothing about the chest of feeble children may lead to it. Paralysis of the vagus is said to cause it, and muscular paralysis from disease of the cord may lead to it. The most frequent cause is some catarrhal condition of the respiratory tract that induces the formation of a plug in a small bronchus; *e.g.*, capillary bronchitis, catarrhal pneumonia and bronchitis with tenacious secretion. Twenty-five per cent. of the total mortality of very young infants may be safely set down to pulmonary collapse, following bronchitis. Collapse from *compression* of the lung results from fluid, pus, air or blood in the pleural cavity; from mediastinal tumors, from rachitic and spinal deformities, and, rarely, from abdominal tumors.

Symptoms.—In the new-born, atelectasis is shown by feeble breathing, slight motion of the chest, a low, almost inaudible, "whining" cry, lividity and coldness of extremities, constant sleepiness, and often muscular twitchings and convulsions. The child cannot nurse. Since the *foramen ovale* and *ductus arteriosus* so often remain *open* in congenital atelectasis, anomalies of the circulation may cause asphyxia, convulsions, suffocation and death. Blood clots may form in the cerebral sinuses.[2] In *collapse* there is labored breathing, dyspnœa, frequent respirations (70 to 100 per minute), and a cough with muco-purulent expectoration. Children utter the low, whining cry. Passive hyperæmia and œdema of the extremities and central organs are common results of pulmonary collapse. The pulse is small and feeble, the skin cool, the urine scanty. There is an interval between inspiration and expiration, instead of after expiration. The whole act is "shallow."

Physical Signs.—*Inspection* shows *compensatory retraction* of the most yielding portions of the thorax during the act of *inspiration*, and the intercostal spaces retract. On *percussion* precordial dulness is increased; there may be dulness when there is much condensation, but if emphysematous patches develop about the collapsed lobules the dulness may have a tympanitic quality. On *auscultation* respiratory sounds may be feeble or absent. Later there may be bronchial breathing and bronchophony. Râles may be due to associated bronchitis; they are coarse and sonorous. The physical signs of *compression* are merely those of the causative condition, *e. g.*, hydrothorax, pleurisy with effusion, etc., etc.

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[1] Lichtheim's recent experiments go to prove Virchow's assertion that air, shut in by closure of a bronchus, is absorbed by the blood-vessels, and also that elasticity of the lung acts until the air is completely absorbed.—*Arch. f. exper. Pathologie u. Pharm.*, vol. x., p. 54.

[2] Virchow's Archiv., Bd. xl., p. 240.

Differential Diagnosis.—*Pneumonia* is distinguished by the fever, flushed face, fine râles, lobar instead of lobular outline of dulness, pain, and absence of "inspiratory retraction." *Miliary tuberculosis* is distinguished by the fever, cough, and wasting, all of which will precede the physical signs. The history of the parents will here aid us. In *pleurisy with effusion* the flatness and change in line of flatness with a change in the patient's position will establish the diagnosis.

Prognosis.—Extreme atelectasis is rarely recovered from. Occurring with whooping-cough it is especially fatal. Emphysema, bronchitis, lobular pneumonia, tuberculosis and pleurisy may complicate it. Asphyxia or complications cause death. When *compression* occurs from tumors, hydro- or pneumo-thorax the prognosis is more unfavorable than with other causes. Cheesy pneumonia or phthisis may follow atelectasis or collapse.

Treatment.—In the new-born the treatment should be that described in works on diseases of children and obstetrics. Efforts at full inspiration should be encouraged. Cold water may be poured over the neck and chest. A stream of water thrown on the nuchal region is said to excite violent and strong inspiratory impulses. Counter-irritation and stimulating embrocations are recommended. The catarrh that induces collapse must be treated with stimulating expectorants, or, in children, with emetics. Arsenic, belladonna, and salts of potash and ammonia are recommended. In compression remove the cause when possible, *e.g.*, the emphysema and hydrothorax. In all cases tonics and stimulants with good nourishment are demanded ; the "depletory" plan is never indicated. Inhalation of compressed air may do good. Never let the diaphragm's action be impeded by clothing or a distended abdomen.

PULMONARY EMPHYSEMA.

Pulmonary emphysema is seldom met with unless associated with more or less bronchitis ; and emphysematous persons are especially liable to attacks of spasmodic asthma. Emphysema is essentially a chronic affection ; it comes on slowly, and when once developed is permanent.

By the term is understood either an abnormal accumulation of air within the air-cells or an infiltration of air into the sub-pleural and interstitial connective-tissue. There are two recognized varieties, termed, first, *vesicular* emphysema ; second, *interlobular* emphysema. The first is by far the more frequent and more important affection. There are no definite rules for the diagnosis of interlobular emphysema, and it rarely occurs except in connection with advanced vesicular emphysema. When the unqualified term emphysema is used, reference is always had to the vesicular variety.

Morbid Anatomy.—In emphysema, there may be simple dilatation of the air-cells without rupture of their walls; or there may be dilatation of the air-cells with rupture of their walls. The rupture of the air-cells leads to the formation of what may be called air-sacs, which vary in size from that of a pin's head to that of a pigeon's egg, and even larger. Both forms of the affection, the vesicular and the interlobular, are generally present in cases in which these larger air-sacs have formed. The changes which take place in the anatomical structure of the lung in this affection are as follows: in slight cases there is dilatation of the infundibula, and a diminished prominence of the alveolar walls, followed, later, by their rupture and partial disappearance; as a result, a small air-sac is formed, in which little ledges and filaments of tissue alone mark the site of the alveolar septa. At this time there is no well-marked line of demarcation between the infundibulum and the alveoli. As the disease advances rupture of the walls of these little air-sacs occurs and establishes a communication between their cavities. The openings thus made between the air-sacs are at the very central portion of the sac, where the wall is thinnest. By this gradual enlargement and the union of many small sacs, a large air cavity is formed, across and along the walls of which exist remains of the original tissue. These larger air-sacs communicate with the bronchi, which are sometimes enlarged. The result of this destruction of the alveolar septa is the abolition of the capillary plexus which is normally spread over the walls of the air-cells. At times ovoid collections of fat granules are seen in the thinned septa. Whether these fat cells are in the nuclei of the capillaries, or in the inter-capillary cells is undetermined;—probably they are in both. This fatty metamorphosis as a rule precedes the dilatation, and is not constant. Fatty granules are found in the protoplasm about the nuclei of the epithelial cells taken from an emphysematous vesicle. The small branches of the pulmonary artery are the longest retained; they become dilated and looped, and communicate by anastomosis with the pulmonary vein, and thus the circuit of the pulmonary circulation is kept up, but it is not nearly so free or abundant as that which exists normally. The pulmonary circulation is therefore materially interfered with by this structural change.

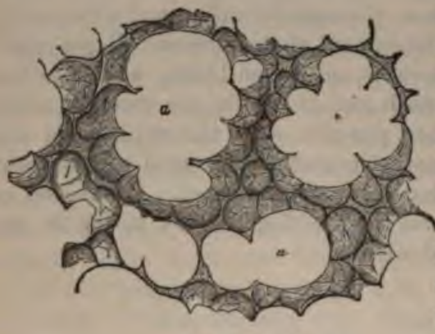

FIG. 35.

Pulmonary Emphysema, first stage.

Ectasy of the central cavity of the infundibula, a, a, a. 1-100. *After Rindfleisch.*

Well-marked emphysema generally affects both lungs; it is most marked in the upper lobes, especially along their anterior borders. Emphysematous degeneration throughout both lungs is rare. If the emphysema is compensatory its site will vary with that of the producing cause. When it is the result of strong pleuritic adhesions, it most frequently affects the anterior border of the lung. In partial collapse of the lung following obstruction of the bronchi, or in inexpansibility from disease of its structure, emphysema will usually be limited to the vicinity of the bronchial obstructions or the structural disease. When emphysema is the result of forced inspiration with closure of the glottis, as occurs in violent spasmodic croup, etc., the apex and anterior borders of the lungs are mainly involved.

Emphysematous lungs do not collapse when the thoracic cavity is opened. In well-marked cases, the lungs meet and overlap each other in the median line. The left overlaps the superficial cardiac region, both extend lower than normal, and the heart is pushed downwards and nearer to the median line than normal. The diaphragm may also be pushed below its normal position, and all of the abdominal viscera crowded out of their normal situations in consequence. In some cases the liver has been so displaced as to lie entirely below the free border of the ribs. The lungs removed from the thoracic cavity bear the impress of the ribs as furrows on their surface. Indentations made by pressure of the fingers on the surface of the lung are permanent, showing a loss of elasticity. The dilated alveoli may at times be seen on the surface of the lung through the pleura, or on section may be found distributed through its substance; they are, however, much more apparent after the lung has been blown up and dried. They appear as whitish or gray prominences, or as spherical vesicular appendages filled with air. When the air-sacs are large they protrude beyond the surface of the lung, and generally have a globular form; in some cases they seem to be separated by a neck from the rest of the lung, looking like appendages to it. In well-marked examples of emphysema, the whole anterior surface of the lungs may be covered over with air-sacs, sometimes resembling the lungs of reptiles. The color of an emphysematous lung is usually abnormally pale; it is soft and cushion-like to the touch; it crepitates but little when pressed between the thumb and finger; it sinks in water less readily than healthy lung-tissue, for though its volume is increased, its weight is diminished. By pressure the air can be forced out of the larger and smaller sacs into the bronchi. The evidences of bronchitis are usually present in the bronchial tubes. The parenchyma of the lung may present lesions which may be either a cause or a complication of emphysema. Phthisis and pneumonia, although of rare occurrence, are not as infrequent as many writers would have us suppose. As a rule in advanced cases of emphysema, the right heart will be found hypertrophied and dilated; as soon as the systemic circulation is interfered with, the left ventricle becomes hypertrophied, and this hypertrophy will for a time compensate for the obstruction to the return circulation, but as a result of this interference when it is long-continued, anatomical changes take place in the liver, kidneys and spleen, which are similar in character to those which occur in connection with valvular heart lesions, and give rise to general dropsy; changes of this class, however, belong to the remoter lesions of emphysema.

Senile emphysema differs from the variety which has just been described in the following respects: the lungs are not only diminished in weight but very markedly in size; the lobes are usually united, and their fissures directed vertically instead of horizontally; the lower lobes having lost the most in bulk, their surface is irregular, and their structure is composed of enlarged air vesicles and sacs which are the result of the natural atrophy of the lung-tissue which takes place in old age. In the aged the walls of the emphysematous cavities are usually deeply pigmented. The lung often consists merely of a number of large cavities.

In *interlobubar emphysema* an air-vesicle or sac ruptures, so that the air escapes into the interlobular cellular-tissue, forming sacs of large or small size. These sacs, or rather these collections of air, may form beneath the pleura, or, extending between the lobules of the lung and along its vessels, reach its root, spread into the mediastinal cellular-tissue, and be distributed over the neck and subcutaneous cellular-tissue of the body. The size of the air-sacs beneath the pleura may be only that of small vesicles, and these limited to the circumference of a lobule, or they may reach the size of the stomach. "They look like a membrane uplifted by foam." They may be distinguished from the vesicular dilatations by being freely movable beneath the pleura. Perforation of the pleura, producing pneumothorax, is a rare result of interlobular emphysema. More or less interlobular emphysema is always present in advanced vesicular emphysema.

Etiology.—The causes of emphysema may be divided into primary and secondary, or compensatory. Primary emphysema may exist independently of, or be associated with bronchitis. Among its causes are forced expiratory efforts, the glottis being closed or narrowed as in violent coughing, straining at stool, etc. In a few rare instances the emphysematous distention is produced during strong inspiratory efforts. In both instances the disease is developed in the upper lobes of the lung. Another cause of this variety of emphysema is, that there exists in many persons either an hereditary or an acquired impairment of the elasticity of the lungs which renders them more readily dilatable and more easily torn. There are three prominent theories which have been advanced to account for this: first, that it is due to fatty degeneration of the alveolar walls. The constancy of this change has not as yet been demonstrated. It is true that molecules of fat are sometimes seen in the alveolar septa, but they may be the result rather than the cause of the emphysema. Secondly, there is a theory that the weakness of the alveolar walls is due to the growth of the inter-capillary nuclei. Thirdly, that it is due to the fibroid degeneration of the alveolar septa. No one of these theories has as yet received full confirmation; a co-operation of all of them, more particularly of the last two, is necessary in many cases to satisfactorily explain the production of the disease. Recently another cause for the development of this form of emphysema has been advanced, viz.: an abnormal increase in the capacity of the chest, due to excessive growth of its walls. This theory as yet lacks proof.

The causes of secondary emphysema are conveniently considered under three subdivisions, in all of which the emphysema is best denominated *compensatory*. The *first* of these subdivisions comprises all cases in which the emphysema is developed around small portions of lung rendered inexpansible by disease of its tissue, as, for example, lobular collapse from obstruction of a small bronchus, a lobular pneumonia, a pulmonary infarction, etc.; the lobules adjacent to those that are thus rendered inexpansible become over-distended by a forced inspiration or a forced expiration during a violent fit of coughing. Some would make these obstructions, operating in different parts of the lung, a primary cause. A *second* subdivision comprises those cases where a large portion of lung, either from some internal cause, as pneumonia, hypostasis, atelectasis, etc., or, from some external cause, as pleurisy, etc., is rendered inexpansible, and emphysema is developed in healthy portions. In both of these subdivisions the capacity and mobility of the chest remaining normal, the usual, and especially forced, inspiratory efforts require extra distention of the alveoli to compensate for those rendered more or less useless. A *third* subdivision includes those cases secondary to croup, lobular pneumonia, whooping-cough, pressure on the trachea or main bronchi. The emphysematous distention in this class of patients is produced during inspiration. It is questionable, however, whether compensatory emphysema is ever developed when the walls of the air-cells have not been enfeebled. Interlobular emphysema is produced by forced expiration with narrowed glottis, as during severe cough, parturition, straining at stool, etc. It is usually preceded by vesicular emphysema. It may also occur from perforation of the lung from without, as in fracture of the ribs. Senile emphysema is mainly an atrophy of the alveolar septa, which become obliterated, so that vesicles coalesce. It is due to impaired nutrition, which affects the lungs as well as other organs in old age.

Symptoms.—The prominent and most constant subjective symptom of emphysema is dyspnœa. It is a dyspnœa which is increased by physical exercise, by the occurrence of fresh attacks of bronchitis, and by spasm of the bronchi, such as occurs in spasmodic asthma. When the emphysema is well marked, very slight exertion will give rise to dyspnœa; when the emphysema is slight, only violent exertion will be followed by it. It is mitigated by a warm atmosphere, and returns with increased severity during the cold of winter. There is often a "smothering" sensation in the chest, and when present it is constant. In congenital cases the only symptom during childhood and early adult life is a moderate degree of dyspnœa. In advanced cases of the disease the dyspnœa is liable to be paroxysmal, the paroxysms depending upon a tendency to spasm which emphysema in its development seems to impart to the bronchi. A cough is usually present, but it is due to bronchial irritation, and unless bronchitis exists the cough may be wanting. The expectoration varies with the extent and character of the accompanying bronchitis, and it is not uncommonly a part of the history of the emphysema; if it occurs independently of bronchitis it will have nothing characteristic about it. Usually there is no pain in the chest dependent upon the emphysema. In advanced cases the countenance is peculiar and somewhat characteristic; it is of a dusky hue and has a puffy appearance which contrasts remarkably with the wasted appearance of the rest of the body. The nostrils are distended, thickened, and vascular, and expand with each inspiration; the angles of the mouth are drawn downward, the voice is feeble, the patient stoops in the act of walking, and his whole body has a cachectic appearance; the capillary circulation of the extremities is markedly imperfect, as shown upon the slightest exertion. There is a gradual, though steady loss of flesh and strength. Usually, the disease is not attended by febrile excitement; the pulse is not accelerated, but is markedly feeble, and the temperature of the body sub-normal.

The other symptoms observed in connection with emphysema are indirect, and due to interference with the circulation. Not only is there always disturbance of the capillary circulation in the extremities, but the face and neck present a fulness or even a turgidity of the blood-vessels altogether abnormal. The distention of the jugular veins, and the lividity of the face and hands, are unquestionably due to the interference with the circulation through the right heart, but do not occur until that stage is reached in which there is more or less hypertrophy and dilatation of the right ventricle. Patients who have reached this stage become very purple in the face after and during fits of coughing, often presenting the appearance of impending suffocation. The paroxysms are perfectly characteristic; an attack of coughing comes on, grows more and more severe, gathers more or less of the spasmodic element, and when it has reached its climax the face and hands become livid, and the patient is completely exhausted. Vertigo is a common symptom in advanced emphysema; it is most apt to be developed during a fit of coughing, and depends upon the interference with the return circulation from the head. Slight hæmoptysis may occur. Emphysema of itself does not give rise to dropsy, although in advanced cases the feet and ankles are almost always œdematous. The œdema is the result of cardiac or renal complications. Ordinarily, there is more or less disturbance of the digestive organs in these advanced cases; the disturbance is due to catarrh of the stomach, the result of passive hyperæmia of the mucous membrane of the stomach from failure of the right heart. For a like reason other functions are more or less disturbed. Emphysematous patients are especially liable to hemorrhoids, and very often have profuse bleeding from the rectum. As has been already stated, the development of emphysema is almost always slow; in rare instances it advances rapidly, and it is then called *acute*. If, from the rational symptoms, there is any doubt as to the diagnosis of emphysema, the doubt will disappear after a physical exploration of the chest, for the physical signs in a well-marked case are characteristic.

Physical Signs.—On *inspection*, it will be noticed that there are alterations in the shape and movements of the chest. There is an unnatural elevation and arching of the sternum (as if from congenital deformity), and an unnatural bulging of the infra-clavicular and mammary region, which gives to the chest a more rounded appearance than in health: this has been termed "*barrel-shaped.*" The scapulæ are brought forward, and there may be antero-posterior curvature of the spine, which gives to this class of patients a stooping posture which is habitual. The muscles of the neck are unnaturally prominent. The lower portion of the chest seems contracted, and the intercostal spaces are depressed and wider than above. If the emphysema is extensive, the apex of the heart will be found beating lower down than normal and more toward the median line; if the right side of the heart is extensively dilated there will be an epigastric impulse—this impulse is due to an increase in the size of the heart, and to its being crowded to the right, and lower down in the thoracic cavity. In some instances, when the general symptoms of emphysema are well marked, the lungs are atrophied instead of abnormally dilated, and no bulging of the chest (either general or local) occurs. The movements of the chest walls are also altered and peculiar. At the upper portion expansion on inspiration is diminished or entirely wanting; the whole chest moves vertically up and down with inspiration and expiration, as if it were passively lifted from the shoulders, and composed of one solid piece; while below, the chest, instead of being dilated with inspiration, is contracted. The respiratory efforts are labored, and the breathing is chiefly abdominal. The diaphragm seems to be more actively engaged than the chest walls in the process of respiration. In cases far advanced, the existence of emphysema can be made out by inspection alone.

On *palpation* the vocal fremitus varies: it may fall below, or equal, or it may exceed that in health. In senile emphysema, the vocal fremitus is usually increased.

The intensity of the *percussion* sound is increased, the pitch is lowered, the pulmonary quality of the sound is greatly diminished, and it becomes *vesiculo-tympanitic*—that is, there is added to the vesicular element a tympanitic quality which is the characteristic percussion sound of emphysema, and is not met in connection with any other pulmonary disease. The percussion note is not materially affected, either by forced inspiration or by forced expiration. The precordial region is usually resonant, owing to the distended lungs coming between the heart and the wall of the chest.

On *auscultation*, the inspiratory sound is either short or feeble, or actually suppressed, while the expiratory is greatly prolonged, the ratio of the two being as one to four instead of four to one. As a rule, the pitch of both the inspiratory and expiratory sound is lower than in health. In some extreme cases of emphysema, the respiratory sounds are of equal length, greatly exaggerated in intensity, and of a harsh, sibilant or sonorous quality, the harsh quality undoubtedly being due to diminution in the calibre of the minute bronchial tubes. In some cases, when interlobular and vesicular emphysema are combined, a crumpling sound is heard, which has been designated as the "crumpling sound of emphysema." This sound has been said to resemble the crepitant râle, but it more nearly resembles the sound of crumpling parchment, than the crackling sound of the crepitant râle; but "*Laënnec's râle*"—a modification of the sub-crepitant râle—is very often heard. The vocal sounds vary greatly; they may be diminished, or altogether absent, or their intensity may be greatly increased. The heart sounds are feeble. The sphygmograph may afford valuable information.

Differential Diagnosis.—Slight emphysema cannot be diagnosticated with certainty; but those advanced emphysematous cases which give rise to severe dyspnœa and cyanosis are readily distinguished, by a physical examination of the thorax, from other diseases which manifest similar symptoms. The disease with which emphysema is especially liable to be confounded is *pneumothorax*. If the physical signs of the two diseases are properly appreciated, it is not difficult to distinguish between them. In emphysema the percussion sound, although somewhat tympanitic in character, still retains a pulmonary quality, and there is a vesicular element to the respiratory sound, while in pneumothorax the percussion sound has a well-marked purely tympanitic character, and the respiratory sound, if audible, is amphoric in character with no vesicular element. Emphysema affects both sides, pneumothorax only one side. The symptoms of pneumothorax come on suddenly, while those of emphysema are slowly developed, and are never so urgent as those of pneumothorax. A diagnosis of compensatory emphysema may not be made out during life, but the fact being well established that it does almost invariably exist in certain conditions, the probability of its existence should always be borne in mind in the study, examination, and treatment of those pulmonary diseases in which it is liable to occur.

Prognosis.—Emphysema rarely if ever destroys life; but, when once developed, is never recovered from, and incapacitates the person to a greater or less degree for active exercise, rendering life at least uncomfortable. It strongly predisposes to bronchitis and renders existing bronchitis severe. Acute bronchitis of the smaller tubes is an extremely grave affection when it occurs in an emphysematous person. Again, emphysema develops heart disease. The impediment to the pulmonary circulation, which exists as the result of emphysematous changes in the lung substance, gives rise to an overloaded state of the right cardiac cavities, which in time leads to their permanent dilatation and to hypertrophy of their walls; insufficiency of the tricuspid valves follows, and the resulting regurgitation through the tricuspid orifice into the right ventricle causes obstruction to the systemic venous circulation, and as a result there is congestion and a permanent disturbance of the function of the kidneys, liver, etc. In giving a prognosis in any case of emphysema, the liability to this complication should be considered. Emphysema also predisposes to fatty degeneration of the different organs and tissues of the body, the result of an impoverished state of the blood. The occurrence of these secondary affections renders emphysema a serious disease. It is undoubtedly a more serious affection when it occurs in childhood or adult life, than in old age. Pleurisy, asthma, bronchitis and anæmia are the most frequent complications.

Treatment.—The treatment of this affection will be briefly considered under two heads: first, the treatment of the disease itself; secondly, the treatment of secondary changes in other organs, which changes are more or less directly induced by the emphysema. Accepting the view that the lesions in this disease in the lung-tissue are the result of imperfect or disordered nutrition, we may reasonably expect that, by improving the nutrition, the progress of the degeneration may be checked or arrested, and perhaps even the elasticity of the unaffected portion of the lung may be restored. The most rational method of treatment is that by which we aim to remedy faulty nutrition in other organs and tissues. With this object in view, the drug which is of the greatest service is iron. This remedy should be taken daily with meals, for a long period, by persons who have emphysema or in whom it is developing. In this class of cases, the preparation which I prefer is the ethereal tincture of the acetate of iron; sulphate of quinine in small doses may be given with the iron in most cases with benefit. Strychnia, which has some reputation in the treatment of this disease, I am confident has no power in arresting its development, and it has seemed to me to increase the frequency and violence of the paroxysms of dyspnœa, and thus hasten rather than retard the emphysematous development. If an emphysematous patient has dyspeptic symptoms the mineral acids in combination with bitter vegetable infusions will be found of service. When there is a tendency to great emaciation, I have found cod-liver oil of service. Stimulants, vinous and spirituous, when taken in small quantities after or during meals, often give beneficial results, and when their use is followed by marked improvement in the general condition of the patient, they should be used in the treatment of the disease.

The regulation of the diet, and the general management of the emphysematous patient is, however, of much greater importance than the medical treatment. The diet should be of the most nutritious character, and composed largely of animal food; overloading the stomach should be especially avoided, as well as everything which has a tendency to produce flatulence. The food should not be bulky or watery in character, and should be as digestible as possible; the quantity of liquids taken into the stomach should always be small. Exercise in the open air should be taken systematically, but fatigue should be avoided. All sudden, violent exercise, or great physical exertion must be strictly prohibited. The condition of the skin should be carefully considered. Emphysematous patients should not expose themselves to cold. All localities where attacks of spasmodic asthma are liable to be developed should be carefully avoided, as also everything which may develop dyspnœa or predispose the patient to asthmatic attacks. The inhalation of compressed air is highly esteemed by the Germans. The patient should live in the open air as much as possible, and in such conditions as will make the minimum of effort sufficient for respiration. The rule for all emphysematous persons is to change their residence to that locality where they suffer the least and are not troubled with dyspnœa. The treatment of those complications which accompany, or are induced by, the emphysema is also of importance in arresting the progress of the disease. Of these accompaniments, bronchitis (generally chronic) stands first. The dyspnœa may be so urgent as to demand treatment of itself; the quebracho bark has recently received much attention as the drug for emphysematous dyspnœa. In addition to all that can be accomplished by change of climate and other hygienic measures in the management of that form of bronchitis which is so constant an accompaniment of emphysema, there is one drug which I have found especially serviceable, viz., iodide of potassium. It should be given in doses varying from five to twenty grains, three times during the day, and its administration should be continued at intervals over a long period. The treatment of diseases of the heart, liver and kidneys, which occur as complications or accompaniments of emphysema, will be considered in connection with the history of cardiac, renal and hepatic diseases.

CANCER OF THE LUNGS.

There is no variety of cancer which has not been found in the lungs. Cancer here is usually secondary to cancerous development in other parts of the body. It may extend to the lungs by direct peripheral extension of a cancer of the mediastinum or other adjacent parts, or by metastasis from distant cancer; and in either case it begins in the connective-tissue either of the walls of the air-cells, the interlobular tissue, the bronchial tubes, or the pleural or subpleural tissues.

Morbid Anatomy.—Medullary or encephaloid cancer is the most frequent variety met with in the lungs; next is scirrhus, then epithelioma, and melanotic cancer. Medullary cancer of the lungs occurs in the form of nodules of various sizes scattered through the lung-substance, or a large portion of lung may become the seat of medullary infiltration. Discrete nodules of medullary cancer cause great destruction of tissue and hemorrhage. Primary cancer usually involves only one lung, and is often infiltrated or disseminated in nodules the size of peas, while secondary cancer affects both lungs and is generally nodular, the nodules varying in size. By the union of the nodules an entire lung sometimes becomes involved. After a time the cancerous tissue may undergo fatty degeneration and softening, and the pulmonary vessels and bronchi are either involved in the cancerous development or are obliterated by its pressure. Scirrhus develops most frequently in the bronchi.

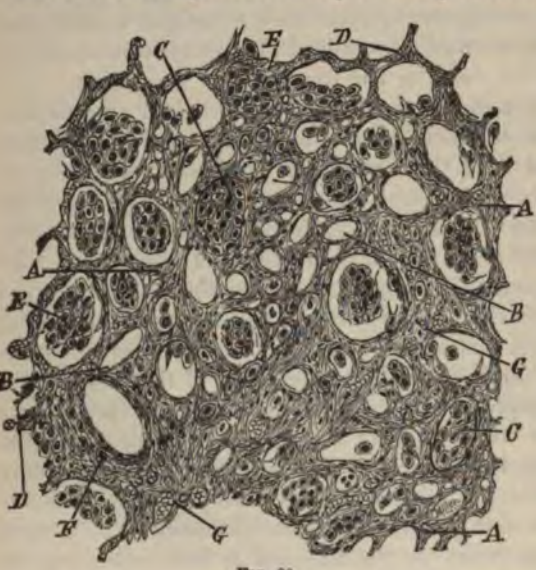

FIG. 36.

Cancer of the Lung.

Section of Lung through a small cancerous nodule. The illustration shows only a part of the nodule with the air-vesicles bounding the upper and left hand sides.

A. Fibrous stroma of the cancer.
B B. Empty alveoli of the cancer.
C C. Cancer alveoli filled with large nucleated cells of various forms.
D D. Walls of air-vesicles bordering the nodule.
E E. Lung alveoli filled with cells of same character as those filling the alveoli of the cancer. × 250.

The unaffected portion of lung-tissue may be normal, œdematous or pneumonic; extensive pleuritic thickenings and adhesions are present in most cases. Authorities differ as to which lung is oftener involved, and there are not sufficient statistics to decide the question. The lymphatics of the lungs are the channels along which the process extends. Hemorrhagic pleurisy, cancer of the pleura and bronchial glands, and hydrothorax, usually co-exist. The infundibula are filled with cancerous elements, about which is extensive pigmentation. The alveolar walls may be intact, but their vessels are engorged. There is no newly formed stroma in pulmonary cancer; the alveolar septa take the place of a stroma.

Etiology.—The etiology of pulmonary cancer is the same as the etiology of cancer in general. Hereditary predisposition may be regarded as an element in its production. It is most frequently met with between the ages of twenty and sixty, and is more common in males than females. As has already been stated, it is generally secondary to cancerous developments in other organs of the body. In the female, cancer of the breast usually precedes the development of cancer of the lungs. It may be secondary to carcinoma of the bones, testicles, uterus, stomach, liver, or œsophagus.

Symptoms.—Cancer of the lung usually comes on very insidiously, with few subjective symptoms. There is usually pain in the chest and a cough accompanied by a muco-hemorrhagic expectoration resembling currant-jelly, which occasionally contains cancerous elements. More or less dyspnœa is present, especially if mediastinal tumors co-exist. The cancerous cachexia may or may not be present. As the disease advances, emaciation, fever, night-sweats, with failure of strength, become more and more marked, and this steadily increasing weakness and emaciation is one of the most constant rational symptoms. The "pressure effects" producing lividity, œdema, dysphagia, and laryngeal symptoms, are like those of a thoracic aneurism. The glands in the axilla and above the clavicle are nearly always enlarged. If dyspnœa, cough, hæmoptysis, pain in the chest, rapid emaciation, and cachexia should come on in one from whom a carcinomatous breast had been extirpated, there would be reason to suspect the development of cancer of the lung. Signs of pleurisy, bronchitis, emphysema, or catarrhal pneumonia, may mask the signs of pulmonary cancer.

Physical Signs.—These will vary according to the seat and extent of the cancerous development. If the lung is extensively involved with nodular cancer, *inspection* will show enlargement of the affected side with widening of the intercostal spaces and deficiency or entire absence of respiratory motion. Vocal fremitus may be diminished or absent. On *percussion* there will be complete dulness attended by friction over the space corresponding to the cancer. The signs of a cavity are sometimes present. On *auscultation* the respiratory sounds may be feeble or absent, or, if a large open bronchus is intimately connected with the cancerous mass, bronchial respiration may be heard. Disseminated cancer of the lungs cannot be distinguished, by physical examination, from general bronchitis. In the infiltrated form the lung is often contracted, and, as a consequence, there is *retraction* of the chest-walls on the affected side.

Differential Diagnosis.—Pulmonary cancer is liable to be confounded with *pleurisy with effusion.* In cancer, the percussion dulness usually begins at the upper portion of the chest, while in pleurisy it begins at the lower portion. In cancer the dulness is most marked in front, in pleurisy it is most marked behind. In cancer there are isolated spots of resonance in the area of dulness, while in pleurisy the dulness is uniform over all the space occupied by the fluid. In pleurisy the line of dulness changes with the position of the patient; this never varies in cancer.

It may also be mistaken for *thoracic aneurism* and for *phthisis.* The history, the long duration and the physical signs of the latter will soon enable a diagnosis to be made. It may also be mistaken for *fibroid induration* of the lung, but its secondary character, more rapid course, greater marasmus and emaciation, and the absence of wooden dulness over an extensive tract, with retraction of the chest-walls, will suffice to make a diagnosis.

Prognosis.—The prognosis is always unfavorable, death occurs either from local or general causes in from six months to two years.

Treatment.—This is altogether palliative, and is restricted to the relief of symptoms.

OTHER NEOPLASMS IN THE LUNGS.

Non-malignant growths in the lungs are of little pathological or clinical interest.

Sarcomatous growths starting in the alveoli or in the inter-alveolar septa are always secondary; primary sarcoma of the bones is very apt to be followed by secondary pulmonary sarcoma. Melanotic sarcoma occurs with extensive pigmentation.

Fibromata occur as small hard masses varying in size from that of a pea to that of a hazel nut. They rarely occur singly.

Lipomata are usually situated beneath the pleura in the form of slightly flattened spherules.

Enchondromata—usually secondary to enchondromata of bones—are met with as discrete, irregularly roundish masses sometimes reaching the size of an egg and partly or wholly calcified—at times densely ossified.

Osteomata occur in lungs which are the seat of fibroid pneumonia. Virchow describes one the size of a man's fist. They are usually small and multiple. They may be branched, following the line of new connective-tissue developments.

Simple melanotic tumors in the lungs are similar in constitution, both to the naked eye and microscopically, to the interstitial pneumonia of miners, except that the black granules are small and round instead of angular. A melanotic tumor of the lung may invade the vertebral column in such a manner as to destroy the bodies of one or more of the vertebræ, thus giving rise to one variety of Pott's disease.

Dermoid cysts, myxoma, and *hæmatoma* are very rarely met with in the lungs; any of these tumors may displace, compress, or cause atrophy and absorption of lung-tissue; they may cause an excess of air in one part of the lung and a deficiency in another; they may cause congestion or anæmia; may ulcerate and form cavities; may induce acute local catarrhal pneumonia or fibroid induration. They may lead to bronchial irritation, induce pleurisy, or erode adjacent organs or bones. The general symptoms of non-malignant tumors within the lung are dyspnœa, which may depend upon pressure on the heart or trachea; aphonia from pressure on the recurrent laryngeal nerve; pain from pleurisy or pressure; cyanosis and dropsy of the head, neck and upper extremity, the result of pressure on the veins; and changes in the pulse from pressure on the pneumogastric. There may be dysphagia. Cough and expectoration come from pulmonary hyperæmia and œdema.

On *inspection* there may be local bulging or general enlargement of the affected side with diminished respiratory movements. Vocal fremitus may be increased, or, if the tumor is large and presses on a large bronchus, it may be diminished or absent. There is an irregular outline of dulness over the seat of the tumor. On *auscultation* there may be bronchophony and bronchial breathing, or feeble respiration and feeble voice sounds over the seat of the tumor.

Differential Diagnosis.—*Pleurisy, phthisis* and *pneumonia* are excluded very readily by their febrile phenomena, constitutional symptoms and physical signs. The *prognosis* is always unfavorable, and the *treatment* is wholly symptomatic.

SYPHILITIC DISEASE OF THE LUNG.

The most common and certain changes in the lungs which can be ascribed to syphilis are gummata.

Morbid Anatomy.—They vary in size from a pea to an egg, and are single or multiple; they appear in the lungs as well-defined rounded tumors, often surrounded by a fibrous capsule, and are usually situated in the deeper pulmonary structures. Syphilitic fibroid infiltration originating about the interlobular blood-vessels, about gummata, or from an ulcerating peribronchitis does not become caseous, but may ulcerate or become gangrenous. "*Syphilitic pneumonia of the new-born*," *white hepatization*, or "*epithelioma*," as it is variously called, is a diffuse infiltration of one or both lungs. The organ is heavy, enlarged, dense, resistant and indented by the ribs. White dry spots are seen on *section*. There is thickening of the alveolar walls and minute bronchi, and thickening and obliteration of the pulmonary vessels. Syphilitic affection of the bronchial tubes is, in such cases, extensive. Gummata may be developed in the nodules of syphilitic pneumonia. The bronchial glands are enlarged and often cheesy. Abscesses may form from suppuration in gummatous patches. The pleuræ may show fibroid thickening. Senile syphilitic gummata bear a close resemblance to caseous tubercle, but are much less friable—syphilitic patients often become phthisical, and there are good grounds for the belief that the phthisical developments commence in a proliferation of the pulmonary connective-tissue which terminates in the formation of gummata, and that these gummata have a course and results similar to those of tubercle.

Symptoms.—The symptoms are either the physical signs of a tumor, or of interstitial pneumonia. The *diagnosis* is reached by exclusion. The *treatment* is antisyphilitic.

ATROPHY OF THE LUNG.

This may be general or partial.

Morbid Anatomy.—An atrophied lung is small, dry, anæmic, and somewhat pigmented; it pits readily and can be compressed into a very small space. In extreme old age the lungs atrophy, they crepitate less, the pleura over them is less moist than normal, and they cannot be inflated as normal lungs can. They lie close to the vertebral column, and their surface is uneven and "crumpled;" the fissures change their position; the lobes may be attached to one another by pedicles; the alveoli have no definite form; and the cells are enlarged. The change in the lobes may bring the apex down to the base of the thoracic cavity. Atrophied lungs are "marbled" by lines and dots. The pulmonary artery and its branches are diminished in size, and the bronchial tubes are thinned. The first step toward atrophy is a general disappearance of the capillaries in the alveolar septa. Some fatty degeneration is always present. When it is the result of pressure by tumors, or liquid in the pleural cavity, the atrophy is generally limited to one lobe, and the atrophied part presents the lesions of interstitial pneumonia. Pigmentation and atrophy, whether local or general, are usually associated. It is commonly best marked in the superior lobes. Sometimes the lobes appear to be adherent to one another. The *right heart* is generally found in a condition known as "brown atrophy." Bronchitis nearly always complicates it. The diaphragm is thin, flabby, and pale.

Etiology.—Old age, pulmonary emphysema, and general marasmus are frequent causes of pulmonary atrophy. Pressure of a tumor or fluid accumulation within the thoracic cavity may induce local atrophy.[1]

Symptoms.—Dyspnœa, cyanosis, and œdema and coldness of the extremities are its only constant rational signs.

Inspection reveals a small thorax; the lower ribs are approximated, giving a "pigeon-breasted" appearance. The whole thorax moves as if it were one piece, as in emphysema, and the chest movements are restricted.

Percussion.—The percussion note is particularly loud, clear, and resonant; but the pulmonary *area* is less than normal. The extent of the precordial dulness is increased.

Auscultation.—The respiratory sounds lose their vesicular character and are feeble.

Atrophy of the lung admits of no treatment.

HYDATIDS OF THE LUNG.

In this country hydatids of the lung is a rare disease. There is usually one tumor, and its most common seat is the lower portion of the right lung.

Morbid Anatomy.—The cysts vary in size from that of an egg to that of a cocoa-nut. They are usually single, but may be multiple. They may be situated wholly within the lung, or be an outgrowth from the liver into the pleural cavity. The walls of the cysts vary in thickness and density. They develop in the interstitial tissue to which the parent sac is firmly adherent.

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[1] Buhl (in Virchow's Archiv., Bd. XI., p. 275) describes an atrophy observed by him in three cases of typhus fever analogous to acute yellow atrophy of the liver. He thinks it is due to a high grade of desquamative pneumonia, which latter disease will then come in the list of causes.

These cysts may cause serious pulmonary complications by their pressure. They may suppurate and be discharged into the bronchi, and then a cavity may remain. In many cases a pulmonary hydatid cyst is the result of an hydatid of the liver which has ruptured through the diaphragm. Some authorities state that *primary* hydatids of the lung is a condition yet to be met with. General pleurisy is of rare occurrence; for the slow growth of the tumor excites local adhesions rather than a general pleurisy. In some instances an hydatid cyst ruptures into the pleural cavity and causes empyema. Bronchitis, pneumonia, and gangrene may be excited in the surrounding tissue by the pressure of the hydatid tumor.

Etiology.—Hydatids of the lung are nearly always secondary to hydatids of the liver. The affection is met with most in the Norse countries of Europe, where men and animals live together.

Symptoms.—Hydatids of the lung, when small, give rise to no symptoms by which they can be detected; but as they enlarge they excite bronchitis, attended by cough, with muco-purulent expectoration, pain in the chest, a sense of suffocation, hæmoptysis, night sweats, pallor and emaciation. When blood is expectorated gooseberry-like skins (the sacs of echinococci) or hooklets may be found in the expectorated matter. Unless the daughter-cysts, or hooklets, are expectorated the diagnosis can never be positive. When an hydatid attains any considerable size it may cause bulging of the chest wall and displace the mediastinum and diaphragm. The circumscribed dulness on percussion, which may extend to the right or left of the median line, with absence of respiratory sound and vocal fremitus over the area of dulness, is a strong evidence of pulmonary hydatids.

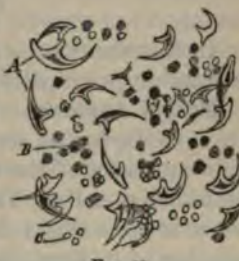

FIG. 37.

Hydatids of the Lung.

Microscopical appearance of elements found in the sputum.

A. Hooks from head of Tænia Echinococcus.

B. Pus corpuscles.

C. Red blood discs. × 250.

Differential Diagnosis.—The rupture of the cyst and the escape of its contents into a bronchial tube are its only diagnostic features and will prevent it from being confounded with any other condition. If an hydatid is superficial a portion of the fluid may be withdrawn by aspiration, and a microscopical examination will establish the diagnosis. It is impossible to distinguish between hydatid tumors at the base of the right lung and those in the right lobe of the liver.

Prognosis.—These tumors sometimes disappear by spontaneous retrogression, or by discharge into a bronchial tube; or suppuration may be established in the cysts which afterward undergo calcification. Recovery occurs in fifty per cent. of cases. Rarely do patients die from emaciation or marasmus. They may die from suffocation, when the cysts rupture into the bronchi, from long-continued suppuration, or from an empyema established by the rupture of a cyst into the pleural cavity. Inflammation of any of the three adjacent serous membranes may cause death, or this may result from extensive hemorrhage and from gangrene.

Treatment.—They should be treated as hydatids of the liver. It is a question if they should be injected with iodine.

PLEURISY.

Pleurisy is either a partial or general inflammation of one or both pleuræ. It may run an acute, sub-acute, or chronic course, and have for its products fibrin, serum and fibrin, serum, fibrin and pus, or new connective-tissue. I shall describe four varieties of pleurisy :—

(1) *Plastic or Acute Pleurisy ;* (2) *Serofibrinous or Sub-acute Pleurisy ;* (3) *Suppurative Pleurisy or Empyema ;* (4) *Interstitial Pleurisy.*

It is infrequent that the inflammatory products are confined to any one element. It is rather the preponderance of either the serous, fibrinous, or cellular element of the exudation that gives character to the disease. Most cases of pleurisy present at different stages, in varied proportions, all the inflammatory products except pus.

PLASTIC OR ACUTE PLEURISY.

In this variety the symptoms are well defined, the course rapid, and the exudation principally fibrinous.

Morbid Anatomy.—The first stage of the inflammatory process is marked by a reddening of some part of the pleural membrane from hyperæmia of the capillaries of the serous and sub-serous tissue with degeneration of the endothelial cells. The pleura loses its natural glistening appearance on account of a slight fibrinous exudation and the swelling and increase in number of its fixed connective-tissue cells. These changes take place during the first forty-eight hours. Following this, the fibrinous exudation increases and the free surface of the pleura assumes a rough, shaggy appearance. If any serum exudes it gravitates to the most dependent portions of the pleural sac. In the substance of the pleura and in the fibrinous exudation new cells are now found which are young connective-tissue or pus cells. These cells are at first more numerous on the inner surface of the pleura. As the inflammation progresses they increase in number and collect on the free surface of the pleura under the fibrinous exudation. By the fifth day of the pleurisy new blood-vessels are formed in the fibrinous exudation and become connected with the original vessels of the pleura.

The nature of the subsequent changes will depend upon the intensity of the inflammatory process ; in the milder types the fibrin gradually diminishes and disappears, some of the cells become fatty and are absorbed, and the remainder enter into the formation of a basement substance which gradually increases and finally a permanent new connective-tissue forms upon the inner surface of the pleura. If the inflammatory process subsides without much serous effusion, the opposing surfaces of the pulmonary and costal pleuræ come into contact and adhesions are formed between them composed of permanent connective-tissue containing long, slender vessels. These adhesions follow the general law that governs all new connective-tissue : they may be permanent, or—their blood supply becoming insufficient—they may undergo fatty degeneration and be absorbed, the thickened pleura alone remaining to tell of the past inflammation. When an individual has once had this form of pleurisy he will always have a permanent lesion. This pathological process may be completed in two weeks, or a serofibrinous effusion may not be absorbed for months, and then the pleuritic thickening becomes very extensive.

Etiology.—The etiology of plastic pleurisy is sometimes very obscure. Exposure to wet and cold has been regarded as one of its most frequent causes, but it is very doubtful if it ever occurs as the result of simple exposure to wet and cold. In all cases that have come under my observation where it has followed such exposure, I have been able to find some previously existing predisposing cause. It may be the result of a penetrating wound, or blows upon the chest walls. Fracture of the ribs, if the broken ends of the ribs penetrate the pleura, may cause it. It is often a complication of other diseases, such as pyæmia, the exanthematous fevers, acute and chronic alcoholismus, acute rheumatism, Bright's disease, pneumonia, etc. Sometimes it is the result of extension of inflammation from adjacent organs and tissues. While a certain proportion of cases of acute pleurisy are undoubtedly of tubercular origin, the claim that all pleurisy is tubercular has not been sustained. It may occur at any age. Although it has been claimed that it never occurs in young children, my experience leads me to believe that it is of quite frequent occurrence in children of two or three years of age, and pus is usually formed in the pleurisies of children which occur as complications or sequelæ of the exanthematous fevers. Whenever acute pleurisy occurs on the right side, it is important to determine if it is, or is not, the result of an extension of inflammation from the liver.

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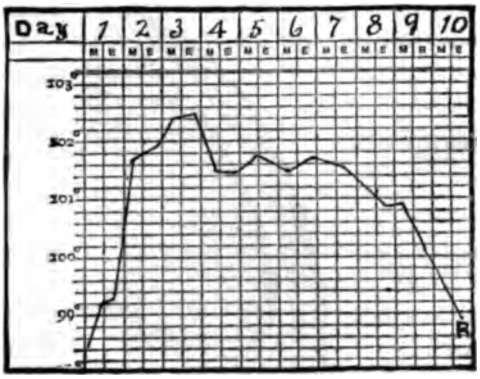

FIG. 38.

Temperature in a case of *Acute Pleurisy*. Patient æt. 24. Recovery.

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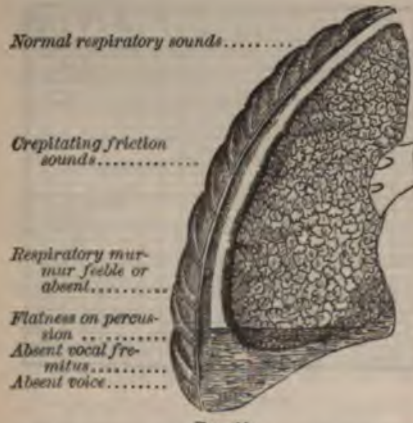
Physical Signs.—During the first twenty-four hours of plastic pleurisy, *inspection* will show the movements of the chest wall on the affected side to be more or less restricted. *Palpation, percussion,* and *mensuration* will give negative results. On *auscultation* the respiratory murmur will be found feeble over the affected side, and jerking in character both on inspiration and expiration, and a grazing friction sound will be heard; this friction sound will be most intense at the end of inspiration.

As the plastic exudation takes place *inspection* will show a greater loss of expansive motion on the affected side; and on *palpation* there will be a diminution of the vocal fremitus over its seat. On *percussion* there will be slight dulness over the seat of the pleurisy. The amount and extent of the dulness will correspond to the amount of the plastic exudation. Upon *auscultation* the respiratory murmur will be distant and feeble or entirely absent, and a crepitating friction sound will be heard both with inspiration and expiration; it is usually most intense with inspiration; this sound sometimes very closely resembles the so-called crepitant râle and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Flatness on percussion
Absent vocal fremitus
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FIG. 39.

Diagram illustrating Physical Signs in Acute Pleurisy with a small amount of Effusion. After Da Costa.

may be mistaken for it. At times this sound loses its crepitant character and becomes rubbing and sticky; it is always due to the rubbing together of the two roughened pleural surfaces. Often it will not be heard unless the patient cough or take a deep inspiration. If the pleurisy is confined to the diaphragmatic or mediastinal pleura the friction sound will not be heard.

If a considerable fluid effusion accompany the plastic exudation, the expansive movements on the affected side will be *more restricted,* and the vocal fremitus at the bottom of the pleural cavity will be markedly diminished. Upon *percussion* there will be flatness over the region occupied by the fluid. It is difficult, however, to recognize the presence of a small amount of fluid effusion, for the level of the fluid is not appreciably changed by changing the position of the patient. On *auscultation* the respiratory sounds will be absent below the level of the fluid, and feeble above it; and both respiratory acts will be accompanied by the friction sound.

During the period of absorption of the fluid and plastic exudation there will be a gradual return of the pulmonary resonance and of the normal, vocal, and respiratory sounds, and as the roughened pleural surfaces play upon each other, the friction sound will assume more of a rubbing character. In some instances the friction sounds remain audible for a long time after the disappearance of all the other signs of pleurisy. Retraction of the affected side does not follow acute pleurisy except in rare instances, when the inflammation has been very severe and a large plastic effusion has taken place. In those having extensive plastic exudations in acute pleurisy which compress the lung, the respiratory sound may assume a bronchial character and be mistaken for that of a pneumonic condition.

Differential Diagnosis.—In the majority of cases the diagnosis of plastic pleurisy is easily made. *Acute pneumonia* is the only disease with which it is liable to be confounded. In both affections there are dyspnœa, fever, and cough; but in pleurisy the temperature rarely rises above 100° F., while in pneumonia it usually reaches 103° F. within the first twenty-four hours. The cough of pleurisy is short and hacking, and is attended by little or no expectoration, whereas in nearly every case of pneumonia expectoration is present, and the substance expectorated is *characteristic of the pneumonia.* The countenance at the onset of pleurisy is pale and anxious; in pneumonia it is flushed and the cheeks have a purple hue. There is also a very marked difference in the physical signs of the two diseases. In pleurisy the vocal fremitus over the affected portion of the pleura is somewhat diminished; in pneumonia it is more or less increased. In pleurisy the respiration is feeble; in pneumonia it is rude or bronchial. In pleurisy a grazing, rubbing, or crepitating friction sound is heard with both respiratory acts. In pneumonia the crepitant râle is heard at the end of inspiration. Sometimes it is difficult to distinguish a crepitating friction sound from a sub-crepitant râle, but, as the sub-crepitant râle is not present until the last stage of pneumonia, the question will not arise if the patient is seen before that period has arrived.

PLEURISY.

Pleurisy is either a partial or general inflammation of one or both pleuræ. It may run an acute, sub-acute, or chronic course, and have for its products fibrin, serum and fibrin, serum, fibrin and pus, or new connective-tissue. I shall describe four varieties of pleurisy :—

(1) *Plastic or Acute Pleurisy ;* (2) *Serofibrinous or Sub-acute Pleurisy ;* (3) *Suppurative Pleurisy or Empyema ;* (4) *Interstitial Pleurisy.*

It is infrequent that the inflammatory products are confined to any one element. It is rather the preponderance of either the serous, fibrinous, or cellular element of the exudation that gives character to the disease. Most cases of pleurisy present at different stages, in varied proportions, all the inflammatory products except pus.

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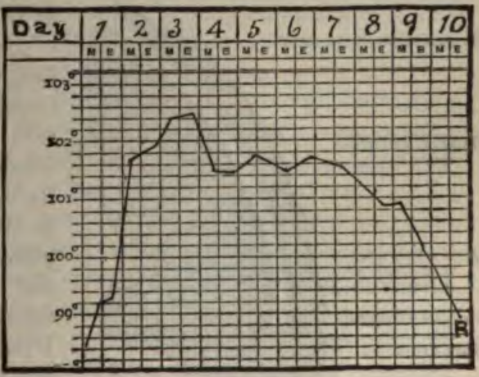

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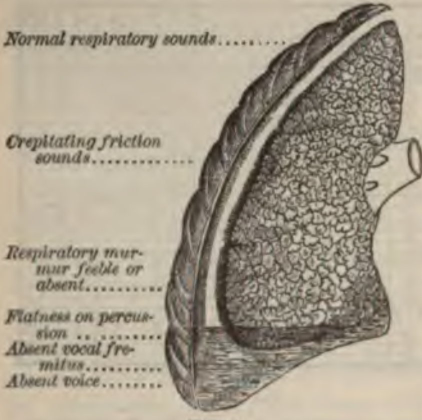
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SEROFIBRINOUS OR SUB-ACUTE PLEURISY.

(*Pleurisy with Effusion.*)

This is the most common form of pleurisy, and the inflammatory process usually invades the whole of the pleura on the side affected. It may commence at any point on the pleural surface, but it most frequently commences on the costal portion.

Morbid Anatomy.—The anatomical changes in this form are similar to those which take place in acute pleurisy, except that the new tissue formations are more extensive, the pleural membrane more uniformly thickened, and more frequently the seat of tubercle, and there is more abundant serous effusion containing flocculi of lymph. The pleural cavity may be partly or completely filled with fluid. The entire pleura becomes coated with a layer of fibrin varying in thickness, usually most abundant on its costal portion. New connective-tissue cells and basement substance are mingled with the exudation. Sometimes the serous effusion contains blood globules from the rupture of the thin-walled vessels in the new connective-tissue. It is the large amount of serous effusion, containing more or less cellular elements, that distinguishes sub-acute from acute pleurisy. This indicates a difference in the grade rather than in the nature of the inflammatory process. When the cell elements are abundant it is characterized as a sero-purulent effusion. If the pleural cavity is not filled with fluid, the effusion will occupy the most dependent portion of the pleural cavity. It may be confined to circumscribed portions of the pleural cavity by adhesions. If it occupy the most depending portion of the cavity the adjacent lung-tissue will be compressed and pushed upward. When the pleural cavity is filled with fluid the intercostal spaces will be more or less bulging, the diaphragm will be pushed downward, and the abdominal viscera upon either side may be displaced downward; the heart will be displaced either to the right or left, according as the fluid occupies the left or right pleural cavity. The lung on the affected side is compressed either toward the vertebral column or upward and inward against the mediastinum. Occasionally the lung occupies the anterior portion of the pleural cavity and the fluid the posterior portion; the direction of the compression is influenced by the location and extent of previous pleuritic adhesions. If no adhesions exist the lung may be compressed to one-eighth of its normal size, and assume a pale-red or greenish color, have a tough, leathery feel, and be entirely void of air. With the compression of the lung there may be compression of the bronchi, but the larger bronchial tubes usually remain pervious.

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Occasionally it is difficult to make a differential diagnosis between *intercostal neuralgia* or pleurodynia, and acute pleurisy. *Intercostal neuralgia* or pleurodynia may be attended by many of the ushering-in symptoms of acute pleurisy. They may come on after exposure, be attended by violent pain in the side, jerking respirations, anxious countenance, and often by considerable fever. On physical examination the respiration may be as feeble as in the first stage of pleurisy. The presence or absence of a pleuritic friction sound, and the painful points on pressure, are the principal points of difference.

Prognosis.—The prognosis in plastic pleurisy is generally good. Its natural termination is in recovery within two or three weeks after its commencement. But it is to be remembered that patients even with the milder form of the disease are liable to have frequent pleuritic thickenings and adhesions between the pulmonary and costal pleuræ. These thickenings predispose to other attacks of pleurisy, and each new attack interferes more and more with the expansion of the lungs and leads to the development of interstitial pneumonia or bronchitis, and finally to fibrous phthisis. If this form of pleurisy complicates any grave form of disease, as septicæmia, pyæmia, Bright's disease, etc., there is a liability to acute empyema. In some very acute cases, where there is a large plastic exudation, death may result in a few days.

Treatment.—The only remedial agent which has seemed to me to have a controlling power over acute pleurisy is opium. The best method of administering it is by the hypodermic injection of morphine. It has been claimed that free blood-letting at the commencement of an acute pleurisy will arrest its progress. But the facts deduced from recorded cases are strongly against this statement. A free general bleeding will undoubtedly relieve the pleuritic pain with great promptitude, but no more so than a hypodermic of morphine, and the morphine does not increase the liability to a large serous effusion, as does general bleeding. For the successful management of ordinary acute pleurisy all that I have found necessary is to place the patient in bed. This is important, however mild the attack may be. The sick room should be well ventilated and kept at an even temperature of about 65° F. The patient should be allowed to assume that position in bed which he finds most comfortable. He should be forbidden to talk, and should be prevented from making any unnecessary movements, and a nutritious diet without stimulants should be given him. If he is robust, three or four leeches may be applied over the seat of pain, and followed by an anodyne poultice. Hypodermics of morphine must be given in sufficient quantities to relieve all pain. After the first week the morphine can usually be discontinued and the patient will be able to sit up, and, at the end of three weeks, to resume his ordinary occupation unless it requires great physical exertion. If there is an abundant plastic or serous exudation the convalescence will be reached more slowly and the recovery will be less complete. In such cases there will be some crippling of the lung, and pain and uneasiness in the affected side will continue for months. After recovery it is well to inform the patient that he may expect pain after active physical exercise. If the patient presents the signs of anæmia, the syrup of the iodide of iron should be given in teaspoonful doses three or four times each day. Stimulants should rarely be allowed before the third week. Counter-irritation by means of cups and blisters is rarely of service in the treatment of this form of pleurisy. I have found in some cases, when the pain in the side continued after the friction sound had disappeared, that the use of the constant current over the affected side for twenty minutes at a time gives almost instantaneous relief.

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If recovery takes place the fluid disappears by absorption, the fibrin undergoes fatty metamorphosis, liquefies, and slowly disappears. As the fluid disappears the thickened pleural surfaces come in contact, and more or less extensive adhesions form between the two surfaces. On account of the changes which take place in the pulmonary pleura the lung-tissue does not expand to its normal dimensions, but more or less retraction of the chest walls on the affected side takes place. The longer the fluid remains in the pleural cavity the more extensive will be the retraction. As the fluid disappears, the organs which have been displaced by its pressure return to their normal positions. If the retraction is considerable they will be displaced upward, and the heart may be drawn from its normal position to the right or left.

Etiology.—The causes of this form of pleurisy may be the same as those of plastic pleurisy. In a large proportion of cases it is secondary to some form of organic disease, as chronic Bright's disease of the kidneys, pulmonary phthisis, etc. Occasionally it seems to occur idiopathically, or at least from causes not well understood. It is a clinical fact familiar to every careful observer that sub-acute pleurisy is not infrequently the first step to the development of phthisis. It is claimed that serofibrinous pleurisy is always of tubercular origin. While it is undoubtedly often induced by tuberculosis of the pleura, it is at present not proven that such is its sole cause. The weak and enfeebled, rather than the strong and robust, are liable to attacks of sub-acute pleurisy.

Symptoms.—This form of pleurisy may come on suddenly with active symptoms, or insidiously with very mild symptoms. In the majority of cases the symptoms are mild. There is no chill of invasion; it comes on insidiously after exposure to wet, cold, and fatigue, in the enfeebled or in those who are suffering from some chronic disease. It is rarely attended by any noticeable pain in the side, or at least not by the severe pain which attends acute pleurisy. On close questioning the patient will state that some time before exposure an uneasy sensation in the affected side, attended occasionally by a sharp pain of short duration, was experienced. This form of pleurisy is often so insidious in its approach that the patient will be unable to tell when he commenced to be sick; for a period of several weeks he will have gradually lost flesh and strength, yet will have been able to attend to his ordinary avocations if they required but little physical exertion. There will be slight dyspnœa on exertion, with slight febrile excitement at night. Sometimes there is an almost continual cough with a scanty muco-purulent expectoration; at other times the cough will be entirely absent. Usually when this class of patients consult a physician the only subjective symptoms will be a frequent, small, feeble pulse, and slight heat and dryness of the skin, the temperature rarely rising above 101° F. The countenance will be pale and anxious, and the breathing short and catching in character. On speaking, especially after exercise, the sentences are uttered in a broken, interrupted manner. The patient will be unable to lie comfortably except on his back, or on the affected side with his head slightly elevated. The pulse, usually small and feeble, will vary from 110 to 120 beats in the minute; in fact, there will be no subjective symptom which will enable one to reach a positive diagnosis.

In those cases which are ushered in by active symptoms the invasion will resemble that of acute pleurisy. There will be rigors followed by a temperature of 102° or 103° F.; the pulse will be full and frequent, the pain on the affected side well marked and the breathing rapid and shallow. Patients will sometimes ascribe the pain to the lumbar region, as in nephritic colic, for which it is sometimes mistaken. After a few days the febrile symptoms abate, but do not entirely subside, and the serous effusion, which is much larger than that in acute pleurisy, for a time steadily increases, then remains stationary for a number of days or even weeks, and then there is a sudden renewal of the febrile symptoms, the dyspnœa is greatly increased, the cough becomes more constant and harassing, the patient is unable to lie down, and the fluid rapidly increases; in twenty-four hours the pleural cavity,—which previously has been only half full of fluid,—becomes entirely filled, and the dyspnœa becomes so urgent, and the danger from collateral congestion and œdema of the opposite lung so imminent, that immediate relief is demanded by paracentesis. With the rapid increase of the effusion the pain in the side subsides. However ill-defined the rational symptoms may be in acute pleurisy, its physical signs are more distinctive than in any other thoracic disease.

Physical Signs.—The physical signs of pleurisy with effusion will vary with the amount of the fluid effusion. At its onset, before there is much fluid effusion, a friction sound will be heard over the affected side with more or less feebleness of the respiratory sound. After the pleural cavity partly fills with fluid, the vocal fremitus will be diminished or absent at the bottom of the pleural sac below the level of the fluid; there will be flatness on percussion, and an absence of vocal and respiratory sounds. A change in the position of the patient will change the level of the fluid and the line of flatness. Above the level of the fluid the percussion resonance will be normal or exaggerated, and in some cases tympanitic in quality. The respiratory murmur will be exaggerated, and at the level of the fluid it may assume a bronchial character. The vocal sounds may be intensified, or a distinct bronchial voice may be heard. All of these physical signs are most marked posteriorly.

When the pleural cavity is completely filled with fluid, and the lung is compressed backward against the spinal column, important modifications in the physical signs take place.

Inspection will show an enlargement of the affected side, and a bulging of the intercostal spaces. The respiratory movements on the affected side will be changed from an up-and-out movement to a direct up-and-down motion, while on the unaffected side the expansive respiratory movements are increased. If the effusion is in the left pleural cavity the heart will be displaced to the right, and the apex beat may be noticed under the right nipple; if it occupies the right pleural cavity the apex beat will be carried to the left, beyond its normal position.

The circumference of the affected side at the end of expiration, will be one or two inches greater than that of the healthy side; but at the end of inspiration the difference will be but slight. The expansive motion in inspiration on the healthy side may be two or three inches greater than on the affected side.

in a limited number without lesions to account for it. A large serous effusion may take place suddenly, and cause death by its interference with respiration and circulation. Cases may be protracted over a period of months, and finally a sero-fibrinous effusion may change into a sero-purulent one, and a sub-acute pleurisy may thus become an empyema ; in giving a prognosis it is to be remembered that in most cases that recover, more or less extensive adhesions result, which cause permanent crippling of the lung, and lead to the development of more or less extensive compensatory emphysema, chronic bronchitis, and fibroid induration of lung-tissue. When the new tissue formations are extensive, and the general health much impaired, in those who have a strong hereditary or acquired tendency to pulmonary phthisis, there is always danger that the new tissue may become tubercular, even when the pleurisy was not of tubercular origin.

Treatment.—The main thing to be accomplished in the treatment of sub-acute pleurisy is to remove the fluid effusion as rapidly as possible, at the same time taking care to sustain the patient. The principal means which have been employed for the accomplishment of this object are hydragogue cathartics, diuretics, diaphoretics, and blisters applied in succession over different parts of the affected side. On account of the anæmic condition of the majority of these patients, general or local bleeding, as well as the use of mercury, is now very rarely employed ; for a like reason I very much question the beneficial effects claimed for cathartics, diuretics and blisters ; it is very questionable if the condition which favors the absorption of the fluid in the pleural cavity can be reached by the employment of any of the so-called depurative remedial agents. It is claimed that the use of hydragogue cathartics and diuretics quickly removes large quantities of fluid from the body, and consequently the fluid portion of the blood is greatly diminished, and that whenever a cavity contains fluid, the absorbents and blood-vessels of the part take it up to replace that lost by the blood, and thus fluid in the pleural cavity is absorbed. There is little doubt but that hydragogue cathartics and diuretics will hasten the absorption of non-inflammatory serous effusion in simple hydrothorax, but there is no evidence that they have power to promote the absorption of inflammatory products from the pleural cavity in sub-acute pleurisy. It is certain that by the action of these depurative means the vital powers of the patient are greatly enfeebled and the processes of digestion and nutrition seriously interfered with. It is also well established that when the nutritive processes are going on most rapidly absorption takes place most rapidly. Consequently anything that interferes with these processes is contra-indicated in the treatment of this form of pleurisy. There are also other conditions which greatly impede the absorption of the fluid effusion in pleurisy. When the pleural cavity is distended with fluid, its absorption is impeded or prevented by the obstruction offered to the flow of blood through the pleural and sub-pleural vessels by the pressure. Under such circumstances it is useless to resort to diuretics and hydragogue cathartics. The mechanical withdrawal of a sufficient amount of liquid to relieve the tension of the cavity and remove the pressure from the lung and the sub-pleural veins is an absolute necessity before the processes of absorption can commence. If the surface of the pleura is covered by a thick layer of exudative material, this layer is interposed between the sub-pleural vessels and the fluid effusion, and must greatly interfere with the absorption of the liquid; as it becomes thicker and thicker by successive deposits of fibrin, it is obvious that the longer the liquid remains in the pleural cavity the thicker the fibrinous deposit becomes, and the less is the probability that the liquid will be absorbed. Against these conditions cathartics and diuretics are powerless.

For many years I have rarely employed any depurative agents in the treatment of sub-acute pleurisy. The remedial agent which seems to me to have the greatest power in promoting the absorption of an effusion is the syrup of the iodide of iron. In connection with the administration of iron the patient should take the largest amount of the most nutritious food, with wine or some form of alcoholic stimulant. The principle of treatment is to employ all those remedial and hygienic measures which improve nutrition.

As so little can be done by medication to excite or hasten the absorption of pleuritic effusion, the question of the employment of mechanical means for its removal presents itself. There is some difference of opinion in the profession upon this point. One class of observers claim that a single removal of the fluid is of little service, and that the danger of admitting air into the pleural cavity is so great that if the operation is frequently performed a serous effusion is very apt to be changed into a purulent one, thus jeopardizing life. On the other hand, the advocates of the operation maintain that if the fluid is permitted to remain in the pleural cavity it becomes purulent. The causes which impede or render impossible the absorption of the fluid seem to me reasons in favor of its early mechanical removal, especially as the practice of aspiration has inaugurated a new era in the management of these cases, and has removed all objections to such early removal. When a perfect instrument is used and a small needle introduced into the pleural cavity, the entrance of air is impossible. In any case of pleurisy, when the accumulated fluid remains stationary for one week, or is increasing after the cavity has become half filled, and especially when the cavity is completely full, there should be no delay in aspirating. With every day that the lung remains compressed, and with every addition to the plastic deposit upon the pleural surfaces, the chances of its absorption are diminished, and the danger that the lung will be permanently crippled is increased.

The following rules should be observed in the performance of aspiration of the chest. Place the body of the patient in the erect posture, leaning somewhat forward, with the arm of the affected side thrown partly across the chest. This position of the arm is preferable to any other for the reason that the integument is not made unnaturally tense over the intercostal spaces. Select a needle of small size for the first tapping, and introduce it to the depth of at least one inch into the fifth or sixth intercostal space, at the junction of the axillary and infra-scapular regions. After the needle has been introduced the fluid may be permitted to flow through the instrument until the patient complains of a sense of constriction about the chest, when the withdrawal of the fluid must be stopped. The amount of fluid that can be withdrawn at the first aspiration, if the cavity is distended, will depend upon the length of time which the fluid has remained in the pleural cavity. If it has accumulated rapidly, the cavity may frequently be emptied without giving rise to any unpleasant symptoms; if, however, it has been slow in its accumulation and the pleural cavity has contained a large quantity of fluid for a considerable time, only a small amount can be withdrawn without producing a severe attack of dyspnœa. When this is the case the patient may be permitted to remain quiet for a few days, and then the operation should be repeated as often as it can be without producing unpleasant symptoms. The sense of constriction about the chest always indicates that no more fluid should be withdrawn at that time. It is claimed by some that aspiration of the chest in pleurisy may cause death suddenly or within twenty-four hours after the aspiration, and that the cause of death under such circumstances cannot be accounted for, as there are no lesions found after death which are sufficient to produce it. I cannot understand how this is possible if the aspiration is performed with sufficient care; it certainly has never happened in any of my cases. I can conceive how the sudden withdrawal of a large quantity of fluid from the left chest might cause a severe attack of syncope from which a crippled heart might not rally. I have no hesitation in recommending this operation in all cases, provided it be done according to the rules just given, and I am not disposed to delay aspiration long after the pleural cavity has once become filled with fluid, for I am convinced that its early removal tends to promote a more rapid recovery, and prevents those changes in the pleura which lead not only to a tedious convalescence, but also to an incomplete ultimate recovery.

SUPPURATIVE PLEURISY, OR EMPYEMA.

(*Pleurisy with a Sero-fibrinous and Purulent Effusion.*)

This is a suppurative inflammation of the whole pleura, usually confined to one side of the chest. It may be *primary* or *secondary*. When it is primary it usually commences as an acute affection; when secondary, it is sub-acute or chronic from its commencement.

Morbid Anatomy.—The pathological changes in this form of pleurisy are most extensive and best marked on the costal, diaphragmatic, and mediastinal portions of the pleural membrane. In primary suppurative pleurisy there is poured out a large amount of plastic material which undergoes histological transformation into pus, and thus a large amount of thick pus is rapidly formed in the pleural cavity. In the secondary variety of suppurative pleurisy a sero-purulent effusion will slowly accumulate in the pleural cavity, varying in consistency in different cases, sometimes being quite thin and mainly composed of serum, at others extremely thick and containing comparatively little serum. This purulent fluid usually occupies the most dependent portion of the pleural cavity; it may, however, be confined either to the posterior or anterior half of the chest by old adhesions.

The manner in which large purulent accumulations are formed in the pleural cavity is as follows:—in acute suppurative pleurisy with sero-fibrinous exudation, a large number of pus cells form in the connective-tissue of the pleura and also on its surface, from which they are washed into the cavity, along with the fibrinous exudation, by the serous effusion. Sometimes the accumulation is very large and takes place very rapidly. This is characteristic of the pleurisies which occur in connection with pyæmia. A sero-fibrinous exudation may become purulent when a fresh cause of inflammatory irritation gives rise to an active cell-exudation; the new irritation may come from the admission of air into the pleural cavity, or from some change in the fluid which has previously occupied the cavity, or, perhaps, from suddenly developed sepsis. Under these circumstances a variety of cell-formative processes are established. Some are produced in the plastic exudation, and some in the pleura itself. The clear serum becomes turbid, shreds of false membrane are loosened from their connection with the underlying tissue and undergo liquefaction, and the whole, or a large portion of the pleural membrane becomes a suppurating surface, and thus a large amount of pus is formed in the pleural cavity. If the pleural cavity is aspirated at the commencement of the purulent process in such cases, the first fluid removed will be found to contain a moderate number of cells; at a second operation, a week or two later, a large number of cells may be found, and it is usual under such circumstances to attribute the increased number of cells to the effects of the first aspiration. This is not, however, a legitimate inference, for the increase in the cell development is the natural result of the morbid process which was in operation at the first aspiration.

Purulent accumulations in the pleural cavity may become so large that death may ensue in consequence of the depression caused by their production. The tendency of suppurative pleurisy is never toward convalescence, unless by spontaneous openings. The inflammatory process is not limited to the pleura; it may extend from the costal pleura to the connective-tissue underneath, to the periosteum of the ribs, causing necrosis, or it may perforate the walls of the chest and be discharged externally. In some instances the lung may be perforated and the discharge take place through a bronchial tube, or the diaphragm may be perforated and the pus find its way into the abdominal cavity. If the patient survives the emptying of the pleural cavity, repair is accomplished by the rapid and abundant formation of cicatricial tissue; the pleural cavity is contracted in every direction like a huge cicatrix, the chest walls on the affected side retract to their fullest extent, and the thoracic and abdominal viscera are dragged out of their normal positions to help fill the space formerly occupied by lung-tissue. In some cases of circumscribed empyema the fluid portion of the pus is absorbed and the solid constituents undergo cheesy transformation, the salts of lime are deposited and the thickened pleura becomes calcified. The bony or calcareous plates which are occasionally found in the pleural cavity at post mortem examinations usually have their origin in an empyema.

Etiology.—The cause of suppurative pleurisy is not always readily determined. It may be of traumatic origin. When it occurs spontaneously it is always associated with some vice of constitution, such as results from some exhausting disease, or the debility which attends chronic alcoholismus. It often complicates acute and chronic infectious diseases. In the enfeebled it is frequently developed from a sero-fibrinous pleurisy which has continued for a long time, but in most instances, under such circumstances, the occurrence of the suppurative process is due to some new infection, or to some new local excitement of pleuritic inflammation. Chronic tubercular pleurisies are very apt to be suppurative in character. It may be secondary to abscess of the liver, or to the opening into the pleural cavity of a vomica in the lung in chronic phthisis. An abscess in the abdominal cavity or in the chest walls may open into the pleura, and establish a suppurative pleurisy.

Symptoms.—The rational symptoms of empyema will vary with its character. Those cases in which the inflammatory processes are acute at the onset, accompanied by the rapid production of fibrin and pus, will be ushered in by chills, followed by a rapid rise in temperature, and a rapid, full pulse. There will be severe pain in the affected side, with the signs of great prostration. The prostration is greater than in acute fibrinous pleurisy, and the countenance early assumes an anxious expression ; if the inflammatory products become gangrenous the prostration is extreme, and the patient presents the appearance of one suffering from peritonitis ; typhoid symptoms manifest themselves very early ; and these cases usually terminate fatally within two or three weeks. In other cases the active symptoms subside after a week or ten days, and symptoms of the more chronic form of empyema are developed. The symptoms of chronic empyema are often very obscure ; the presence of pus in the pleural cavity in these cases cannot be determined either by the rational symptoms or by physical signs. The patient rarely suffers from local pain—there is simply a sense of uneasiness, or weight in the affected side ; there is a gradual loss of flesh and strength ; the countenance assumes a pale, anxious expression ; and there is an irregular diurnal chill followed by profuse sweats. Ordinarily the patient has a cough with a scanty muco-purulent expectoration, the voice becomes weak and there is more or less dyspnœa, and the patient gradually assumes the appearance of one in the last stages of pulmonary phthisis. If empyema occurs as a complication of septicæmia or pyæmia, its commencement is also, at times, very insidious. In these conditions patients sometimes pass into a semi-comatose state. Not infrequently pyæmic patients make no complaints which would direct attention to the pleura, and the pleural cavity may be found two-thirds full of pus, without having given a single symptom of its presence.

If an empyema is about to open externally, it will usually make itself manifest by a protrusion between the ribs, which gives a sense of fluctuation, and after a time grows red, and finally a valvular opening is formed and pus is discharged. If the opening takes place through the lung into a bronchial tube, the discharge of pus is ordinarily preceded by symptoms of pneumonia ; the patient will have a chill, followed by a cough and a more or less profuse expectoration containing blood, which will be followed by a profuse purulent expectoration, which will afford marked relief. The profuse purulent expectoration will occur two or three times a day; the chest walls gradually retract, and finally the expectoration will cease altogether and the pleural cavity become obliterated. If the opening takes place into the peritoneal cavity, its occurrence is usually followed by a rapidly fatal peritonitis. If the communication is established with the intestinal canal, pus will appear in the discharges from the bowels. If the patient survives the establishment of either an external or internal opening, spontaneous or artificial, a connective-tissue development takes place in the pleural cavity, and as the contents of the cavity are being discharged retraction of the chest and displacement of the abdominal and thoracic viscera take place; this process is necessarily slow, and years may elapse before it is completed.

Physical Signs.—The physical signs of empyema are essentially the same as those of pleurisy with effusion, except that the level of the fluid is not so readily changed by a change in the position of the patient; if, however, the physical signs indicate the existence of fluid in the pleural cavity in one who is very much debilitated, who has a constant cough with muco-purulent expectoration, hectic fever with profuse sweats, and whose history indicates that the fluid has existed for a long time, one may be almost certain that the fluid is purulent.

Differential Diagnosis.—Unless a fistulous opening exist, a positive diagnosis of empyema is impossible, except by an explorative puncture. When such a puncture has been made, and some of the contents of the pleural cavity have been drawn off and subjected to microscopical examination, it is not possible to confound an empyema with any other thoracic disease.

Prognosis.—The prognosis in empyema is unfavorable. In acute suppurative pleurisy death may occur at the end of one or two weeks. In the more chronic cases it may take place from gangrene produced by decomposition of the inflammatory products in the pleural cavity. Statistics show that in empyema of slow development, where spontaneous openings occur, about one in five recover, while in those in whom artificial openings are established the rate of mortality is greater. This class of patients die from the exhaustion produced by the accumulation of large quantities of pus, and from the exhaustion which attends a prolonged and abundant purulent discharge. A large number of these patients live for a year or more. The judicious use of the aspirator will tend to render the prognosis more favorable in the acute cases. I am confident that the early introduction of a drainage tube into the pleural cavity in chronic cases will save many lives. In estimating the prognosis in this disease, the treatment to which the patient is to be subjected must always be considered.

The majority of empyemic children recover, while in adults, although for a time recovery seems almost certain, phthisis is sooner or later developed.

Treatment.—In the treatment of this affection it is useless to attempt to produce absorption of the purulent accumulation by remedial agents. Its removal can only be accomplished by mechanical means—either by aspiration or by making a permanent opening in the chest walls. If aspiration is resorted to a large-sized needle should be used, and no attempt should be made to empty the cavity at the first operation. Remove only a small portion of the accumulation, being governed by the same rules which have been given for the removal of serous effusions, and allow from three to six days to elapse between successive aspirations. At each aspiration something in excess of the amount which was taken at the previous séance should be removed. Never continue the removal of pus in empyema after the patient complains of constriction in breathing, even though only three or four ounces have been removed. If the aspiration is to be successful the fluid will become thinner at each aspiration, and retraction of the chest wall will be noticed. If the fluid becomes thicker and emits an unpleasant odor, a permanent opening should immediately be made. In empyema occurring with septicæmia and pyæmia the accumulation will exceed in quantity that removed, unless the aspirator is used daily. Under such circumstances a free opening should be made. If a permanent opening is to be made, let it be made in the axillary line in the seventh or eighth intercostal space. After a free opening has been made into the chest cavity, a quarter-inch rubber drainage tube should be introduced, and so fastened that it will remain. Often when there is little space between the ribs a portion of bone should be removed, that the tube may not be compressed during respiration. A double drainage, by making two openings in the chest cavity, is rarely advisable. As regards the washing out of the pleural cavity after the introduction of the drainage tube, although it is strongly advocated by some, my recent experience is very positively *against it*, even when the purulent discharge has an offensive odor. Thrice have I had reason to believe that my patients have died from the direct effects of washing out the pleural cavity with a weak solution of carbolic acid. From the commencement empyemic patients must receive a most nutritious diet with moderate stimulation. Tonics, such as quinine and iron, are always indicated; cod-liver oil will be of service if it does not interfere with stomach digestion. The patient must be kept in the open air as much as possible, and a change of climate is often attended by very marked improvement. The majority of cases of empyemic children will recover if aspiration is performed early and repeated at short intervals. In most adults it will be necessary to make a permanent opening in the chest wall.

INTERSTITIAL OR ADHESIVE PLEURISY.

Interstitial pleurisy may commence as a primary disease, or be the sequela of an acute, plastic, or a sub-acute effusive pleurisy. In any case there are more or less extensive new connective-tissue formations over a greater or less extent of the pleural surface.

Morbid Anatomy.—The essential lesion in this form of pleurisy is the formation of new connective-tissue over the pleural surfaces. This hyperplasia may or may not have its origin in a pleurisy which gives fibrin, serum, or pus as its product. As a result the pleura becomes thickened sometimes to the extent of half an inch; an equally important and constant lesion is adhesion between the costal and pulmonary pleuræ. These adhesions, however they originate, are progressive, and, after a time, become very extensive. In some cases the two surfaces may become closely agglutinated to each other throughout their whole extent, and then the entire space between them may be obliterated. As a result of these adhesions the expansive motion of the lungs is interfered with, and sometimes to such an extent as to cause constant dyspnœa. The heart may be displaced to the right and backward. In one case where the adhesions were extensive over both lungs, the heart cavities were much dilated, and, a loud ventricular murmur always being heard, valvular disease of the heart was diagnosticated by good observers who saw the case during life. This form of pleurisy often leads to the development of fibrous phthisis.

Etiology.—It occurs most frequently in rheumatic and gouty subjects. It is often associated with general fibroid degeneration. When it occurs as a sequela of sero-plastic pleurisy it is developed with the disappearance of the sero-plastic effusion.

Symptoms.—Its development is always slow and often intermittent. Its most constant early symptom is a dull pain over the affected part, accompanied by a sense of constriction. An early symptom is dyspnœa on exertion, which steadily increases with the advance of the disease, and becomes so severe that even slight exertion, such as going up stairs, will give rise to such severe paroxysms that signs of collapse sometimes follow. There is usually a dry, hacking cough, frequently attended by free bronchial hemorrhages. I have frequently found these pleuritic adhesions the only apparent cause of quite profuse bronchial hemorrhage. As the adhesion becomes extensive, the patient loses flesh and strength, and in some cases the ordinary symptoms of chronic phthisis are present. If there is much displacement of the heart the patient will be troubled with cardiac palpitation on slight excitement or physical exertion, so that his dyspnœa and cough are often supposed to be due to some obscure cardiac lesion. Often after this class of patients have suffered much and seem to be steadily getting worse, periods of remission occur, during which for months and perhaps years they will seem to be recovering. The appetite returns, they gain flesh and strength, the dyspnœa becomes less, and then, while they are apparently recovering, they suddenly get worse, all their aggravating symptoms return greatly exacerbated, and they rapidly pass into a decline.

Physical Signs.—*Inspection* shows diminished expansive motion of the affected side, or of the entire chest if both pleuræ are affected.

Palpation shows diminished vocal fremitus over the seat of the adhesions.

Careful *mensuration* of the chest will often establish the diagnosis when doubt exists as to the exact character of the changes.

On *percussion* there will be slight dulness, which will be most marked at the part where the adhesions are most extensive.

On *auscultation* the respiratory sound will be feeble, sometimes scarcely audible even during a full inspiration; friction sounds will be heard. These friction sounds are creaking or crepitating in character, very loud, and often resemble mucous râles and gurgles, for which they are sometimes mistaken, but the loss of chest expansion and the feebleness of the respiratory sounds will readily correct the mistake.

air does not enter the pleural cavity through the opening in the chest wall, but comes from the lung through an opening in the pulmonary pleura, the lung being torn at the same time that the opening is made through the walls of the chest. The commonest example is in connection with fracture of the ribs, in which the lung is sometimes torn by the broken end of the bone, and air escapes through the rent into the pleural cavity. Entrance of air into the pleural cavity usually occurs either in connection with pulmonary phthisis, gangrene of the lung, empyema, or pulmonary emphysema. It is most frequently met with in connection with pulmonary phthisis. Abscess of the bronchial glands, and ulceration of the œsophagus or stomach, may lead to it. When an empyema has existed for a long time an opening may be established by ulceration through the lung into a bronchial tube, thus permitting the fluid to be expectorated, and air to enter the pleural cavity. In pulmonary emphysema, a sac containing air which has been formed upon the surface of the lung may rupture, and air enter the pleural cavity and develop pneumothorax; the consequent pleurisy will rapidly develop a pyopneumothorax. At the post-mortem examination of one who has died of pyopneumothorax, it is often difficult, and sometimes impossible, to find the opening in the pulmonary pleura, for the reason that in some instances it becomes covered with a fibrinous deposit, and in others the opening has been closed some time before death by an inflammatory process in the lung substance about the opening.

Symptoms.—The symptoms which attend perforation of a lung, and the escape of air into a pleural cavity, are usually well marked, but they are somewhat variable. First, there is a class of cases in which the symptoms are severe in character, the patient is suddenly seized with a sense of faintness followed by hurried respiration and great dyspnœa. Pain may or may not be a symptom; its existence indicates inflammation. The dyspnœa is in part mechanical, in part reflex. It is extreme, comes on suddenly, is soon followed by well-developed cyanosis, the patient passes rapidly into a state of collapse, and, in some instances, death occurs in a few hours. Usually, however, the patient survives the shock of the perforation, and, after a time, becomes comparatively comfortable, suffering, however, from more or less dyspnœa. He is unable to lie down, able only to recline upon the affected side. Some say they experienced a sense of "tearing," and felt as if a fluid "were being poured inside the chest."

As the pleural cavity becomes filled with the fluid effusion (which may result from the attending pleuritic inflammation), the dyspnœa and cyanosis increase, and general dropsy gradually develops. Aseptic air alone will not cause inflammation or rise in temperature. It is a purulent accumulation in the pleural cavity which proves fatal, and not the pneumothorax, for with its development the temperature rises and the patient becomes more manifestly hectic, if hectic has previously existed. When the purulent accumulation becomes very abundant the patient dies from the exhaustion produced by the intensity of the fever or from collateral hyperæmia and œdema of the opposite lung. In some cases the symptoms which attend the entrance of air into the pleural cavity come on more insidiously. The difficulty of breathing may be gradually developed, and the existence of air in the pleural cavity may not be recognized until after considerable fluid has collected in the pleural cavity. When pneumothorax occurs in connection with pulmonary phthisis, its occurrence is marked by very active symptoms, pain being prominent, followed by all the evidences of collapse. When occurring in connection with pulmonary emphysema its development is very insidious.

Physical Signs.—The physical signs of pyopneumothorax are very characteristic, and, if properly appreciated, will always enable one to recognize its existence. By *inspection* there will be noticed a bulging of the intercostal space and an increase in the size of the affected side, which becomes more prominent than in sub-acute pleurisy and has a "rounded" look. There will be the displacement of viscera seen in sub-acute pleurisy when the pleural cavity is distended with fluid, and there will be *absence of motion* on the affected side, while upon the unaffected side the respiratory movement will be also decreased in force and frequency, but to no such great extent, the breathing being almost wholly abdominal.

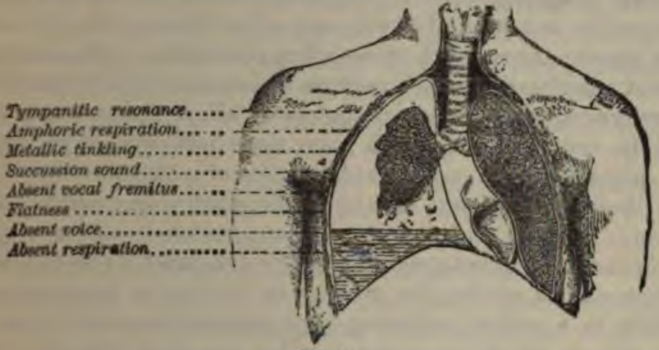

FIG. 41.
Diagram Illustrating the Physical Signs of Pyopneumothorax.

Upon *palpation* there will be entire absence of vocal fremitus upon the affected side, unless there are old adhesions. The heart, at the same time, is felt pushed from its normal site. Thus far there is no difference between the physical evidences of pyopneumothorax and sub-acute pleurisy.

On *percussion*, when the patient is sitting or standing there will be tympanitic resonance from the summit of the affected side to the level of the fluid. Below the level of the fluid there will be complete flatness.[1] A change in the position of the patient will change the level of the fluid, and also, of course, the character and site of the percussion note.

Upon *auscultation* there will be found an entire absence of all respiratory and vocal sounds below the level of the fluid; but, as soon as its level is reached, if the opening from the bronchial tube which admits the air into the pleural cavity still remains pervious, amphoric respiration or "echo" will be heard, and it will be *metallic* in character. Metallic tinkling is almost uniformly associated with amphoric respiration, and is produced in a variety of ways. It may be produced by agitation of the liquid from the vibration of the voice, or by coughing and full inspiration, or by dropping of liquid from the walls of the cavity upon the surface of the fluid. It is more frequently produced by agitation of the fluid from speaking and coughing. The characteristic physical sign of this disease is the *succussion sound*, which is a metallic, splashing sound, produced by abruptly shaking the chest while the ear is resting upon the surface. Over the affected side no vesicular breathing can be heard, while over the healthy side the vesicular breathing is exaggerated.

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[1] Except in pyopneumothorax it is rare to find *an exactly horizontal line* of demarcation with pleural effusions.

Differential Diagnosis.—When pyopneumothorax is fully developed, it is scarcely possible to confound it with any other disease, but it is possible to confound pneumothorax with some other conditions. The only physical evidences of a perforation which permits the entrance of air into the pleural cavity, are tympanitic percussion, absence of all respiratory sounds on the affected side, and intense dyspnœa; the same development of signs might occur in connection with complete *obstruction* of a large *bronchus*. Again, it is said that pneumothorax may be confounded with extreme pulmonary *emphysema*. Patients suffering from these diseases present a somewhat similar appearance; in both classes there is tympanitic percussion, but in the emphysematous patient the tympanitic percussion is present over *both* lungs, while in a patient suffering from pneumothorax it is present only upon the side on which the perforation has occurred. Besides, there is a vesicular element to the tympanitic note in emphysema never found in pneumothorax. In emphysema there will also be heard some respiratory sounds. The expiration is prolonged and *low* pitched in emphysema; not so in pneumothorax. The breathing is broncho-vesicular in emphysema; not so in pneumothorax, where respiratory sounds are *absent*. Succussion is present in pyopneumothorax; *not* in emphysema. If errors in the differential diagnosis of these two conditions are possible, they will be made at the commencement of the attack.

A large *cavity* in the lung substance may be mistaken for pyopneumothorax. I have never met with a pulmonary cavity of sufficient size and with the conditions to produce the succussion sound. Amphoric respiration and metallic tinkling may be developed in a large cavity, but the succussion sound will be absent; on the other hand, when amphoric respiration and metallic tinkling are present in hydropneumothorax the succussion sound will also be present. In a cavity râles would be loud and numerous; vocal fremitus is very often exaggerated; the chest wall above it would probably be slightly *depressed*, and finally, the heart, etc., would not be displaced.

With a knowledge of the history of the patient and a proper appreciation of the physical signs, it is hardly possible to confound pyopneumothorax with any other form of disease. In no other disease are the physical signs so characteristic and unequivocal, and in a large proportion of cases the rational symptoms are equally diagnostic.

Prognosis.—The prognosis in pyopneumothorax is always unfavorable. All authorities agree that when it occurs in connection with advanced pulmonary phthisis or gangrene it generally proves fatal within five or six days; but in pneumothorax without pleurisy the prognosis is favorable. Every day the patient lives betters the outlook; the majority of fatal cases die within two days, the period of survival in the remainder rarely extending beyond the sixth day. When recovery has taken place in cases of pyopneumothorax, either they have been of traumatic origin, the result of great muscular strain in connection with extensive pulmonary emphysema, or an empyema has discharged itself through a bronchus. There is record of a few recoveries where the rupture occurred in the early stage of phthisis. When recovery does take place it is reached in the following manner:—plastic material is poured out in the tissue surrounding the opening in sufficient quantities to completely close it; the air and fluid are thus imprisoned in the pleural cavity; the air is rapidly absorbed by the pleural membrane, and if the closure is sufficiently firm to persist after the air has been removed, the case will be thus changed from one of pyopneumothorax to one of empyema. Cases have been related in which perforation of the lung and pneumothorax were present without any fluid collecting in the pleural cavity. Such cases are of such rare occurrence that they can hardly be taken into consideration as regards prognosis.

Treatment.—The treatment of this affection is almost necessarily palliative. At the very onset of the attack, when the patient is suffering from the shock of the perforation, a full hypodermic injection of morphine will be found of service, and it may be repeated once or twice a day for the first few days. If the patient survives for a few days, stimulants may be advantageously administered, and he must be sustained by a most nutritious diet. Among drugs, musk and chloroform are recommended by so good an authority as Dr. Walshe. Hot poultices and soothing fomentations give relief when applied over the chest. The quebracho bark is now given for relief of the dyspnœa. When the dyspnœa is extreme and the distress of the patient is very great, and a considerable quantity of fluid has accumulated in the pleural cavity, the question will arise whether a free opening shall be made through the chest walls. As a rule, this must be regarded as a palliative measure, and should be resorted to only in extreme cases. If it be resorted to, a fine trochar should be inserted into the chest, and the air permitted to escape through a connecting tube under water, until an equilibrium has been established. It may give relief for a time, and it is justifiable to resort to it when the fluid collection is abundant and the febrile excitement is intense. It may delay the fatal termination. Walshe recommends general bleeding or dry cupping.[1]

HYDROTHORAX.

Hydrothorax is a non-inflammatory fluid effusion into one or both pleural cavities; it often accompanies general dropsy. The fluid is generally clear, of a yellowish-green color, and may be sufficient in quantity to compress to a considerable extent one or both lungs. It may occur in any chronic exhausting disease which causes general hydræmia. It rarely occurs as the sole morbid process in the human body. In a large number of autopsies a small amount of clear or bloody serum will be found in the pleural cavities, which is merely the result of post-mortem changes; such conditions should not be regarded as evidence of hydrothorax.

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[1] *Dis. of Heart, etc.*

Etiology.—Any disease or condition (*e. g.*, mitral disease especially) that impedes, and raises the pressure in, the venous circulation will cause it. Thus it may be caused by the pressure of enlarged glands; tumors and venous thrombi may induce it; also diseases of the heart and kidneys, the cancerous and other cachexiæ. Hydrothorax generally occurs in connection with general anasarca, such as is developed in Bright's disease.

Symptoms.—It generally comes on insidiously, and its development is attended by no febrile symptoms. Its occurrence is marked by steadily increasing dyspnœa, until the patient reaches a condition of extreme distress, and orthopnœa; the lips become livid, the finger-ends blue, and the respiration gasping. He is unable to lie down and can speak only in monosyllables. On physical examination there will be found the signs of fluid in both pleural cavities. There may be a short, dry cough. If the effusion is large, the action of the heart will be embarrassed, as shown by a small, feeble pulse. All the phenomena which attend this condition are due to mechanical pressure caused by the presence of fluid in the pleural cavities, and patients die cyanosed as the result of diminished breathing capacity.

Differential Diagnosis.—Ordinarily the diagnosis of hydrothorax is readily made. It may be confounded with *sub-acute pleurisy*, but generally the history of the case will determine the character of the effusion. Then, its simultaneous occurrence on both sides in connection with general dropsy, without any irritant or attendant fever, will be sufficient to enable one to make the diagnosis of hydrothorax. It may be mistaken for *pulmonary œdema*; the two conditions are very likely to occur together. but in pulmonary œdema a crackling sound will be heard over the œdematous lung, which sound is not present in hydrothorax, and there will be copious, watery (perhaps blood-stained) expectoration, which is absent in hydrothorax. In *emphysema* the increased resonance, and in *bronchitis* the sputum and râles, will suffice to differentiate the three conditions. An *enlarged* (painless) *liver* will not be mistaken for dropsy of the chest. The physical sign of hydrothorax is fluid in both pleural cavities which is freely movable by a change in the position of the patient, and is not attended by friction sounds or vocal fremitus. The introduction of a trochar and withdrawal of the fluid will decide the case.

Prognosis.—The danger attending hydrothorax will depend to a great extent on the general condition of the patient at the time of its occurrence. When it occurs in connection with general anasarca in Bright's disease, or in extensive heart disease, it may prove the direct cause of death. The majority of cases yield readily to treatment, and life may be prolonged months, even years, by judicious management.

Treatment.—The general treatment of hydrothorax corresponds to that for the removal of dropsical accumulations in other parts of the body. It is a simple dropsical effusion, and can be removed by the administration of remedies which diminish the quantity of water in the blood. Such remedies are the hydragogue cathartics, diuretics and that general class of agents employed for the removal of fluid from the areolar tissue. Elaterium is the best. Digitalis should be given (F. Anstie), and as soon as its effects show, the muriated tincture of iron (gtt. xx every six or seven hours) should be given. In many cases it will be impossible to wait for the effects of diuretics and hydragogue cathartics, as the patient will die unless immediate relief from the pressure of the fluid is afforded. Under such circumstances the aspirator may be used with advantage. Those remedies may be employed which are of service in the treatment of general anasarca.

HÆMOTHORAX.

Hæmothorax is the escape of blood into the pleural cavity; it is never a primary affection. The escape of any considerable quantity of blood into the pleural cavity may occur in connection with syphilis, cancer of the lung or pleura, from the bursting of an aneurism, the rupture of the pleura following an extensive pulmonary apoplexy and accompanied by escape of blood from the lung, and the rupture of a vessel. It may be due to traumatic causes, a vessel being injured, as in fracture of the ribs. Sometimes blood is mixed with pleuritic effusion, the product of pleuritic inflammation in those of a scorbutic or purpuric diathesis. Fluid blood in the pleural sac soon excites inflammation, whose products are usually serum with a variable admixture of pus.

The *symptoms* of hæmothorax are those of liquid accumulation in the pleural cavity, with the accompanying evidences of internal hemorrhage, pallor, syncope, etc. In hæmothorax, dyspnœa is sometimes greatest at the onset, diminishing gradually.

In those cases where there is no appreciable traumatic cause for the bleeding, all that can be done is to keep the patient at rest. Opiates are not contra-indicated. Stimulants may be necessary. In some instances relief may be obtained by the performance of paracentesis.

PULMONARY TUBERCULOSIS.

(*Phthisis.*)

Recent investigations have established a pathological unity in the morbid processes of pulmonary phthisis, based on the development of tubercle tissue.

While all forms of tubercular disease must be considered identical in their origin, and the primary lesion in each to be tubercle, the wide variations in the morbid changes which are found in the lungs of phthisical subjects, as well as the marked differences in their clinical history, compel us to recognize clinically two distinct varieties of pulmonary tuberculosis:

I. *Acute tuberculosis.*

II. *Chronic tuberculosis.*

The pathological changes which characterize pulmonary tuberculosis depend upon the following factors:

1. The amount of infective material received, and the rapidity with which it enters the lung.
2. The channels by which it reaches the pulmonary tissue.
3. The inherited or acquired predisposition of the affected person to the development of tubercular tissue.
4. The constitutional diathesis predisposing to either suppurative or fibroid changes in connection with inflammatory processes.

When large numbers of bacilli enter the lung through the blood-vessels, the resulting disease is usually part of an acute general miliary tuberculosis.

When moderate numbers are received more gradually, or through the air-passages, the resulting tubercular development is composed, in varying proportions, of gray miliary tubercle and foci of caseous (tubercular) pneumonia. The succeeding phthisical processes may be rapidly necrotic, (acute phthisis); gradually destructive, with suppuration and varying amounts of fibroid development, or largely fibroid in character (chronic phthisis). As the bacilli make their way through the air-passages to the alveoli, they may become fixed at any point on the epithelial surfaces or within the pulmonary tissues. The more frequent seats of their primary location are (1) the junction of the terminal bronchioles and the alveolar walls, (2) the bronchial, and (3) the alveolar surfaces. Many remain at these points, others make their way, or are carried by the leucocytes, to the substance of the tissues, becoming finally fixed in the perivascular lymph channels, or the interstitial spaces of the bronchial and alveolar walls.

The primary changes induced by their presence are hyperplasia of the fixed cells, and round cell infiltration. These result, within the lymph channels and interstitial spaces, in the formation of gray miliary tubercle, and, in the alveolar cavities, in the development of larger masses of tubercular tissue, forming the tubercular pneumonia, which, under later changes, becomes caseous pneumonia. In both cases the round cell infiltration affects the adjacent tissue to a greater or less degree, and is the first indication of subsequent inflammatory processes.

It is this secondary inflammation which largely determines the morbid anatomy in the several forms of phthisis, and which, in connection with the tubercular tissue, results in that pulmonary consolidation so characteristic of all phthisis.

The second effect of tubercle bacilli is manifested in the marked tendency shown by all tubercular tissue, as well as the products of the associated inflammation, to undergo "coagulation necrosis," granular and caseous degeneration, so that only the newly formed products show the translucent appearance so characteristic of *gray tubercle*, while all older tubercular tissue has become necrotic or converted into caseous foci. Degenerated miliary tubercle may not be distinguished from that which was primarily pneumonic or disseminated. The tubercle bacilli themselves are found disseminated throughout the tubercle tissue and less abundantly in the inflammatory products. When caseous foci become exposed to the air, their development is rapid.

The mechanical effects of the tubercular developments have an especially important bearing upon the nutritive changes in the lung. Direct compression of the parenchymatous elements is associated with obstruction of the lymph channels and vessels, and occlusion of the smaller bronchioles, all of which increase the necrotic tendency. The vascular obstruction is augmented by thrombosis, by endothelial proliferation, and by *endarteritis* and *endophlebitis obliterans.*

The inflammatory changes of phthisis are prominently necrotic, suppurative, and fibroid. They affect all the tissues of the lung. The fibroid changes, which are present in greater or less degree in all cases, cause thickening of the vascular walls, induration, and subsequent weakening of the bronchial tubes, and a development of fibrous tissue in and about tubercular foci and throughout the pulmonary tissue. Extreme degrees of fibroid change result in fibroid phthisis.

Necrosis and suppuration are associated processes. When necrosis is acute, it precedes marked suppuration; when more gradual, it follows a diffuse or localized purulent infiltration of the tubercular and inflammatory products.

Necrotic masses seldom remain long intact. They break down gradually or in sloughing masses, leaving, as they are thrown off in the sputa, the typical cavities. When fibroid changes have been slight, these cavities will have soft, sloughing, pus-infiltrated walls, and will continue to enlarge by ulceration. If fibroid developments have been more abundant, the walls will be firmer, and granulation may take the place of ulceration and the cavity be healed, or its walls converted into a firm, smooth, pyogenic tissue.

Cavities by dilatation may be formed at points in the bronchial walls where tubercular fibroid degeneration has occurred. These bronchiectatic cavities are more abundant in fibroid phthisis. Aside from the purely tubercular lesions, adjacent parts of the lung may present the evidences of simple bronchitis and catarrhal pneumonia. Secondary infection may occur in other parts of the lung, the bronchi, larynx, or intestinal canal, by direct contact of the tuberculous secretions; while ulceration of a caseous focus into a vein may be followed by a general infection, and an irruption of gray tubercle throughout the body.

A tubercular pleurisy is always present when phthisical processes are near the surface. It is plastic in form and results in adhesions. If a tubercular nodule ulcerates into the pleural cavity, a sero-purulent exudation will be rapidly developed.

Two stages can be recognized in the anatomical changes in the lungs:

1. The stage of consolidation, corresponding to the primary tubercular developments and the infiltration of the lung by inflammatory products.
2. The stage of softening and excavation; beginning with the lique-

faction and removal of the necrotic tissues, including formation of cavities.

A consideration of the pathology of the disease makes it patent that these two stages will always be found side by side in the same lung whenever cavities have formed.

ACUTE PULMONARY TUBERCULOSIS.

(Acute Phthisis.)

Morbid Anatomy.—The development of tubercle in acute pulmonary tuberculosis usually occurs at the apex of the lung, either in front or posteriorly.

When resulting from a rapid infection, as the rupture into a bronchus of a caseous gland, the consolidation assumes the form of a caseous broncho-pneumonia. In most cases, however, while the pneumonic consolidation is prominent, more or less miliary tubercle is formed. The inflammatory processes are acute; but little fibrous tissue is formed, and a *coagulation necrosis* may break down the affected parts even before caseous changes are prominent. The vascular as well as the less resistant tissues may be included in the necrotic change, and profuse hemorrhages follow removal of the affected mass. In less acute conditions, caseous changes are more prominent, and may include the areas of coagulation necrosis as well as other tubercular tissue. Owing to the slight amount of fibrous change, the cavities formed usually have soft sloughing walls. The attendant pleurisy is usually well marked and plastic in character.

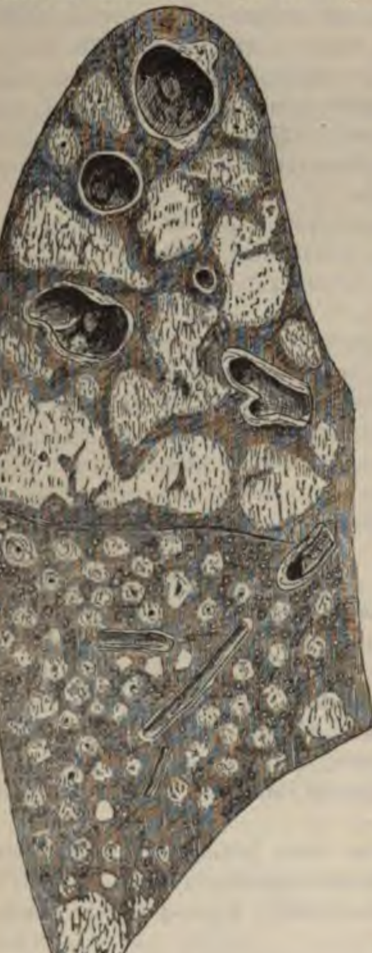

FIG. 42.
Lung in Acute Pulmonary Tuberculosis.
Showing areas of consolidation and cavities.

On removing the lungs of acute phthisis, the surface over the affected portion may appear normal, or mottled by yellow-gray areas, corresponding to points of solidification. More frequently it will be covered by a plastic pleuritic exudation. The consolidated portions may include an entire lobe of one lung, or be scattered in smaller masses throughout one or both lungs. On section, the consolidated portions are better marked. The cut surface has much the appearance of the red hepatization of lobar pneumonia. Scattered here and there, more or less abundantly, on the dark reddish-gray background, may be seen gray miliary tubercles; yellow, or yellowish-white masses of varying size, representing degenerated gray tubercle or caseous pneumonic foci; sharply outlined areas of coagulation necrosis, which are still dark reddish-black or a dirty yellow, from caseous change; and finally, here and there a dirty sloughing cavity with ragged, pus-infiltrated walls, and containing remnants of the dead tissue or soft clots. Under the microscope the alveoli are seen distended by fresh or degenerated tubercle, which may extend into the smaller tubes. Smaller bits of the same tissue stud the bronchial walls, and in the vascular lymph spaces are seen obstructing the lumen of the vessel. Round cell infiltration is present throughout the affected lung.

In the peripheral zones the primary changes of epithelial proliferation and cellular infiltration may be the only evidences of bacillary activity. Less frequently than in chronic phthisis, bronchial ulcers and sub-epithelial abscesses may mark the points of proliferation in lymph follicles, or softening and rupture of tubercle nodules, but cicatrices and fibrous-tissue formations are never prominent and seldom appreciable.

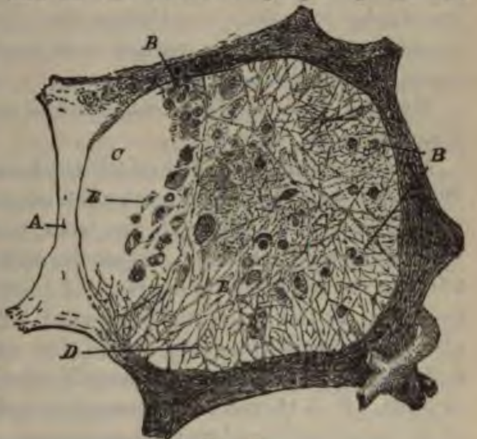

FIG. 43.
Acute Pulmonary Tuberculosis.
Section of lung showing a single alveolus in stage of hepatization.
A. Wall of alveolus, with infiltration of pus at B, B.
C. Cavity of alveolus nearly filled with changed epithelia and a few pus-corpuscles.
D, D. Fibrillar mesh enclosing the cell elements. × 300.

Etiology.—The direct exciting cause of acute as of all forms of tuberculosis is the bacillus tuberculosis. This bacillus is received directly or indirectly from some other tuberculous subject.

The channels of communication are numerous and varied. The most frequent are by particles of dried sputa floating in the air; by the use of dishes, napkins, handkerchiefs, and other personal belongings of phthisical subjects; by the use of milk of tuberculous women or cows, and possibly by tuberculous meat. Other unique methods of contagion are occasionally noticed.

Although the bacillus is accepted as the sole *exciting* cause of phthisis, its omnipresence, and the impossibility, under the present social conditions, of avoiding a more or less intimate contact with it, render the predisposing causes of its development, without which it is inactive, of paramount clinical importance. These causes are general and local. The most important *general causes* are an inherited or acquired feebleness of constitution, anti-hygienic influences, climate, and soil.

The *local causes* are found in inflammatory conditions of the pulmonary tissues.

Inherited Tendency.—At present proof is wanting that the bacillus is ever conveyed to the fœtus *in utero,* although some observations appear to show that such may exceptionally be the case. The supposition that the bacilli are present in the tissues at birth, and remain latent, to become active later in life, is based upon the frequent development, in children, of tuberculous lesions of the joints without previous disease of the lung. The fact of an inherited vice of constitution, and its influence in determining tubercular growth, are so decided that many observers have claimed that phthisis can never be developed by those who have no such tendency. Every-day experience disproves such sweeping statements. We have no knowledge of what constitutes the tubercular diathesis, although it is grossly manifested by certain well-marked signs to which we give the name "scrofulous."

My own statistics show that such a predisposition is inherited in more than eighteen per cent.

Mothers transmit phthisical tendencies more certainly than fathers. But when *one* parent alone is affected, the mother is more apt to transmit to the daughters than to the sons, and *vice versâ.* The stronger the hereditary predisposition, the earlier will be the development of the disease, and the more acute its course. A phthisical vice of constitution may be inherited by the children of the aged, of drunkards, of those enervated by excesses, and of those who at the time of the birth of their children were suffering from some form of constitutional disease, such as cancer, syphilis, or gout. It is therefore necessary, in order to fully determine the influence of an hereditary tendency in any given case, to know the condition of the parents at the time of the individual's birth. Children of consanguineous marriages are especially liable to pulmonary phthisis.

Anti-hygienic Surroundings.—Second only to hereditary influence are anti-hygienic surroundings. Impure air, improper quality and insufficient quantity of nutritious food, are among the most prolific of this class of causes. Bad ventilation and impure air, an indoor life, especially when large numbers are crowded into a small space, are strong predisposing causes. The frequency of phthisis in clerks, printers, tailors, milliners, seamstresses, factory employees—who live in a hot, close, dust-laden atmosphere—proves this. Of indoor workers those are most liable to phthisis who exercise least at their vocations. Compositors suffer oftener than the press-hands in the same room. Prison and cloister statistics show a mortality from phthisis of from forty to fifty per cent., while that among the people at large is only fifteen per cent. Careful examinations of the dust and air of prisons for bacilli have shown that this difference cannot be accounted for on the theory of contagion alone. The moister the air and the higher the temperature of the apartment, the more liable is phthisis to be developed. If, in addition to these anti-hygienic conditions, are added insufficient and improper clothing, want of cleanliness, alcohol drinking, prolonged lactation, and repeated miscarriages, it is evident that the feebleness of constitution which predisposes to phthisis can be acquired as well as inherited.

I am convinced, from a careful analysis of my records, that the phthisical developments depend as much upon the anti-hygienic influences under which childhood has been passed as upon hereditary tendencies. These predisposing anti-hygienic influences embrace the important problem of infantile diet. Few mothers, especially among the wealthier classes, are in a condition properly to nourish their own offspring. The habit which prevails of feeding children until they are one, two, or even three years of age upon barley-water and pap, has a great influence upon the future physical development of the child. In determining the influences which have predisposed to phthisis in any case, it is important to consider not only the condition of the parents at the time of the birth of the individual, but also the hygienic influences under which his childhood and early life were passed. One of the great objects of early physical training should be to overcome hereditary physical tendencies; this can be accomplished, in the majority of cases, by good hygienic surroundings and systematic physical training during infantile and early life. It is especially important that the children of phthisical parents should be placed under such influences, during infancy and childhood, as shall ensure the greatest physical vigor. All these predisposing influences tend to arrest physical development.

Climate has long been regarded as an important factor in the development of phthisis. We know of no climatic condition which renders its development a necessity, or that makes its development impossible; yet there is no question but that it occurs with greater frequency in one climate than in another. It is rare in the torrid and frigid zones, and frequent in the temperate. *Altitude* is more important than climate, for most high elevations are antagonistic to its development. The condition of the *soil* of a region or locality favors, or is antagonistic to, phthisis: light, sandy, porous soils are antagonistic; while heavy, hard, clayey, and impermeable soils are favorable. A damp, cold atmosphere, an impermeable soil, and sudden changes in temperature, are the most favorable conditions for developing phthisis. Want of sunlight acts also as a strong predisposing cause. It seems probable that the climatic and telluric conditions influence both the resisting power of the individual, and the vitality of such bacilli as are distributed through the air.

Local Causes.—One who carefully studies the clinical features of a large number of cases of phthisis must be convinced that bronchitis of the smaller tubes and chronic lobular (*catarrhal*) pneumonia are the starting-points of a large number of cases of phthisis. Some call these "exceptional" catarrhs.[1] That an apparently simple catarrh leads to the development of tuberculosis in one case and not in another may be explained by the fact that one individual is in a condition to resist the bronchitis, while in another all the predisposing causes of tuberculosis are in operation, and the catarrh then furnishes the favorable soil for the development of bacilli, while it decreases the resisting power of the tissues. The relation which pneumonia bears to the development of phthisis has been sufficiently considered under the head of its morbid anatomy. From a clinical standpoint there seems to be no question but that a non-resolved pneumonia is the starting-point of phthisis in quite a large proportion of cases. The question which it seems difficult to decide is, Are such pneumonias tubercular?

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[1] Williams found in 1,000 cases of phthisis that bronchitis was the origin in 12 per cent. Niemeyer regards bronchitis as the primary and essential developing cause in the majority of cases.

That pulmonary phthisis not infrequently dates from a *pleurisy* is evident to every careful observer. Phthisis which is preceded by pleurisy is often attended by an extensive development of fibrous tubercular tissue, and it may be assumed that the primary pleurisy in all such cases is also tubercular. Bronchial hemorrhage is frequently the first and only sign of phthisical developments. It is claimed that tuberculosis precedes and causes the hemorrhage. Unquestionably such bronchial hemorrhages indicate a vice of constitution which favors phthisical developments; but it requires no argument to prove that the hemorrhage is not of necessity an evidence that tubercles exist in the lung at the time of the hemorrhage. The connection which exists between phthisical developments and bronchial hemorrhage is not always clear.

The *mechanical irritation* of the bronchi produced by the constant inhalation of an atmosphere laden with dust leads to phthisis. The phthisis of knife-grinders, stone-cutters, potters, and coal-miners, are examples of this. It must be stated, however, that the primary fibroid changes in the lungs of such persons do not always become tubercular. The constant inhalation of noxious gases, such as are generated in overcrowded, badly ventilated apartments, is a frequent predisposing cause of phthisis.

Pregnancy, instead of preventing phthisis, as was at one time supposed, predisposes to it, and renders its course more rapid in those who are already phthisical.

Emphysema and *goître* have been by some supposed to afford an immunity against phthisis, but my observations lead me to the conclusion that it is a very frequent attendant of both these conditions. The notion that malaria and marsh fevers are antagonistic to phthisis is disproved by everyday experience. The relation between *diabetes mellitus* and pulmonary phthisis is not well understood, but that one complicates the other very frequently is a clinical fact.

The factors which determine whether a particular pulmonary tuberculosis shall result in recovery, or acute or chronic phthisis, are found in the manner of the infection and the diathesis of the patient. Sufficient quantities of infective material may be received by those of high resisting power to develop an acute phthisis, and small amounts in those of strong tubercular tendency may induce a similar condition. Patients in whom all inflammatory processes tend to necrosis and suppuration are more liable to acute phthisis, while those of a strong fibroid diathesis are not only less liable to tubercular disease, but, even when infected, quite certainly develop chronic forms of the disease.

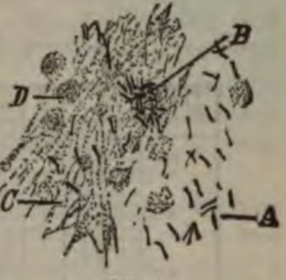

FIG. 44

Tubercle Bacilli from Phthisical Sputum. Stained with Fuchsin.

A. Isolated bacilli.
B. Groups of the same.
The unstained granular débris of pus-corpuscles and mucus are seen faintly at C, D. × 500.

Symptoms.—A young adult who for some time has had a dry, hacking cough, with a gradual but steady emaciation, is suddenly seized with a sharp pain in the side; the pulse becomes rapid and feeble, and the temperature rises to 104° in the evening, while the morning temperature may be normal. With increase in pulse-rate and temperature the skin becomes pungently hot. The fever alternates with night chills and profuse sweats. The cough is soon accompanied by an opaque, purulent expectoration, in which are found numerous tubercle bacilli and yellow elastic fibres. There is rapid loss of flesh and strength; the patient becomes extremely anæmic; and the constant harassing cough causes loss of sleep and extreme exhaustion. The expectoration is usually not abundant until after the breaking down of the lung-tissue has occurred. Patients ascribe the emaciation and weakness to the profuse sweats. The respirations and the pulse-rate increase in frequency with the fever. The pulse ranges from 120 to 135. Cardiac palpitation and sudden acceleration of the pulse-rate follow excitement. In some cases the chill, fever, and sweat occur with such regularity that malarial fever is suspected, or a malarial element is regarded as the prominent feature. Nausea, vomiting, and diarrhœa are often prominent symptoms, and greatly add to the exhaustion which is so marked a feature of the disease. The skin assumes a pearly pallor, the hectic flush is present, and the eyes are bright and glistening. Hæmoptysis may mark the advent of the disease and recur at intervals during its course. It is rarely absent during the entire course of the disease. Anorexia is always a marked symptom. Not infrequently the destructive processes are so rapid as to cause pneumothorax. Acute phthisis usually pursues a steadily progressive course, but it may assume an intermittent character, and have periods of arrest and apparent amendment followed by periods of exacerbation and rapid progress.

Physical Signs.—The physical signs will vary with the seat and extent of pulmonary consolidation, and with the rapidity with which destructive processes are established.

Inspection, during its early stage, shows rapid respiration and imperfect expansion of the upper part of the chest during a deep inspiration; as the disease advances, the loss of expansion becomes more and more apparent, but there is no infra-clavicular retraction.

On *palpation* the loss of motion in the infra-clavicular spaces is more apparent; and if the pleuritic changes are not extensive, there will be increased vocal fremitus.

On *percussion* there will be more or less dullness over the infra-clavicular spaces. If there are large superficial cavities which contain little fluid, there will be amphoric or cracked-pot resonance.

Upon *auscultation*, in the early stage, expiration is notably prolonged and high-pitched, and fine mucous râles with fine crepitation will be heard over the affected district. The respiration is wavy and interrupted.

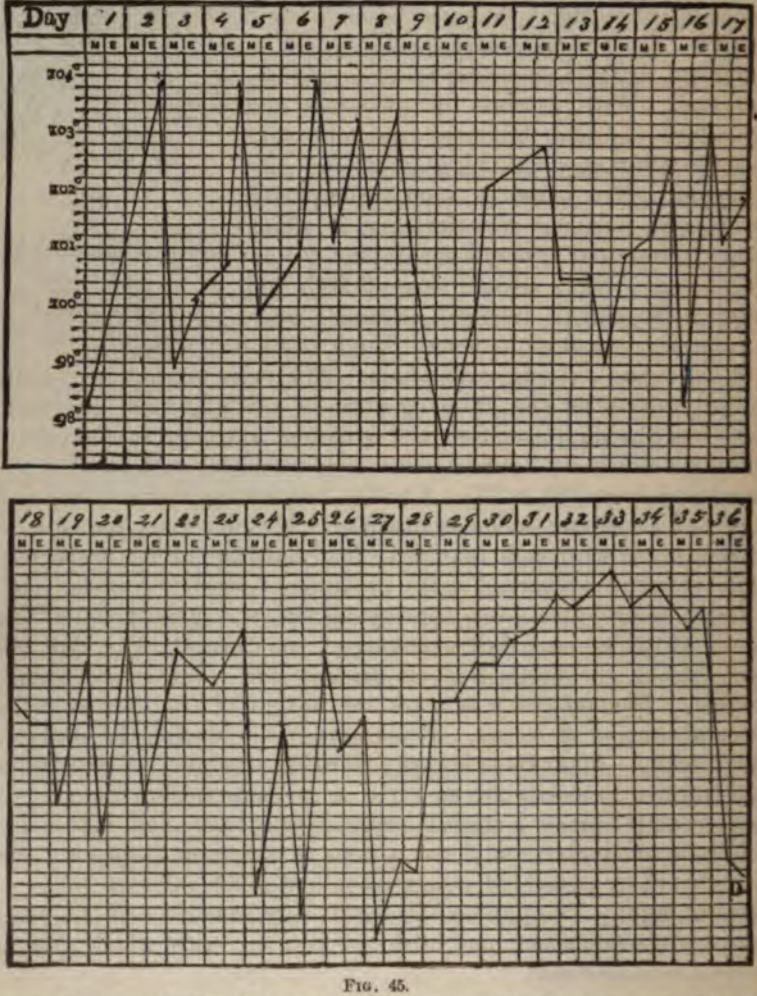

Fig. 45.

Temperature in a case of Acute Phthisis. Patient æt. 25. The larger falls of temperature, on the 5th, 9th, 16th and 24th days followed the administration of sulphuric ether. Falls of a degree and a degree and a half were induced by large doses of the sulphate of quinine.
Death occurred after a steady rise in temperature for a week preceding.

There may be distinct bronchial breathing and bronchophony over a circumscribed space posteriorly in the scapular region. Excavations take place rapidly in the consolidated portions of the lung; they are of varying size and are situated at varying distances from the surface of the lung. Deeply-seated cavities, when filled, give deep-seated dulness, and, when empty, an exaggerated percussion resonance. Over small cavities with lax walls low-pitched, puffing, cavernous respiration will be heard. This is very frequently heard in *acute phthisis where soft yielding walls result from rapid pulmonary necrosis.* Amphoric breathing, gurgles, and metallic tinklings will be heard over large cavities which communicate freely with bronchial tubes. The sub-clavian murmur (discussed in chronic phthisis) is not so liable to be heard in acute as in chronic phthisis.

Differential Diagnosis.—Acute phthisis may be mistaken for *croupous pneumonia*, *bronchiectasis*, and *acute general capillary bronchitis.*

In *pneumonia* the prolonged ushering-in chill, the continuous high temperature, the characteristic sputum, the dulness limited to a lobe, and the pneumonic countenance, are symptoms which readily distinguish it from acute phthisis. In some cases the differential diagnosis cannot be made during the first week. *Bronchiectasis* accompanied by wasting, fetid expectoration, hæmoptysis, and night sweats, with the physical signs of consolidation, may well be mistaken for the advanced stage of acute phthisis. In phthisis the signs of consolidation *precede* those of cavities;—in bronchiectasis they *follow* them. Fever and emaciation are always greater in phthisis than in bronchiectasis, and the symptoms are more steadily progressive. In *capillary bronchitis* there is no dulness on percussion, subcrepitant râles are heard on both sides of the chest, and there is no bronchial character to the respirations. The temperature range is lower than in phthisis. Emaciation is rapid in phthisis, and the signs of the formation of cavities occur early.

Prognosis.—The prognosis in acute phthisis is always unfavorable. Its average duration is from five weeks to five months. A sudden amelioration of the symptoms may occur before the cavities are formed, but the amelioration is one of short duration, and is usually followed by a more rapid progress of the disease. It may be complicated by pleurisy, pneumothorax, hydrothorax, peritonitis, and, rarely, by pericarditis. Death may occur from exhaustion, asthenia, or complications. Acute capillary bronchitis and pulmonary œdema and congestion often lead to a rapidly fatal termination.

Treatment.—Most cases are hopeless; the *dietetic* and *climatic* methods employed in chronic phthisis have no place in the management of acute phthisis.[1] Morphia in small doses—one-twentieth of a grain hypodermically every six or eight hours—has, in my hands, been more satisfactory in staying the progress of the disease, prolonging life, and keeping the patient comfortable, than any other plan.

CHRONIC PULMONARY TUBERCULOSIS.

(*Chronic Phthisis.*)

Chronic pulmonary tuberculosis presents, both clinically and anatomically, three well-marked varieties, dependent upon the form of the tubercular development and the character of the secondary inflammation.

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[1] Dr. McCall Anderson (in London *Lancet*, June, 1877) takes a more hopeful view of these cases, and claims that subcutaneous injections of atropia check the exhausting sweats; and that quinine, digitalis, and opium reduce the temperature, and if they fail, ice-cloths to the abdomen will accomplish the desired result. His reported results are exceedingly encouraging, but the failure of his treatment as tried by others causes many to doubt his diagnosis.

They may be designated as :

1. Pneumonic tuberculosis, caseous pneumonia, or catarrhal phthisis.
2. Disseminated tuberculosis.
3. Fibrous tuberculosis, or fibroid phthisis.

Aside from these distinctive forms, many cases will be seen in which the several tubercular developments are variously commingled.

Morbid Anatomy.—*Pneumonic tuberculosis.*

The primary tubercular developments occur in the bronchi and alveolar cavities, and not only may be, but often are, preceded by localized bronchitis or catarrhal pneumonia.

The tubercular tissue fills the air-cells in one of two ways : (1) by polypoid outgrowths from the alveolar walls, consisting of round and polygonal cells in a basement substance; (2) by masses of similar cells—with or without giant cells—not in connection with the alveolar walls, which partially fill the vesicles of the affected lobules, the intervening space being filled with inflammatory products. These masses vary in size. They may be limited to a single lobule, or may attain the size of a walnut.

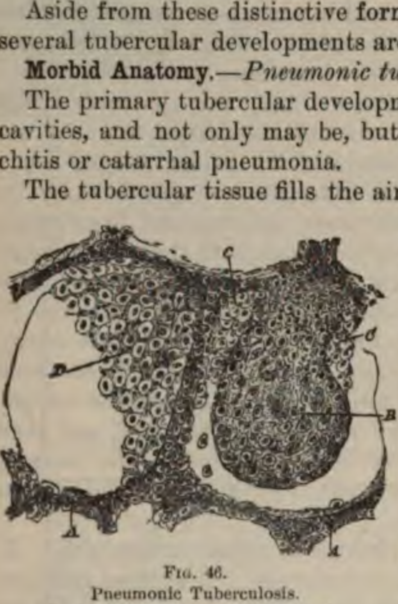

FIG. 46.
Pneumonic Tuberculosis.
Section of lung showing two alveoli.

A, A. *Wall of alveoli covered with changed epithelium.*
B. *"Polypoid outgrowth" from the alveolar wall, nearly filling the air-vesicle—composed of a delicate, granular basement substance in which are imbedded round and polygonal cells.*
C, C. *Epithelial cells between the last and wall of the air-vesicle.*
D. *Alveolus partly filled with epithelial and lymphoid cells in the basement substance.* × 280.

After a time masses of cells obstruct the bronchioles. The nutrition of the bronchial walls at the seat of the obstruction is interfered with, and they become attenuated, or a peribronchitis is developed. This peribronchitis is primarily tubercular, and secondarily necrotic and suppurative or fibrous.

Early in the process lymphoid cells infiltrate the alveolar septa, the bronchial walls, and the alveolar contents. Pressure on the vessels in connection with endothelial proliferation in their lumen, and thrombosis, as well as the direct action of the bacilli, induce caseation of the tubercular and inflammatory products. As caseation advances, all the elements become granular and are agglutinated by a slightly transparent substance which glistens like fibrin upon the addition of acetic acid. These cheesy masses are found in patches; they are friable, and present a gray homogeneous appearance, forming the so-called yellow tubercle, and contain bacilli. Before distinctly necrotic processes supervene, ulcerations may occur in the bronchioles. Ulcers thus formed in the tubes are usually sharply defined and shallow: sometimes they involve adjacent lung tissue as well as the bronchial tubes.

The gross appearance of lung-tissue involved in this form of phthisis, before necrotic changes supervene, varies. It may be of a gray color, hard and glistening, described by Laënnec as "*gray infiltration*,"[1] or it may appear as a colloid jelly-like mass ("*gelatiniform infiltration*" of Laënnec, or the "*colloid caseous pneumonia*" of Thaon).

When a few lobules only are involved, they may become encapsulated, or may undergo resolution. It is rare, however, for a lung to return to its normal condition unless the nodules are small and few in number. Even should the masses be removed, obliteration of the alveoli which they occupied is apt to occur. If a cheesy nodule is encapsulated, cretaceous or chalky material is found in the centre of the fibroid tissue. The lung-tissue between these nodules may be anæmic, hyperæmic, œdematous, or emphysematous, or the seat of atelectasis. The larger the nodule and the more rapidly it has formed, the more liable is it to soften. Cheesy masses may soften, and by a process of ulceration be removed through the bronchi. Absorption of caseous matter by the lymphatics is attended by more or less adenoid hyperplasia, and a group of miliary granules may be developed about a caseous centre, the remainder of the lung not being involved. Sometimes softening and ulceration are so rapid that the process becomes distinctly gangrenous.

Cavities.—The walls of a phthisical cavity are always irregular. At first they are soft and friable; later they become tough, smooth, and fibrous. Bands of dense connective-tissue traverse them, sometimes covered by a layer of granulation-tissue, and vessels and large bronchi often extend across them. Sharply "cut-off" stubs of bronchi often project half an inch from their walls; portions of the wall may stand out like the columnæ carneæ of the heart; or the surface may be uneven or ragged. The connective-tissue trabeculæ extending across a cavity frequently contain blood-vessels, whose rupture may cause fatal hæmoptysis. When cavities are formed, the lung-tissue around the cavity will be indurated, and the cavities will be separated from one another by bands of firm

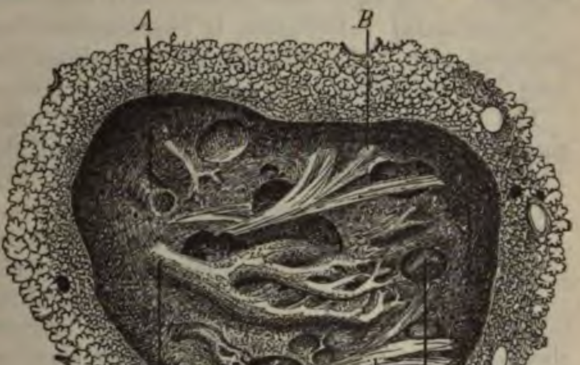

FIG. 47.
A Lung Cavity.
A. Stump of small bronchus.—B, B. Bands of fibrin.—C. Loop of blood-vessels in bed of cavity.—D. Smaller cavities opening into a larger one.

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[1] The infiltrated tubercle of Laënnec is considered as *desquamative pneumonia* by Buhl, and *scrofulous inflammation* by Rindfleisch.

fibrous tissue, and the peri-bronchial and peri-vascular connective-tissue sheaths and the thickened pleura will all be involved in an indurative process. Cavities increase in size by peripheral disintegration, or several small ones may coalesce and form one large, irregular excavation. Phthisical cavities contain air and a grumous purulent fluid of a yellowish or greenish color, with which shreds of lung-tissue may be mingled. If the growth of a phthisical cavity becomes arrested, a "limiting membrane forms on its inner surface." The purulent secretion from it at first is abundant, later it diminishes, and the case becomes one of a "quiescent cavity." A large cavity may, by contraction of the fibrous tissue around it, have its walls approximated, but not united. True cicatrization of a chronic cavity which has a distinct lining membrane rarely if ever occurs.

Ulcerating cavities are those which, having been long quiescent, take on, for some reason, an ulcerative process. A small cavity at the surface of the lung, after having caused a localized pleurisy and a thinning of the friable wall of consolidated lung-tissue which separates the pleura from the cavity, may break through into the pleural cavity and cause pyopneumothorax. Pleurisy is rarely absent in this variety of chronic phthisis; firm adhesions form, and the pleura may be from three-quarters of an inch to one inch thick. These changes—pulmonary and pleuritic—are best marked at the apices.

The *bronchial glands* may be softened, cheesy, chalky, pigmented, and enlarged. The right heart is frequently hypertrophied or dilated.

Disseminated tuberculosis is characterized by gray tubercle granules scattered more or less abundantly throughout the affected portion of the lung, or by masses of them agglomerated by fibrous tubercle-tissue.

The lungs are large, emphysematous, and pale, unless pneumonia, congestion, or œdema is present. The surface of the lungs is often marbled. The apex of the lung is studded with firm, hard, gray, or cheesy nodules, varying in size from a pin's head to that of a pea.

Upon *section* of a lung which shows these changes, muco-pus flows from the cut bronchi. Peribronchitic and inter-alveolar interstitial pneumonia is developed to a greater or less degree around these nodules, with irregular dilatation of the alveoli. These nodules originate chiefly in the lymph-sheaths of the arterioles, in the peribronchial adenoid tissue, or in the small masses of cytogenic tissue in the alveolar walls.[1] These bloodless nodules are incapable of suppuration, resorption, or organization. As the tubercle-tissue about the vessels increases, it causes occlusion of their lumen. The lumen of the occluded vessel is occupied by granular fibrin, and on transverse section a row of white blood-corpuscles and of endothelial cells is often seen between the coagulum and the vessel wall. In recent cases the walls of the vessels are very easily distinguished. But if the centre of the tubercle has become caseous, the vessel wall is also altered and is very indistinct. Thickening of the alveolar walls may also result from the development of tubercle-tissue in them. Many claim that the commencement of the process is a small cellular projection on one side of an alveolus, which, as it grows, pushes the capillaries and the epithelium with it into the alveolar cavity. The bronchioles are thickened and diminished in calibre; sometimes there are complete peribronchial cylinders of new embryonic or tubercle-tissue. The alveolar cavity may be filled by the new formation, the walls remaining intact; or the wall may be destroyed by the growth of the projecting nodule, and thus communication opened between adjoining air-vesicles.[1]

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[1] Rindfleisch states that *the points at which the smallest bronchioles become continuous with alveolar sacs are the situations of the first eruption of the tubercles*, and that the first lesion is a tuberculous infiltration of all the angles and projections situated at these points.

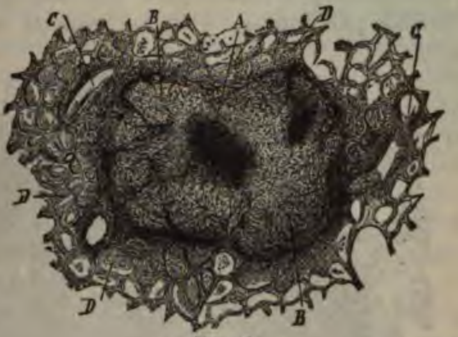

FIG. 48.

Section of lung showing a small miliary tubercle, with surrounding pulmonary alveoli.

A. *Cheesy centre of tubercle.*
B B. *Trabeculæ of the basement tissue of the tubercle, containing lymphoid elements, large cells, etc.*
C. C. *Divided arteries with infiltration of their wall with tubercular tissue.*
D D.D. *Lung alveoli filled with catarrhal products.* × 50.

After coalescence of masses of tubercle-tissue, anæmic necrosis occurs; the cellular elements become granular, atrophied, and fatty, and form cheesy yellow masses. These caseous masses are easily removed from the parenchyma in which they are imbedded. Surrounding these masses are found groups of gray miliary tubercles. The mode of the formation of cavities and their appearance are the same as already described. Between the ulcerous cavities the lung-tissue may be emphysematous, engorged, or pneumonic. The *bronchi* are filled with muco-pus; their mucous membrane is the seat of a catarrh, and is congested and trabeculated. Their walls are thickened, tubercle-tissue is found in them, and ulcers are not infrequent.

The *pleura* is congested, and covered with fleshy, soft vegetations over the parts involved in the chronic phthisical processes. Organized adhesions are common; in them tubercles may be found, and also in the pleura. Tubercles may be found in the larynx, trachea, mesenteric and lymphatic glands, and in the mucous surfaces of the alimentary tract, the peritoneum, spleen, kidney, liver, brain, bladder, and testes. Fatty heart is not uncommon. Thus a chronic local disease of slow and seemingly intermittent progress is found associated with evidence of general infection. The recent dictum is that chronic tubercular growths may heal. Many, however, believe that, once established, the process can never become quiescent.[2] Ulceration, dilatation of the bronchi, and the formation of large cavities are not infrequent attendants on this process.[3]

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[1] Hamilton states that this appearance, which so resembles the condition of the lobules in catarrhal pneumonia, has caused the mistake of regarding tubercle and *catarrhal pneumonia* as identical.

[2] Rindfleisch advances a new doctrine of the "healing processes in tuberculosis of the lung," viz.: it consists in a shrinkage of the infiltration, combined with a formation of new vessels. These vessels do not penetrate deeply into the infiltration, but surround it and supply it with constant, though scanty, nourishment. It is a "fibrinous shrinkage:" the capsule not only *encloses*, but nourishes.

[3] In a variety called by O. Clark and Rindfleisch "*Broncho-phthisis Pulmonalis*" the central feature of the change is *an extensive ulceration of all the bronchi of medium size down to the intra-lobular*

Fibrous tuberculosis, or *interstitial tubercular pneumonia*, is characterized by the diffuse development, in the lungs, of dense fibrous tissue associated with tubercles. The fibrous changes may be primary, an interstitial pneumonia, with subsequent tubercular infiltration, or the inflammation secondary to tubercular development may assume the fibrous character, and involve not only the tubercle, but the pulmonary tissue as well.

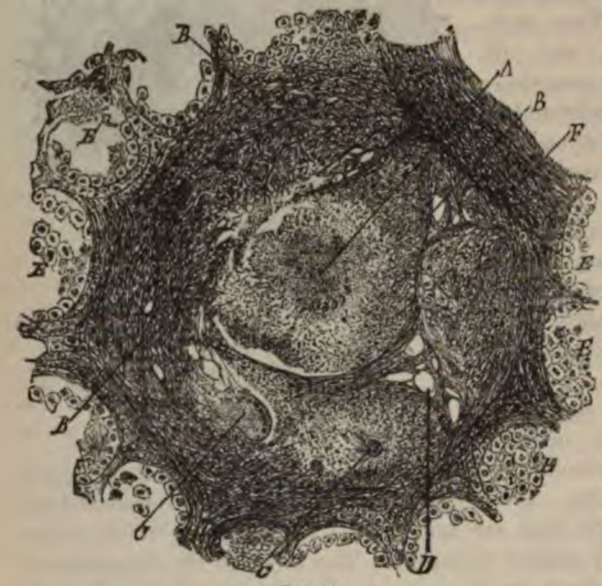

FIG. 49.
Section of lung showing a small tubercular nodule with surrounding alveoli.
A. Cheesy centre of nodule.
B, B. Fibrillar basement substance containing cells and nuclei.
C, C. Giant cells.
D. Vacuoles.
E, E, E. Alveoli, surrounding the tubercle, containing pneumonic products.
F. Part of wall of an artery infiltrated with tubercle-tissue. × 250.

The affected lung is diminished in size. The pleural surfaces are adherent and greatly thickened. A section of a portion of lung that is the seat of this variety of phthisis presents a smooth or granular surface, or it has a striated appearance. If granular, the granules are imbedded in the fibrous mass. The fibrous tissue contains more or less pigment material, which gives to the cut surface a bluish or gray color. If the process is old, the lung will be tough; if recent, it is less resistant and less leathery. When the disease has existed for a long time, the apex, and sometimes the whole lung, is converted into fibrous tissue, all traces of the normal lung-tissue being obliterated. The indurated tissue may be studded with nodules. The nodules may be small masses of dense fibrous tissue containing a few cells at their centre, masses of tubercular tissue, granulation-tissue enveloping tubercles, or cheesy masses with tubercles and interlacing fibrous bands. Giant cells may or may not be present in the nodules. The connective-tissue growths may begin in the walls of the bronchi, alveoli, or blood-vessels, in the septa, or in the pleura. The alveoli are at times dilated, at times narrowed; they are always deformed. The alveolar epithelium undergoes slight multiplication and swelling. New cells also form in the walls. Cheesy masses, specks of cretaceous material, and cavities of varying size, are found in the hard lung.

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branches. Spaces of all sizes are seen—on section—connected with one another. Rindfleisch states that a "desquamative pneumonic process" accompanies this peculiar form of tuberculosis of the lungs.

The bronchi are at times thickened, at times thinned. Bronchiectatic cavities (cylindrical, fusiform, or sacculated) are found, chiefly in the apex. The appearance of these cavities is similar to that described in chronic interstitial pneumonia. Through ulcerative processes, cavities, often of large size, result from these bronchiectases. As the disease progresses, more and more of the tubercle-tissue is changed into connective-tissue. But while the growth of connective-tissue is extra-alveolar, the tubercle-tissue is both extra- and intra-alveolar. The early stage of connective-tissue development consists in the accumulation of a large number of small cells looking like granulation-tissue and lymphoid cells; while, in the later stages, we find dense fibrillated tissue containing a few cells—and those spindle-shaped—and an abundant supply of irregular blood-vessels. Tubercles may also be found in the pleura.

Etiology.—The same factors are concerned in the development of chronic as of acute tuberculosis. Local inflammations, however, play a more prominent part in rendering the pulmonary parenchyma less resistant to bacillary invasion.

Some authorities state that bronchitis, catarrhal pneumonia, or pleurisy do not predispose to phthisis. My own experience is to the contrary. The only possible inference to be drawn from the fact that, of two persons living under similar conditions, one suffers from phthisis and the other does not, is that the failure of the bacilli to develop in the one case, and their success in the other, depends upon the vital power of the tissues to resist their invasion and to prevent their growth.

It is well determined that inflammatory processes decrease the power with which the tissues resist the invasion of other bacteria; and not only analogy, but all the well-established principles of pathology as well as clinical facts, prove beyond question that inflammatory, and particularly suppurative processes, favor the development of tubercle bacilli.

Pulmonary irritants which lead to fibrous formations are not only less liable to be attended by tuberculosis, but tend to prolong its course, and even to result in arrest or cure of the disease. Anthracotic changes show but little tendency to become tubercular, and when infection does occur fibrous phthisis results.

Symptoms.—There are certain symptoms which characterize the early stages of each variety of tuberculosis.

Pneumonic tuberculosis usually commences as a bronchial catarrh. The cough is paroxysmal and accompanied by tenacious muco-purulent sputa, now and then blood-stained. There is a gradual but steady loss of flesh and strength; the patient grows pale and has an occasional night-sweat. These symptoms are accompanied by the physical signs of slight consolidation at the apex of one or both lungs, with those of localized bronchitis of the small tubes. A localized pleurisy is very certainly present in these cases. Sometimes this variety begins with more acute symptoms, and the physical signs of apical lobular pneumonia are present. In such cases the pneumonia does not resolve, and the fever takes on a distinctly remittent type, with a more rapid loss of flesh and strength, and a copious purulent expectoration, often blood-streaked. Night-sweats become profuse and exhausting, and there are the physical signs of progressive consolidation of lung-tissue. At any time during the early stage the physical processes may be arrested, and during the period of arrest there may be a great improvement in the general condition of the patient, and complete recovery is possible. But in a large proportion of cases a return to the anti-hygienic conditions in which its primary development occurred, or a fresh bronchitis, lights up anew the phthisical process.

Disseminated tuberculosis may for a long period give no distinctive signs; for interstitial pleurisy, chronic bronchitis, and emphysema nearly always accompany it, their prominent symptoms masking those of tuberculosis. Patients with this form become emaciated; their dyspnœa resembles that of emphysema. The expectoration is in the earlier stages mucous, and later it becomes muco-purulent. Hæmoptysis is common; and hectic fever is more pronounced than in any other variety. There are no periods of improvement, though there may be periods during which the disease remains stationary. Pleurisy, laryngitis, and intestinal catarrh are more marked than the pulmonary symptoms. As the disease advances, its symptoms resemble those of fibroid phthisis.

Fibrous tuberculosis—or *fibroid phthisis*—comes on very insidiously; it may be ushered in by one or more attacks of hæmoptysis. In most cases it commences with the physical signs of a localized bronchitis and pleurisy at the apex of one lung. Cough, and a muco-purulent expectoration with more or less pain in the affected lung, may exist for a long time before there is any marked impairment of the general health. After a variable period the patient begins to lose flesh and strength, the cough increases, and the expectoration becomes more abundant. There is a progressive loss of appetite, but at no time is the temperature high or the pulse rapid. Dyspnœa becomes more and more marked, especially on exertion. Retraction of the chest walls under the clavicle commences quite early and is steadily progressive. The limited play of the chest walls is the most distinctive early sign. This variety of phthisis rarely occurs in young persons, and it is often associated with a rheumatic, gouty, or syphilitic taint, or is the result of mechanical irritation.

Of the symptoms which are common to all varieties of tuberculosis, the significance which is to be attached to the presence or absence of bacilli in the sputa is of paramount importance.

Without entering upon a discussion of this question it may be stated that single examinations of the sputa cannot determine either the presence or absence of tuberculosis. The presence of small numbers of bacilli may be possible after prolonged exposure to infection, when tubercle has not formed. Persistent absence or presence of bacilli, or large numbers in the sputa, however, are almost positive evidence of the character of any pulmonary disease. The number of bacilli in any given specimen has little or no diagnostic significance, since the rupture of a caseous focus may have filled the sputa temporarily when there is little actual disease in the lung. Few of those who rely least upon the presence of bacilli for prognosis would care to find their own sputa persistently loaded with them. On the other hand, bacilli may be absent for a considerable period from the sputa of those who are unquestionably suffering from extensive pulmonary tuberculosis. They may be present at one time, and absent at another in any given case. We are often able to recognize phthisical consolidation before bacilli appear in the sputa.

In analyzing the symptoms which are common in all varieties of chronic phthisis I shall first consider the *cough*. It is the earliest and most constant of all the phthisical symptoms. It is present early and continues throughout the whole course of the disease. At first it is dry and hacking. It may exist before there are any physical signs, and then there is little or no expectoration; it may amount only to a "clearing of the throat." The severity of the cough without expectoration is a measure of the extent to which the pleura is involved. The younger and more excitable the patient, the more paroxysmal is the cough. It is usually worse in the morning on rising, or just after lying down at night. Lying on the affected side often brings on violent paroxysms. Some cough after the slightest exertion; others have a varying number of paroxysms during the day, and can estimate how long an interval of rest they will have between the paroxysms. The loss of sleep occasioned by the cough may add much to the discomfort and wasting of the patient. In advanced phthisis, when cavities have formed, the cough becomes "hollow" in character. Expectoration may accompany cough from its commencement. At first it is tenacious, glairy, frothy, and mucous; then yellow purulent spots are found in it. It is always important to ascertain whether pallor, fever, and emaciation have been preceded by cough and expectoration, or whether emaciation was the primary symptom. The sputa are gelatinous and faintly pink when the infiltration is extensive. Vitreous, gelatinous, rounded masses may be mingled with yellow catarrhal expectoration, and these are evidences of a recent pneumonia. Dots and streaks of blood in catarrhal sputa indicate a lobular pneumonia; and when this occurs, fatty, swollen, and granular bronchial and alveolar epithelium will be found intermingled in the mass. The sputa in the earlier stages—often for months—are muco-purulent. When shreds of elastic tissue are found, it indicates softening and destruction of lung-tissue. Elastic fibres are generally found in compact, airless, uneven masses, which readily sink in water. As cavities form, the sputa become more purulent, sometimes being wholly composed of fluid pus, which may be fetid and greenish, and contain elastic fibres coming from the alveolar wall, organic matter, fat-crystals, pigment, young cells, and small masses of cheesy matter, and the tubercle bacilli; the latter are present in the sputa of all varieties of advanced phthisis. The quantity of matter expectorated varies with the extent of the bronchial catarrh and the number and size of the cavities. It may be expectorated readily, or only with difficulty. Usually, the more feeble the patient the more difficult the expectoration. In rapidly formed cavities the expectoration may contain fragments of bronchioles and blood-vessels, with shreds of lung-tissue.

Hæmoptysis is a very important symptom of phthisis, and may occur during any stage of the disease; the blood may simply streak the sputa, or a pound or more may be expectorated at one time. Hemorrhages that occur in the early stage of pulmonary phthisis are, in the majority of instances, bronchial, and the blood expectorated is arterial in color. When streaks of blood appear in the sputa, the bleeding usually comes from the vessels of the alveolar walls. Profuse hemorrhages in the later stages of phthisis have their origin in cavities in the lung substance. Hemorrhages that occur in the early stages may be profuse, but they are rarely dangerous; hemorrhages in advanced phthisis may be the immediate cause of death. Hæmoptysis usually comes on with coughing. There is a sensation as if a fluid were trickling underneath the sternum, and there may be violent cardiac palpitation, oppressed breathing, and a peculiar sweetish taste in the mouth. In profuse hemorrhage the rapid flow of blood into the mouth may excite vomiting and be mistaken for hæmatemesis. For some time after the primary hemorrhage blood is coughed up, and the color of the spitting becomes darker and darker. Sometimes without warning there is sudden filling of the mouth with hot arterial blood.

Many English writers describe a hemorrhagic phthisis. In this variety an apparently healthy man has a sudden and profuse hemorrhage, recurring daily for some time, and followed by cough and slight expectoration for a few days, with no physical signs of consolidation. These cases often continue for years without any other phthisical symptoms, but sooner or later phthisis is developed.[1] Hæmoptysis often occurs in those who have no physical or rational signs of phthisis at the time of its occurrence, and who do not become phthisical afterward. Although hæmoptysis occurs more frequently in phthisis than in any other pulmonary affection, and there are few phthisical subjects who do not have one or more hemorrhages, yet its occurrence is by no means a certain indication that an individual afterward will develop phthisis.

Fever.—Rise in temperature is so constant a symptom of phthisis that it has led to the expression, "there is no consumption without fever;" but in no two cases is the fever course exactly the same. In some cases the temperature in the morning may be subnormal, only reaching normal in the evening; in others the rise commences at 2 P.M., continues until 8 P.M., and then falls until 5 in the morning. Between 10 and 11 A.M. the temperature is nearly normal. As cavities form, the post-meridian rise occurs later; *i. e.*, 10 to 12 at night. Toward the end of the disease the fever type resembles that of pyæmia. Night-sweats temporarily lower the temperature. When the alveoli are involved in tubercular pneumonic processes, the temperature rises rapidly to 103°–104° F. Hectic fever may occur in any stage of phthisis, but is usually confined to the stage of softening and excavation. It has three stages: *first*, at some time during the day there is a well-marked chill or chilly sensation, which may last from half an hour to an hour, followed (*second*) by a dryness and heat of the surface, the temperature rising from 102° F. to 104° F., the face assuming a peculiar brilliant appearance, and the cheeks having a peculiar rosy tint called the "hectic flush." After a time the fever gradually subsides, and some time in the night (it may be toward morning), the ***third*** or sweating stage comes on. The night-sweats are usually profuse and exhausting, and always indicate the existence of hectic fever. The chilly feeling may be absent, the subsequent fever may be so slight as to be overlooked, but sweats are constant. A steady and continuous low temperature indicates that the phthisical processes are retrogressive; a steady and continuous high temperature indicates that they are progressive. In fibroid phthisis the temperature rarely rises more than a degree or two above the normal. In the absence of local symptoms, the thermometer alone may detect pulmonary phthisis in the aged.[1] An intermittent temperature indicates a milder process than a remittent or continuous febrile action.

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[1] Tubercular disease of the vascular walls is the primary and chief event in such cases.

The *pulse* in chronic phthisis bears no uniform relation to the temperature; it is always feeble. It varies greatly in frequency and force, but rarely in rhythm; it is accelerated by slight exciting causes. In the early stages its excitability is one of its most characteristic features. The arterial tension is below the normal. In the early stage of *fibroid phthisis* it is rarely over 100. In a few cases it is abnormally slow. An improvement in the other symptoms is not always accompanied by an improvement in the pulse. In the last stage of all varieties of phthisis the pulse becomes very rapid and feeble.

The *respirations* are more or less accelerated, and after exertion there is dyspnœa. When the patients are quiet, unexcited, and resting in bed, the respirations may be normal or but slightly increased. But on exertion the breathing becomes accelerated and labored. The accelerated breathing is due to the fever, the diminished breathing area, to bronchial obstruction, and to pain in the chest. Anæmia and heart failure may also contribute to it. In the absence of fever the dyspnœa and accelerated breathing diminish. The extent to which the lungs are involved influences the frequency of the respirations. In young subjects the dyspnœa is frequently periodical. During the whole course of fibroid phthisis, shortness of breath on exertion is a constant symptom.

Pain in the chest is not a prominent or constant symptom of chronic phthisis, except in connection with pleuritic changes. Dry and interstitial pleurisies are common; yet they seldom cause severe pain, but rather a sense of tightness and constriction on taking a full inspiration. Intercostal neuralgia is frequent and may be confounded with the pain of a localized pleurisy. Dragging pains in the side are most marked in fibroid phthisis. Pain on swallowing should always cause one to carefully examine the larynx. It usually announces the co-existence of laryngeal phthisis.

Emaciation is an early and constant symptom of phthisis; but it is not always progressive. Fever is the chief cause of the wasting and pallor that are so common in all varieties of phthisis. The higher the average range of temperature, the more rapid the emaciation. The pulmonary change may be *preceded* by progressive emaciation, but in all such cases the average temperature is a degree above the normal. Emaciation may be a part of the constitutional tendency of the individual, but such emaciation forms no part of the phthisical wasting. While emaciation, loss of strength, and progressive anæmia are recognized premonitory symptoms, they cannot be regarded as diagnostic. Emaciation may not be continuous in all cases; there are periods when the patient may even regain lost weight and muscular strength. The anorexia, dyspepsia, diarrhœa, profuse expectoration, and hæmoptysis are all causes of the emaciation. Phthisical wasting occurs not only in the fat and muscle, but in the organs and blood as well.[1] Slow, gradual wasting belongs to the history of *fibroid phthisis*.

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[1] Sir William Jenner makes three clinical types of chronic phthisis in reference to temperature—the *insidious*, the *active febrile*, and the *adynamic*. In the first the morning temperature is normal; in the second, the morning temperature will be about 100° or 101°, and the evening temperature 103°-104°. In the third, morning and evening temperatures are both high and not very different; but between these limes irregular fluctuations occur.

The symptoms indicating *disturbances in the alimentary tract* are important. Anorexia is often for a long time one of the most prominent symptoms. It may be accompanied by nausea, vomiting, and pain in the stomach, due either to reflex causes or subacute or chronic gastric catarrh. At the autopsy we often find a normal gastric mucous membrane in one who during life gave the symptoms of acute gastric catarrh. The most common cause which acts in a reflex manner to produce vomiting is a violent fit of coughing. It is important to distinguish between the vomiting due to reflex causes and that due to gastric catarrh. With dyspeptic symptoms the tongue and pharynx are frequently covered with aphthæ. The most important interference with digestion which occurs during the progress of phthisis is due to changes which take place in the small and large intestine. These intestinal changes are marked by more or less tympanitis and by diarrhœa which is often very troublesome and difficult to relieve; few altogether escape these symptoms. Diarrhœa may occur in any stage, but it is more likely to occur during the later stages; in some cases it alternates with hectic fever. It is usually most severe at night. The profuse watery diarrhœa which comes on late in phthisis is called *colliquative diarrhœa*. Hemorrhoids and *fistulæ in ano* are frequent troublesome complications of phthisis, and should always be relieved by surgical interference in the early stages of the disease. The cure of a *fistula in ano* or the healing of an old ulcer is often followed by phthisical developments; and scrofulous joint disease, psoas and lumbar abscesses in children are often followed by phthisis in early adult life.

Cerebral symptoms are rarely pronounced in any stage of phthisis; there is no chronic disease in which the mind is so clear. The hopefulness and buoyancy of spirits which attend its development are remarkable. The least improvement is hailed by the patient as an indication of commencing recovery. He speaks lightly of his unpleasant symptoms, and is very reluctant to admit that his disease is of a serious nature; rarely will a phthisical patient admit that recovery is not possible.

Laryngeal symptoms of phthisis have been considered under the head of Chronic Laryngitis. The pharynx is sometimes the seat of tuberculous processes. Arrest of menstruation is a very frequent occurrence in females who are consumptive. In young females this is sometimes the first noticeable symptom. Its occurrence in advanced phthisis indicates extreme exhaustion, and it is often followed by a more rapid progress of the disease.

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[1] Malassez states that the red discs are diminished in number. The hæmoglobin is also diminished. Leucocytes, fibrin, and calcic phosphate are in excess. Granular masses agglomerate into patches varying greatly in size; and, on a warm stage, they appear to develop into or give rise to organisms which move about in the blood.

The *skin* is pale, and traversed by prominent blue veins. Sudamina and pityriasis versicolor are often observed. The nails curve and become claw-like. The terminal phalanges of the fingers become "clubbed," and this is by some regarded as an important diagnostic symptom, but it occurs frequently in other chronic thoracic affections. It has been regarded as (1) a form of scleroderma beginning in the phalanges and extending centrally over the body; (2) as due to interference with peripheral return circulation; and (3) as an hypertrophy of connective-tissue.[1] The hair becomes thin, dry, gray, and falls out. Œdema of the feet and legs is not an infrequent symptom during the last stage, and its gravity is well recognized by the non-professional. Its occurrence indicates that a fatal issue is not far distant. It may be due to secondary changes in the vessels, but in a large proportion of cases it is due to thrombosis of the veins of the lower extremities, the result of an enfeebled heart.

Physical Signs.—There are two recognized stages in chronic phthisis: a stage of *consolidation*, and a stage of *softening and excavation*.

As the disease advances, evidences of the two stages will be found in close juxtaposition, areas of consolidation surrounding cavities and points of commencing softening.

The physical signs of the stage of *consolidation* vary with the extent of the consolidation according as it involves large areas or small disseminated patches.

Inspection reveals diminished expansion—on inspiration—in the supra- and infra-clavicular regions of the affected side. If there are extensive pleuritic thickenings and adhesions, or if extensive fibroid changes exist, flattening and retraction, most marked at the end of a full inspiration, will be found on the affected side or over the seat of the phthisical development.

Palpation shows more distinctly the loss of expansion on the affected side. Vocal fremitus is slightly increased over the affected lung, although extensive pleuritic changes may render the vocal fremitus less distinct.

Percussion.—The percussion sound will vary with the extent of the consolidation and the condition of the lung-tissue surrounding the consolidated portion. There is always more or less pulmonary resonance. If the consolidation is slight, the percussion sound may remain normal, and localized emphysema may give rise to exaggerated resonance even when consolidated lung-tissue exists. When practising percussion, to recognize a slight consolidation at the apex of the lung, it is important to percuss *from* the trachea rather than toward it. In all cases percussion should be performed at the end of a full inspiration and at the end of a full expiration. Dulness usually appears first under the scapula, next over the sternal end of the clavicle, and gradually extends down, being limited, for a long time, to the apex of the lung. If the dulness is slight at first, it gradually increases and may reach complete flatness.

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[1] In 1,776 cases Pollock found clubbing of the finger-ends in about 25 per cent.

Auscultation.—The auscultatory signs vary greatly in different cases, and at different times in the same case. Over the affected portion the respiratory sounds may be feeble or exaggerated, interrupted, "cog-wheeled," or wavy. The breathing may be *rude* or *bronchial;* or, when rude in character, it may be rude and wavy, rude and interrupted, at the same time being exaggerated, or it may be feeble and rude. At the commencement there may be only a loss in the vesicular character of the inspirations, with a slight rise in the pitch of the expiration.

The pitch of expiration as compared with that of inspiration indicates the extent of the consolidation.

Prolonged expiration, when high-pitched, is very significant. The expiration is prolonged in emphysema, but low-pitched. Wavy or jerking respiration is regarded by some as a friction sound, by others as the result of a narrowing of the bronchi which interferes with the entrance of air into the lung substance. Accompanying or preceding changes in the respiratory murmur, crepitating sounds are heard; they may be crumpling or creaking in character. Small mucous and sub-crepitant râles, if present, are heard loudest after coughing, and, if the consolidation is extensive, they have a metallic ring. It is claimed by some that all the râles that are heard in this stage of phthisis are produced upon the surface, and not in the substance of the lung. This statement is too sweeping, for these sounds are usually circumscribed. They can be changed by coughing, and are often entirely removed by violent coughing, and can be heard before the inspiration is completed. If they were pleuritic, they would remain after coughing, and would retain their distinctly crepitating character at different examinations. Pleuritic sounds are present in a large proportion of cases, but they can be very readily distinguished from râles produced in lung substance. Carefully conducted post-mortem examinations show that, in a large proportion of cases of phthisis, the pleuritic changes are secondary to the changes in the lung substance. Besides, by inflating phthisical lungs after they are removed from the body, sounds similar to those heard during life are distinctly audible if a stethoscope is pressed firmly upon their surface. A systolic murmur over the subclavian artery of the affected side, heard loudest during expiration, indicates that the pleural surfaces at the apex of the lung on that side are adherent. Vocal resonance is usually increased in proportion to the percussion dulness.

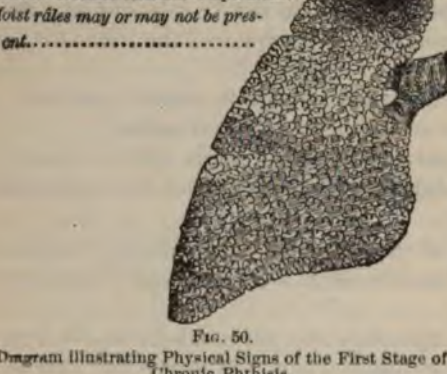
Increased vocal fremitus
Slight dulness on percussion
Exaggerated vocal resonance
Rude or broncho-vesicular respiration.
Moist râles may or may not be present

FIG. 50.
Diagram illustrating Physical Signs of the First Stage of Chronic Phthisis.
Partial infiltration at the apex of the lung.

In the *second stage,* or stage of *softening and excavation,* the physical signs of consolidation become more marked, and new auscultatory signs are developed.

Inspection shows a greater frequency of respiration and a more marked depression above and below the clavicle on the affected side, as well as an increased difficulty in local expansion. In fibroid phthisis the retraction is more marked than in any other variety.

Palpation shows a more marked diminution in expansion of the affected side. On forced inspiration—both hands being placed on the chest equally far from the median line—the fingers that rest over the affected lung will move but slightly compared with those on the opposite side. Vocal fremitus is increased.

Percussion elicits more uniform and widely-spread dulness, which assumes a wooden or tubular character.

Auscultation.—Bronchial breathing and broncophony become more distinct; numerous moist, crackling râles, unchanged by coughing, are heard over a circumscribed space, and have a distinct, sharp, metallic character, unlike the crepitation and bubbling sounds which were heard during the first stage.

As excavation becomes more marked, *inspection* shows greater depression in the infra-clavicular region than existed in the preceding stage, and there is more complete absence of expansive movements during the respiratory acts.

Palpation gives results similar to those of the early part of the second stage. Over large cavities containing air and communicating with a bronchus, vocal fremitus is intensified.

Percussion.—The percussion sound will vary according to the condition of the cavities and their surroundings; over large superficial cavities partly filled with liquid there will be amphoric or "cracked-pot" resonance, if there is a free communication with a bronchial tube. Deeply-seated cavities, when filled, will give deep-seated dulness, and, when empty, an exaggerated percussion sound. A metallic amphoric note is obtainable only from a cavity whose transverse diameter is at least 1¼ to 1½ in.[1] Occasionally, cracked-pot resonance will disappear and remain absent for some time, and no evidence of a cavity can be found where one was known to have previously existed. This happens when the bronchial tube which has communicated with the cavity becomes obstructed in such a manner as to prevent the ingress of air and the egress of fluid.

Auscultation.—Over small cavities with lax walls, low-pitched, puffing, cavernous respiration will be heard. When cavities are surrounded by firm, tense walls, and are of large size, communicating freely with a larger bronchus and are situated near the surface, a musical, or amphoric, respiration is heard. The amphoric echo is sometimes most marked on inspiration; at other times on expiration. The clearness of the amphoric note is no way influenced by the presence of a moderate amount of fluid in the cavity. But when the fluid in the cavity has its level at or above the opening of the bronchus, the incoming air may bubble up and cause *gurgles*. These have a metallic quality, and vary according to the character of the fluid,—the thinner and more watery the fluid, the more bubbling the sounds; the thicker the fluid, the more crackling are the sounds. Gurgles are always most distinct and abundant during and after cough. When very large cavities with rigid walls contain thin liquid, metallic, tinkling sounds may be produced by coughing and speaking. The vocal sounds over large cavities have a metallic or musical quality. Whispering pectoriloquy is a diagnostic sign of a cavity.

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[1] Merbach and Leichtenstern.

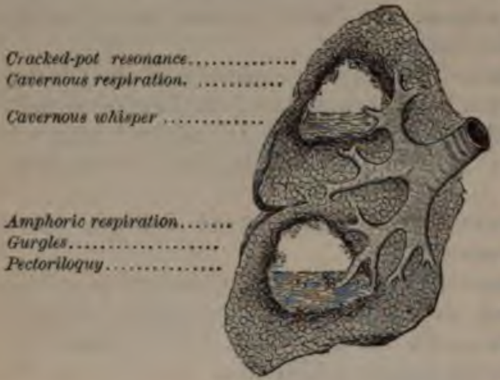


FIG. 51.

Diagram illustrating Physical Signs of Cavities in the Third Stage of Chronic Phthisis.

Differential Diagnosis.—The early stage of chronic phthisis may be confounded with *bronchitis, pulmonary infarction, pleurisy, acute lobar pneumonia, anæmia* with cough and expectoration, and *cancer of the lung.* The evidence of consolidation of lung-tissue is essential to the diagnosis of phthisis. So long as *bronchitis* is accompanied by a temperature of 100° F., and the physical signs show that the bronchitis is general, phthisis is readily excluded; but if the temperature rises to 103° F., and localized crepitant râles develop at the apex of either lung, accompanied by dulness on percussion over the seat of the râles, with a bronchial character to the respirations, then there is reason to believe that phthisis is being developed. If, with these signs, there is gradual loss of flesh and strength, the cough becoming hacking in character, and the expectoration containing fine yellow streaks and blood-stains, it is almost certain that phthisis is developing. The diagnosis between chronic bronchitis and fibrous phthisis rests upon the evidences of consolidation and retraction in phthisis, and their absence in bronchitis.

Infarctions are attended by hæmoptysis and localized areas of dulness. Their etiology, however, is very different from phthisis, heart disease being their chief cause. The blood expectorated in phthisis is of a bright scarlet color; in infarctions it is dark and in the form of *coagula.* Infarctions are most frequently situated in the lower lobes; in phthisis the dulness is apical. The temperature in infarction is usually lower than in phthisis, seldom exceeding 102° F.

In *pleurisy with effusion,* flatness will exist from the base of the lungs to the level of the fluid; the line of flatness will change with a change in the position of the patient; the breathing will be exaggerated above the line of dulness; the range of temperature is lower and does not undergo such marked diurnal changes as in phthisis. The cough is more hacking and is not accompanied by expectoration, and vocal fremitus is diminished or absent. If, after the disappearance of the fluid, the lung remains compressed and bronchial, or broncho-vesicular breathing is present, with feebleness of the patient, hacking cough and "short breath," the differential diagnosis between it and fibrous phthisis is difficult. A localized pleurisy at the apex of the lung, not the result of a general pleurisy, is indicative of tubercular developments.

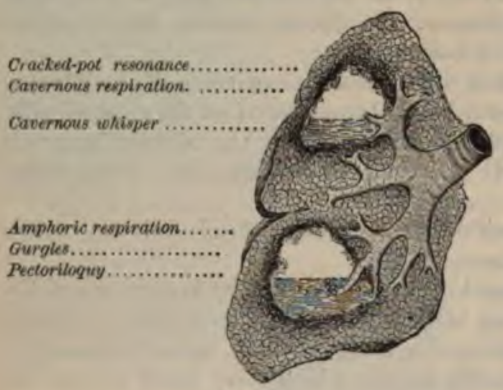
Anæmia with cough aud expectoration is attended by no *febrile* symptoms, and by none of the physical evidences of pulmonary consolidation.

In *cancer of the lung* there is usually bulging of the chest at the seat of the cancerous development; in phthisis there is retraction. In cancer the temperature is often sub-normal, in phthisis it is more or less elevated. The currant-jelly expectoration of cancer is diagnostic. Pain is constant in cancer and intermittent in phthisis. The cancerous cachexia and swollen lymphatic glands also aid in the diagnosis of cancer.

Whenever cavities have formed in phthisis the diagnosis is not difficult if the physical signs are properly appreciated; they can be confounded only with those of bronchiectasis. The rules for the diagnosis of *bronchiectatic* cavities are given under the head of chronic bronchitis. In any case the presence of bacilli in the sputa renders the presence of phthisis probable, and their persistence establishes the diagnosis.

Prognosis.—Chronic pulmonary phthisis is not necessarily a fatal disease. Its morbid processes may be arrested in their early stage in a large proportion of cases. In the advanced stage, or stage of cavities, proper treatment will prolong life, and in some cases permanently arrest the progress of the disease. Recovery has occurred in one-sixth of my recorded cases during the past ten years. Its duration depends on the variety and treatment. In Laënnec's and Bayle's statistics, its average duration is from one to two years. My records of chronic phthisis give an average duration of three years and four months. The younger the subject, the shorter its duration. Phthisis can in no sense be regarded as a self-limiting disease. Some cases, after a period of activity, become stationary and then slowly recover; others slowly but steadily progress to a fatal termination; others, again, pursue a more rapid and fatal course. The course that any case will take is determined more by the conditions under which it is developed than by the natural history of the disease. If an individual has suffered from phthisical developments from which he has apparently recovered, his chances for recovery from a second attack are greatly diminished. The history of phthisical manifestations in early life renders the prognosis unfavorable when the disease develops during middle life.

The prognosis is *unfavorable* when there is a strong hereditary tendency, when phthisis develops early in life, when scrofulous or glandular disease has existed in childhood, when the patient is narrow-chested, when the ordinary pulse-rate is high, and when there is great variation in weight without any apparent cause. Opinions in regard to hæmoptysis vary. Many think its occurrence renders the prognosis favorable, and that there is a larger percentage of recoveries when frequent hæmoptysis occurs. My own experience leads me to the opinion that frequent hæmoptysis in an early stage of the disease is not unfavorable. When œdema of the feet and lower extremities comes on in advanced phthisis, the prognosis is very unfavorable, and a fatal issue is not far off. The following complications character of the fluid,—the thinner and more watery the fluid, the more bubbling the sounds; the thicker the fluid, the more crackling are the sounds. Gurgles are always most distinct and abundant during and after cough. When very large cavities with rigid walls contain thin liquid, metallic, tinkling sounds may be produced by coughing and speaking. The vocal sounds over large cavities have a metallic or musical quality. Whispering pectoriloquy is a diagnostic sign of a cavity.



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Another medicinal agent which has been extensively employed in the treatment of phthisis, and which, for the past twenty years, has enjoyed the reputation of curing this disease, is cod-liver oil. It has been claimed that if this remedy is commenced very early it has the power of arresting the phthisical processes. I am not among those who advocate its indiscriminate use. I doubt if it exerts any specific influence upon the disease; it is more than probable that all its beneficial influence is due to the fact that it furnishes some element essential to the digestion and assimilation of certain nutritive elements. In very many cases the exact manner in which it acts remedially is not well understood. There are three facts which seem to me to afford some clue to the mode of its action: *first*, unless the patient gains in weight while using the oil, it seldom or never proves remedial; *secondly*, flesh and weight may be gained during its administration, and still the phthisical processes steadily progress; and *thirdly*, when it does act remedially, the weight gained is far greater than would result from the oil as a mere element of nutrition. A great gain in weight will sometimes immediately follow the administration of a small quantity of oil. It always acts remedially with more certainty in young persons and children than in the aged; generally, old persons are not much benefited by its use. Those patients who improve under its use take more food than they have been accustomed to previous to its employment, and digest it more perfectly. In some instances diarrhœa will be arrested by its use, and also vomiting of food after eating. In other cases the oil itself will be rejected and its administration rendered impossible. If possible, it should be given in connection with an alkali. At first small doses should be given, not often repeated. A teaspoonful once or twice a day is sufficient to commence with, the dose being gradually increased to a tablespoonful three times a day. No special benefit is to be derived from the administration of large doses. Most patients take the oil best immediately or soon after meals. If it disagrees with the stomach, lying down a short time after taking it will often prevent any disagreeable sensation. Some can better take it upon going to bed at night. It should not be administered in connection with stimulants unless the patient cannot take it in any other way. Regularity and perseverance in its use are essential in order to obtain the full benefit it is capable of producing. If, at times, it seems to disagree with the digestive organs, it may be temporarily omitted, especially during the summer months. The best oil in the market is "Möller's," or what is termed Norwegian oil. Fish-oils of various kinds, cream, glycerine, oils from vegetables, koumyss, malt extracts, pancreatic and pepsin emulsions, etc., have all proved inferior to the simple cod-liver oil. Phosphorus, sulphur, the hypophosphites of lime, soda, and iron, sulphurous acid, the sulphites, are all excellent adjuvants to the oil, but cannot take its place. When intestinal digestion is imperfect, the hypophosphites are especially beneficial. When phthisical subjects become anæmic, iron may be given at each meal if the temperature is below 100° F.; it may be combined with quinine, arsenic, and the mineral acids as *tonics*.

There is a great diversity of opinion as regards the use of alcohol in the treatment of phthisis. Some claim for it a curative power; others maintain that its daily use does harm. The question, therefore, arises, Under what circumstances has experience taught that it is of service, and when it is hurtful? I am convinced that benefit may be expected from the use of alcoholic stimulants only when they increase the desire for food and assist digestion, or when their use is followed by an increase in strength and a disposition to take exercise. On the other hand, if their use causes a rise in temperature and an acceleration of the pulse, followed by a feeling of increased weakness and nervous depression, they will certainly do harm. The belief that alcohol has the power of arresting phthisical development is one which experience does not sustain. The daily use of alcohol for a time may mask phthisical symptoms, and the patient and his friends may fancy that the progress of the disease is stayed; but soon he reaches a condition in which the disease will make rapid progress, and in which a large quantity of stimulants will not give relief. It is unfortunate for a phthisical patient to become addicted to the daily use of stimulants. If an individual with developed phthisis reaches complete recovery while taking alcoholic stimulants freely, I am confident that he would have reached it more rapidly and safely without them. The quantity and kind of stimulants to be used must be determined by the effects; no rule can be given; each case is a law unto itself. Malt liquors and wines do less harm than whiskey and brandy, and are usually more serviceable. Phthisical patients tolerate alcohol to a marked degree.

Cough-mixtures are prescribed by physicians to phthisical patients more frequently than any other medicinal agents. Such mixtures are usually composed of substances which are more or less nauseating; and as the future well-being of every phthisical patient depends upon his powers of digestion, everything that may interfere with the healthy performance of this function must, as far as possible, be avoided. Although a distressing symptom may temporarily be relieved by a cough-syrup, its administration will certainly cause digestive disturbances which will do positive harm.

The relief obtained by cough-mixtures is due, for the most part, to the anodynes which they contain.

This brings us to the question, Should opium be given to phthisical patients? In answer to this question I would say that opium should never be given in any stage of phthisis, except as already noted in connection with quinine, unless the cough is distressing and the patient is unable to obtain the required amount of sleep. Under such circumstances the milder narcotics should first be tried. Opium should be reserved for the later stages of the disease. Its use should be commenced with the smallest dose that will give rest. In the majority of instances I have found that more speedy and satisfactory relief will be obtained from the cough and restlessness during the early stages of phthisis by the inhalation of a few drops of chloroform than from the use of opium; besides, chloroform is less liable than opium to disturb digestion. One must be careful in the use of chloroform; there is danger that phthisical patients may become addicted to its excessive use. Codeia, chloral hydrate, hydrobromic acid, "chlorodyne," creosote, stramonium, and belladonna sometimes act better than opium. All narcotics act only as palliatives, and should be employed only when the symptoms become sufficiently distressing to demand relief. In those cases where a constant hacking or violent paroxysmal cough is excited or kept up by an inflamed or irritable condition of the fauces or larynx, the topical application of sedative or astringent remedies by means of sprays will be found of great service. Of all topical applications for the relief of this condition, a solution of cocaine will be found the most efficacious. It is sometimes imperative to give a stimulating expectorant. Ammonium carbonate in the infusion of wild cherry bark is one of the best. It never nauseates.

Night-sweats are a part of hectic. When quinine does not control them, quinine with opium may do so. Oxide of zinc (gr. ij–iv), gallic or sulphuric acids, arseniate of iron (gr. $\frac{1}{6}-\frac{1}{3}$), ext. of belladonna or sulphate of atropia (hypodermically), muscarine, picrotoxine, ergot,—all may be tried at different times. Atropia is the most reliable. Cold spongings and sponging with acidulated or astringent waters (alum in alcohol) are always agreeable and sometimes efficacious. Capsicum in the sponging water is sometimes serviceable.

Gastric and *intestinal* disturbances are a part of the history of nearly every case of phthisis, and there are two conditions upon which the diarrhœa and distress after eating may depend; viz., either upon a hyperæmic condition of the gastro-intestinal mucous membrane, produced by indigestible food, or upon ulceration of the large or the small intestine. If it depend upon gastro-intestinal hyperæmia, the quantity and quality of the food must be carefully attended to, and a mild saline laxative rather than an astringent must be administered; this should be followed by the daily use of the lacto-phosphate of lime. If the diarrhœa is dependent upon catarrh with or without ulcerations in the small intestine, cod-liver oil and the hypophosphites of lime and soda will often be of service. Five grains of pancreatine given two hours after eating will often relieve the intestinal pain even in the later stages of the disease. If these fail to give relief, ten grains of bismuth, combined with the twelfth of a grain of morphine, after each movement, will almost certainly control the diarrhœa for a time. If the diarrhœa depends upon ulceration of the large intestine, all that can be done is to give temporary relief by opium suppositories. When diarrhœa is persistent and accompanied by rapid emaciation, it is tubercular. Vomiting after meals is often a troublesome attendant of phthisis. Champagne with the food, hydrocyanic acid, pepsin, and a long list of other remedies are recommended for its relief. The most certain relief is obtained by giving the patient a glass of hot water every two hours, followed in half an hour by a teaspoonful of *raw scraped beef* made into a sandwich, at the same time keeping him absolutely quiet in a recumbent posture.

The most valuable remedies for the arrest of *hæmoptysis* are rest and opium. Lead, ergot, ice, and a long list of astringents are recommended. Ergotin hypodermically is much employed. Turpentine is more reliable than any remedy except opium. Local pains in the chest may be relieved by blisters and counter-irritants; strapping the chest so as to render the chest walls immovable often gives marked relief from the pains caused by the circumscribed pleurisies which attend phthisical processes. Dry cupping often gives marked relief from the dyspnœa which accompanies acute phthisical processes.

The *antiseptic* treatment of phthisis has not thus far given satisfactory results, although the recognition of its specific cause has led to the trial of innumerable remedies of this class. Carbolized inhalations have been quite extensively employed, with very favorable results, according to the statements of some observers; but, after quite an extensive trial, my experience is decidedly against their use. The internal or hypodermic use of antiseptics, notwithstanding the strong statements made in their favor by some of their enthusiastic advocates, I have found to fail not only in counteracting the sepsis of advanced phthisis, but in reducing the high temperature which so rapidly exhausts the phthisical patients. The injection of cavities through the chest walls has not been followed by satisfactory results.[1] More recently, favorable results have been reported by careful observers from the use of creosote, both internally and by inhalation. I believe that it is utterly futile to attempt to reach the bacilli imbedded in tubercular or caseous products. It is quite possible that antiseptic inhalations may decrease the infective power of the secretions, and retard secondary inoculations of adjacent lung, and thus be slightly adjuvant to other measures, but their use must be guarded. When used freely they have seemed to me to increase the tendency to hemorrhage. When employed, they are carried more deeply and applied more evenly by the vaporizer and the pneumatic cabinet than any other method of inhalation. The pneumatic treatment has also seemed to me to be of value in relieving pulmonary congestion, assisting the general circulation and promoting general nutrition. It is also an efficient measure for arresting hæmoptysis and freeing the bronchial tubes from accumulations.

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[1] Dr. Pepper, *American Journal Med. Science.* The modus operandi of washing out lung-cavities and the use of drainage tubes in such cases, are fully discussed by Mosler in the October number of the Ber, Klin. Woch., 1878.

The Hygienic Treatment of Phthisis.—The quantity and quality of the air habitually respired is a most important consideration in the hygienic treatment of phthisis. Phthisical patients should sleep in large, well-ventilated and well-lighted rooms with a southerly or westerly exposure. Flannels should be worn next the skin, and the surface must never be exposed to sudden changes of temperature; cold sponging or baths often act as tonics when judiciously employed.

The patient must *live as much as possible in the open air*, and should avoid sedentary occupations, taking systematic daily exercises, but never to fatigue. It is a very great mistake for a phthisical subject to exercise when his temperature is ranging from 102° F. to 104° F.

The dietetic treatment is usually delayed, like most measures, until forced upon the attention by failing digestion. The most absolute rules should be given upon the earliest recognition of the disease, and a system of forced feeding instituted, in which the digestion may be aided by artificial ferments if necessary.

The diet should be varied, and phthisical subjects should become accustomed to drink from one to three quarts of milk each day. The quantity of food taken should be determined by the power of digestion; a phthisical subject should never take more food at a time than can be easily digested. Peptonized foods and preparations of pancreatin will aid a feeble digestion, in the later stages as well as earlier.

The climatic treatment of phthisis is a subject which has recently received much attention, but it is to be remembered that its usefulness is confined almost exclusively to the first stage of the disease, and that no absolute rules can be laid down in regard to it. It is well known that some consumptives thrive best in a warm, moist air, others in a cool, dry atmosphere; some are most vigorous in winter, others in midsummer. Each year's experience impresses on me the conviction that while climate, more than any other agent, has a controlling influence over phthisical developments,[1] each case must be carefully analyzed before any definite directions can be given as to the climate best suited to it. Although we know of no climatic conditions which render phthisis a necessity or an impossibility, still there are conditions which are known to be antagonistic to its development as well as those which favor its development. Scarcely twenty years ago the great desideratum was thought to be a warm, dry atmosphere, but we now know that a cold climate not only does not hasten, but often arrests phthisical processes. The statement has been made that "the higher the altitude the less prevalent is phthisis," but the altitude at which such immunity exists varies with the latitude, and with the idiosyncrasy of the individual.

Mountains and *elevated districts* were thought to be beneficial on account of their elevation alone. But recent investigations show that the absence of atmospheric impurities is the chief element, and that the purity of the air is the chief reason that elevated regions are so beneficial in phthisis. Prof. Tyndall's experiments are of special interest in this connection.[1] Organic germs are more abundant in the air in the city than in the country. Rain and ozone free the air from them, the latter by oxidation. *Rain* cleanses the atmosphere of solid particles and purifies it by washing down ammonia and carbonic acid. The presence of ozone in the air is presumptive evidence of its purity. The air of high mountains and plateaux and along the shore of the ocean is richer in ozone than that of the plains. Prof. Tyndall's experiments show that in early summer the mountains, and in late summer and fall the seashore, have their purest air. The benefit which phthisical patients derive from living near pine forests has long been known. Turpentine exhaled from pine or hemlock forests converts oxygen into ozone, and thus the air of pine forests becomes pure. Direct inhalation of ozone has little power over phthisis; hence it is not the ozone, but the purity of air it induces, that renders the air of certain localities so salubrious. It was formerly thought that resorts where no rain fell for weeks and months were the best suited to phthisical subjects, but experience has taught the reverse. Long-continued rains are certainly unfavorable, but cleansing showers act beneficially. The amount of rainfall is not a sure indication of the amount of moisture in the air of any region, the latter depending more upon the dampness of the soil. The atmosphere of a region with a loose, porous, sandy soil, through which the water filters, and whose surface dries quickly, is never damp; but hard, compact, rocky, or clayey regions, that drain but slowly and imperfectly, hold the moisture and cause a dampness which is a strong predisposing cause of phthisis.[2]

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[1] Laënnec long ago wrote: "Of all the means hitherto recommended for the cure of phthisis, none have been followed more frequently by complete cessation of the disease than change of climate."

Atmospheric temperature is an important element in the climatic treatment of phthisis. Some patients thrive best in a warm sedative climate, others in a cool, stimulating climate. Extended clinical observation leads one to believe that it is neither the heat nor cold of a certain locality, but the *absence of sudden and frequent changes*, which makes it so beneficial to phthisical invalids.

Altitude is regarded by many at the present time as of more importance than any other natural element. As a rule, the atmosphere at elevations of 1,500 or 1,800 feet is purer than on the plains; yet all high altitudes are not thus pure; experiment has shown the atmosphere of some elevated regions to be impure, and that consumptives on such elevations do badly. Something more than altitude is needed to make a given locality suitable to phthisical subjects. Recent investigations show that the similarity in the composition of sea and mountain air at certain times of the year is far greater than was at one time supposed. Mountain air is less dense, less humid, and lower in temperature than sea air, but in both we find excess of ozone and freedom from organic impurities. Both sea and mountain air are cooler and less subject to frequent variations in temperature than the air of the plains. A slight diminution in atmospheric pressure produces no palpable changes. But a great diminution (say one quarter) produces serious disturbances of nutrition, developing a condition which favors rather than retards phthisical developments. The effects of diminished atmospheric pressure vary so greatly in different individuals that no practical deductions can be made.

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[1] After boiling, filtering, and evaporating a vegetable infusion, he hermetically sealed it in flasks, which he transported to the Alps, 7,000 feet above sea level. Some of the flasks were opened during transportation, and in *these* millions of organisms developed in the fluid while the fluid in the flasks that were opened on the mountain remained free from such organisms. By further experiments he showed that dust-laden air was necessary to the procreation of these organisms, and they are diffused through the atmosphere, although the air in different localities may be infected in different degrees.

[2] Laënnec states that the dampness arising from such a condition of soil is one of the most certain developing causes of phthisis, and he makes mention of a locality having such a soil, in which the dampness was so constant and of such a character that two thirds of the resident population died of phthisis.

The question arises, Will this patient be benefited by sea or by mountain air? Beneke's experiments show that tissue changes take place more rapidly on or by the sea than in the mountains. Hence those in whom the process of tissue change needs no hastening, and those with exhausted nervous systems, with an overtaxed brain from excessive mental labor or an all-absorbing business, and who still retain considerable muscular power, should go to the *mountains;* while those past middle life, who have developed phthisis late, who are incapable of much muscular activity, and who therefore require stimulation for the production of tissue change, do best in *sea air*. Sea air is better suited than mountain air to those who cannot bear sudden changes of temperature, while the susceptibility to such changes is greatly lessened by mountain air.

On our own continent is found every variety of climate. Permanent improvement only occurs after a prolonged residence in the place which experience proves best suited to each case. A change of climate should not be made every year. The limited space which can be devoted to the consideration of the localities best suited to phthisical patients in this and other countries will only allow of mention of the most important ones. Every stage of fibroid tuberculosis, no matter how far advanced or *where* the fibroid developments began, is benefited in the high altitudes found in Colorado and about the Rocky Mountains. But there is one grave objection to Colorado as a winter refuge: the enormous monthly, and also the diurnal, range of temperature must severely try any invalid. It is important that every phthisical patient who visits Colorado or any high altitude should place himself under the direction of an intelligent physician before he attempts any exercise.

It is during or before the stage of consolidation that persons with pneumonic phthisis are to be benefited by climatic influences, and a careful analysis of each case is important before directions can be given as to the region most likely to suit his special requirements. The patient must not wander around till he hits upon the place which suits him; much valuable time is thus lost. Except in those who are convalescing from some acute lung disease, a sojourn in a southern climate during the winter seems, after a time, to hasten the degenerative processes. My favorite resorts in the winter, for those recovering from acute pulmonary diseases, are Aiken, S. C., Palatka, Enterprise, and Gainsville, Fla., Thomasville, Ga., and Nassau. These localities are also favorable for those in whom there are evident phthisical tendencies, but in whom, as yet, no physical evidences of pulmonary consolidation exist. My best results, when the evidences of consolidation were present, have been obtained in those who have stayed from one to three years in mountain regions 1,500 to 2,000 feet above the sea. My most decidedly beneficial and permanent results have been obtained in Asheville, N. C., in New Mexico, and in the Adirondack region of New York State. The temperature, rainfall, and surroundings of the latter region are all at variance with preconceived notions of a proper "resort for consumptives," but results are strong in its favor. A camp or tent life in the open air is best for those who can enjoy such life. Excursions and cheerful social intercourse in the open air should always be an object. A dreary spot, even with the best hygienic conditions, will not give favorable results.

I would advocate sanitariums for the phthisical. Not overcrowded hospitals, but cottages and pavilions in sheltered spots, in appropriate climates, and at a given elevation, where privacy and quiet are possible, and where all shall be supervised by a capable and intelligent physician. Minnesota has a dry, cool, exhilarating climate. Southern California, Georgia, and South Carolina have a dry, warm atmosphere. The Bermudas, Bahamas, Florida, Turk's Island, Santa Cruz, and St. Thomas have a warm, moist, and usually healthy climate. The extraordinarily dry belt of country which runs northward from San Antonio, Texas, has begun to endanger the supremacy of Florida as a winter health resort for the consumptive. That this belt offers some climatic advantages for weak lungs over the mild but rather humid air of Florida cannot be doubted. Nassau, the capital of the Bahamas, is a noted resort and one that suits most phthisical subjects past middle life; Matanzas, Cuba, has a dry, warm climate, suitable for a winter home for the enfeebled, but not for those who have developed phthisis. It may be that, for various reasons, a phthisical patient prefers a residence abroad. Dry climates near the sea are Malaga, Riviera, and Algiers. Egypt and South Africa are highly recommended by the English physicians for phthisis. Sea voyages to Australia and New Zealand are recommended in cases of "*hemorrhagic phthisis.*" J. Hughes Bennett finds the lakes of Scotland the best resorts for consumptives in the summer. The Engadine has been strongly advocated by many.

Within the past ten years, *Davos am Platz*, in the Swiss Alps, has been most extensively visited. Williams, Albutt, and other English physicians give very favorable reports of it, and from my limited experience I can fully indorse their statements. It is 5,200 feet above the sea, very dry, but not windy, and not changeable.[1] Davos possesses, also, the unique climatic characteristic of freedom from high winds (the records showing that from October 1, 1880, to March 31, 1881, there were one hundred and thirty-four days with "no wind"), while its "sun temperature" rises even in January, as Dr. Franklin notes, as high as 150°—conditions which admit of much invaluable outdoor exercise by invalids. Some points in Colorado and New Mexico offer all the favorable climatic conditions which are to be found in Davos.

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[1] *Lancet*, 1878, i. 824.

SECTION II.

DISEASES OF THE DIGESTIVE SYSTEM.

(Including Diseases of the Liver, Spleen and Pancreas.)

DISEASES OF THE MOUTH.

The following classification may be made of the diseases of the mouth:

- I. *Stomatitis.*
  - *a. Catarrhal.*
  - *b. Follicular.*
  - *c. Gangrenous.*
  - *d. Ulcerative.*
- II. *The "thrush."*
- III. *The tongue-diseases.*
  - *a. Glossitis.*
  - *b. Cancer.*
- IV. *Inflammation of the parotid gland or "Mumps."*

CATARRHAL STOMATITIS.

Catarrhal stomatitis is an inflammation of the whole, or a portion of the mucous membrane of the buccal cavity and tongue. It may be *acute* or *chronic.*

Morbid Anatomy.—At its onset the mucous and submucous tissue of the tongue and inside of the mouth becomes tumefied, much redder than normal, and *dry.* Later, the mucous and salivary secretions are very much increased. The swelling is greatest over those parts disconnected with bone, as the tongue and cheeks. The tongue becomes covered with a whitish coating, and the red papillæ are visible through it. A copious glairy secretion, slightly acid in its reaction, containing pus and epithelial cells, covers its surface. This secretion has a sourish, but not a fetid odor. In some cases the changes are slight and superficial, in others the tongue is so swollen that it presses on the teeth and becomes indented by them, and the mucous membrane of the cheek and gums fills the space outside of them. The whole surface becomes covered with a tenacious, opaque secretion.

If the process becomes *chronic,* the glands of the mouth become swollen and tender, the filiform papillæ become elongated and pale, and give what is called the "hairy tongue." The tongue is less swollen than in the acute stage; the secretions have a fetid odor. Occasionally, patches of exudation form over the tongue and sides of the mouth, which tend to collect about the teeth. Large diffuse ulcers sometimes occur in adults.

Etiology.—The *acute* form occurs almost exclusively in children during the period of dentition. The *chronic* occurs mainly in adults. Decayed and ulcerated teeth, acid ingesta, and the taking frequently of too hot or too cold fluids, often excite it. The prolonged administration of mercury and preparations of iodine for their specific effect, causes a form which is termed mercurial stomatitis. The excessive use of tobacco is a frequent cause. Gastric catarrh may precede or follow it. It may be an extension of inflammation from wounds of the tongue and fauces. More or less severe catarrhal inflammation of the mucous membrane of the mouth is present in most of the specific fevers, especially in scarlatina. Improper food, bad air, and bad hygienic surroundings will induce it in children.

Symptoms.—The *acute* form commences with a burning, smarting pain in the mouth. The child refuses to take food, or allow the finger to be put in its mouth; it will take freely of cold drinks, is fretful and sleepless, and there is usually a slight rise in temperature. Vomiting and diarrhœa often accompany it. The salivary secretion is increased, and flows from the corners of the mouth, excoriating the parts with which it comes in contact. It may extend into the larynx and cause laryngeal catarrh. When it occurs in adults, there is a slight rise in pulse and temperature, a general feeling of malaise and much difficulty in swallowing. The patient is constantly trying to get rid of the slimy coating on the tongue and mouth, by hawking and spitting. The sense of taste is blunted, and there is usually an unpleasant bitter taste in the mouth. These symptoms are usually accompanied by a dull frontal headache.

In *chronic* stomatitis the breath in the morning has a fetid odor, the taste is vitiated, and there is often great depression of spirits. Rarely is the digestion interfered with.

Differential Diagnosis.—Catarrhal stomatitis may be mistaken for the changes which take place in the tongue and mouth in some of the specific *fevers*. In catarrhal stomatitis the coating of the tongue is soon followed by a copious salivary secretion; while in fevers the tongue becomes dry, and the detachment of brown crusts leaves a glassy, smooth surface. In catarrh, the appetite for solid food and the digestive functions are not much changed, while in fevers there is great thirst and repugnance for food. There are slight, if any, constitutional symptoms in catarrh, while in incipient fever there are marked constitutional symptoms.

Prognosis.—The *acute* form generally terminates in recovery within a few days. *Chronic* oral catarrh is very persistent and stubborn, and rarely yields to treatment.

Treatment.—In young children the diet should be cold milk with lime-water. The mouth should be washed with a slightly alkaline wash, and chlorate of potash given internally. In all cases the cause should be removed, and the bowels regulated with rhubarb and soda.

In *chronic* catarrhal stomatitis, after the removal of its exciting causes, moderately strong alkaline washes should be frequently used, and in obstinate cases a weak solution of nitrate of silver will be found most efficacious. Carbolic acid sprays relieve the offensive odor and other unpleasant symptoms.

FOLLICULAR STOMATITIS.

Follicular, *aphthous,* sometimes called *croupous,* stomatitis is a variety of inflammation of the mouth, in which the mucous follicles are primarily and chiefly affected.

Morbid Anatomy.—On the anterior portion of the tongue, and on the mucous surfaces of the gums and cheeks, there appear small vesicle-like elevations, semi-transparent, and having a red zone about their base; these are called "aphthæ;" some regard them as a peculiar deposit, others as a local croupous exudation. They are often numerous; after they have ruptured they leave an irregular gray surface, resembling a small ulcer, which heals slowly. Occasionally a number of aphthæ coalesce and form irregular ulcer-like or excoriated patches. In the majority of cases the ulcers soon disappear, new crops appear and the disease may run a tedious course. Dirty white or yellow sloughs cover the ruptured aphthæ, and gradually separate, leaving no scar. Follicular ulcers on the inner side of the lips sometimes occur at the menstrual epoch, or during pregnancy and lactation; ulcers like these rarely occur in men.

Etiology.—Aphthæ may accompany any disease of the tongue or mouth. It is, like most oral diseases, chiefly prevalent among children during dentition, and is rare after five years of age. It is idiopathic, or a sequela of one of the exanthemata. Unripe fruit, candy, and indigestible food remaining in the child's mouth will cause it. Bad hygienic surroundings and a weakly, badly nourished state of the body, are its principal predisposing causes. It sometimes prevails epidemically.

Symptoms.—Aphthous stomatitis shows itself in very young children by pain on taking the breast and in swallowing. Older children have pain on talking and masticating. There is a slight febrile excitement and enlarged and tender sub-maxillary glands. Salivation occurs, and the parts about the mouth and chin become excoriated by the saliva, which continually runs over them. Feculent diarrhœa is common, and there is more or less interference with digestion.

Differential Diagnosis.—This cannot be confounded with any other disease.

Prognosis.—It is never fatal; it generally disappears as soon as the causes that produced it are removed.

Treatment.—Correct any intestinal disturbance that may exist with small doses of rhubarb and magnesia, or mild salines; restrict the diet to milk. Wash the mouth with a weak solution of glycerine and borax, or chlorate of potash. In severe cases the mouth should be washed every few hours with a dilute mineral acid, or nitrate of silver. In weak children, when the general health is impaired, stimulants may be given with benefit.

GANGRENOUS STOMATITIS.

Gangrenous stomatitis, "cancrum oris," or sloughing phagedæna of the mouth, is a formidable disease of childhood, in which the tissues of the cheek are prominently involved.

Morbid Anatomy.—There is first a hard swelling developed in the cheek, the skin over it being red, shining, tense, and brawny. In the mouth, at the side of the indenture, there is a deep, ragged, angry, unhealthy ulcer covered with a dark, ashy, or brown colored slough. The adjacent tissue is œdematous, and hemorrhage from the livid and swollen part sometimes occurs. The ulcer in the cheek rapidly extends and deepens, emits a very fetid odor, and often perforates the walls of the buccal cavity. The slough may occupy the whole of one side of the mouth, the teeth may become loosened, and caries, or necrosis of the inferior maxilla, result. If the ulcerative process is not extensive, separation of the slough may occur, and the ulcer heal by granulation and cicatrization. The facial vein may be implicated, and then pyæmia, with multiple abscesses, may result.

Etiology.—This is a very rare disease. It occurs principally in debilitated children between two and five years of age who are convalescing from some form of acute disease, such as scarlet fever. Whether it is contagious or not has never been determined. It sometimes follows the prolonged use of mercurials. Bad air, insufficient food, and anti-hygienic surroundings, predispose to it.

Symptoms.—It commences with pain in the mouth, which is increased by movement of the jaws. Then the local changes already described appear on the cheek and gums, and an abnormal quantity of saliva, mixed with a putrescent fluid, often with blood, flows from the affected side of the mouth. The breath has a peculiarly offensive odor. The adjacent glands become enlarged and tender. As the disease advances, the constitutional symptoms of septicæmia are developed. In most cases the child after a time becomes drowsy, passes into coma, and dies.

Differential Diagnosis.—Cancrum oris may be mistaken for "*malignant pustule.*" Malignant pustule attacks the skin and exposed parts *first*, while gangrenous stomatitis begins in the mucous membrane of the cheek or about the gums, involving the skin *secondarily*. Malignant pustule is at once accompanied by constitutional symptoms, and soon followed by the phenomena of a septic or typhoid fever, while cancrum oris is without pyrexia or loss of appetite at its onset, and severe general symptoms do not come on till late.

Prognosis.—This is an exceedingly fatal disease—nineteen out of twenty die. In the few cases where the process has been mild, recovery has occurred within two weeks from its commencement. The *complications* are pneumonia, bronchitis, and pyæmia. Death may occur from exhaustion or from one of the above named complications.

Treatment.—Prompt measures are indicated at the onset of this affection. Nitrate of silver, and even strong nitric acid, should be thoroughly applied to the slough, and the mouth frequently washed with solutions of carbolic acid and chlorate of potash. The best internal remedies are quinine and hydrochloric acid. The diet should be highly nutritious; stimulants may be freely given, if indicated. When the child cannot swallow, beef tea and brandy enemata should be administered.

ULCERATIVE STOMATITIS.

Ulcerative stomatitis, or *noma*, is a variety of inflammation of the mouth chiefly affecting the gums and spreading over a large extent of surface.

Morbid Anatomy.—The gums are hyperæmic and tumefied. Sometimes they assume a purplish color, separate from the teeth, and are covered with a pulpy gray-white material which disintegrates, becomes soft and dark, and gradually spreads to the lips and side of the cheek. This gangrene-like slough may gradually extend until the gums are destroyed. In some few instances little vesicles precede the slough. If the slough is removed as soon as it appears, the gums underneath will be found red, bleeding and granular. The teeth become loosened and often drop out, the tongue enlarges and has a sodden appearance, and the mucous membrane of the cheek swells so that it often receives the impression of the teeth. Sometimes the bones about the face lose their periosteum and exfoliate. When recovery takes place deep cicatrices may remain and cause more or less distortion of the face.

Etiology.—Noma, or ulcerative stomatitis, is met with only in children from one to ten years of age. It occurs among those who inhabit filthy localities, who are badly fed and compelled to breathe unwholesome air. Dampness seems to exert a predisposing influence, and the disease is most prevalent during the autumn months. It is probably contagious, for well-marked epidemics of it are recognized. It is common after asthenic inflammation and the eruptive fevers. The prolonged use of mercurials will cause ulcerative stomatitis.

Symptoms.—The mouth is hot and painful for some time, and then appear the changes already described. There is pain on chewing or speaking, and there *may* be slight febrile excitement, although constitutional symptoms are not prominent. There is an increased flow of saliva which has a very offensive odor, and is mixed with blood and shreds of the pulpy mass. There is enlargement and tenderness of the sub-maxillary glands. In some cases the child will pick at its mouth and throat, and very often loosens and swallows some of the shred-like sloughs. The appetite may not be impaired, though the bowels are disordered, and the child is restless and sleepless. The upper lip becomes swollen, dark-red, and projects outward, while the mouth is kept widely open to prevent painful contact with the lips or tongue. The excessive salivation soon decreases, but the unpleasant fetor of it and of the breath persists. Late in the disease the adjacent glands become enlarged and tender.

Differential Diagnosis.—Ulcerative stomatitis may be mistaken for "*cancrum oris*," or *gangrenous stomatitis*. It is a local disease, while cancrum oris is attended by constitutional symptoms; it begins in the gums, while gangrenous stomatitis begins in the cheek. The progress of noma is slow compared with the very rapid extension of cancrum oris. The livid redness, the dark swelling, and the ashy slough of cancrum oris are absent in ulcerative stomatitis.

Prognosis.—This is good. Its duration is about eight days, but sloughing about the gums may continue for weeks.

Treatment.—The treatment is the same as in aphthous stomatitis. The chlorate of potash may be used as a wash or gargle and internally, and will usually arrest it. In many instances, fresh air, cleanliness and a restricted diet are all that is necessary to effect a cure. If the ulceration spreads, the application of nitric acid, and sometimes the employment of the actual cautery, must be resorted to. For the profuse salivation which is sometimes so troublesome, belladonna has proved efficacious.

THRUSH.

Thrush, sprue or *muguet* is an aphthous disease of the epithelium of the mouth and tongue, due to the growth of the germs of the thrush-fungus, the *oidium albicans.* It was formerly classed as an exudative inflammation.

Morbid Anatomy.—The mucous membrane of the mouth assumes a dark red color, and upon the most superficial layer of the epithelium there appear numerous small, round whitish spots,—"aphthæ,"—which give to it a flocculent or curdy appearance. These spots are often aggregated in groups of two or three; at first, as they enlarge, they fall off or can be readily removed, but are soon reproduced and run together in patches. The development in thrush of the *oidium albicans* and of its frequent parasitic companion, the leptothrix buccalis, in and between the epithelial cells may continue until the mucous tissue is invaded. The epithelium becomes swollen and loosened, the tongue and inside of the mouth are covered with a yellowish pultaceous, creamy mass, underneath which the mucous membrane is of deep red color, and the papillæ are enlarged. In the new-born it occurs most abundantly about the boundary line between the hard and soft palates; in adults, on the mucous membrane of lips, cheek and end of tongue. It may invade the pharynx, œsophagus and stomach. It has been found in the lungs and air-passages and about the breasts and genitals of infants. A microscopic examination of a patch shows it to contain mucous and epithelial cells, fat spherules, and the spores and filaments of the oidium albicans. The spores are round or ovoid and form masses of varying sizes, while the filaments coming out of the spores are cylindrical, curved or branched, and consist of long cells, which are constricted where they join one another, each cell being filled with granules.

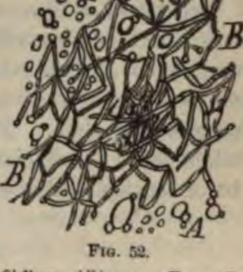

FIG. 52.

Oidium Albicans. From the tongue in a case of "Thrush." A, A. Spores.—B, B. Mycelial threads. × 300.

Etiology.—In children this disease occurs from birth to the second year, and is very rare after that time until adult life. These parasitic plants grow best in the presence of acids, hence the acid secretion of the mouth for the first six or seven months predisposes to it. All food or drink that will produce irritation of the stomach or intestines, and make the intestinal contents acid, predisposes to it. Want of cleanliness in the care of nursing-bottles, spoons, etc., is one of its principal causes, consequently it is more frequently met with in children brought up on the bottle, especially in asylums. In adults thrush occurs toward the end of any long exhausting disease, such as cancer, or consumption.

Symptoms.—In children the mouth becomes hot and painful. The child will not allow its mouth to be touched. An examination shows the mucous membrane to be drier than natural; soon after, the peculiar thrush aphthæ appear, and there is salivation, which is always markedly acid. The lips swell and become everted. Diarrhœa is frequent and the passages are often green and smell of fatty acids; so acid are they at times that they cause an erythema about the anus. If this condition persists, the buttocks and parts around the genitals become excoriated. Besides this diarrhœa, vomiting and purging give additional evidence of gastro-intestinal disturbance. In adults suffering from exhausting disease, the mouth becomes hot, dry and painful before the thrush appears, and there is difficulty in swallowing, after which the mouth and tongue soon present the characteristic appearance of the disease.

Differential Diagnosis.—The presence of the parasite establishes the diagnosis.

Prognosis.—In vigorous children the average duration of this affection is from eight to ten days, but in feeble infants it often lasts for months. Its only serious complication is gastro-intestinal catarrh. Death may result from the exhaustion of the diarrhœa.

Treatment.—The most important thing to be accomplished in the treatment of this affection is to arrest or counteract the acidity of the secretions of the mouth. After each feeding the mouth must be thoroughly cleansed with borax and glycerine, or a weak solution of carbolic acid, and sulphate of soda. The diet should be restricted to milk with lime-water; when there is emaciation, cod-liver oil and the lactophosphate of lime will be of service. The bowels must be regulated as in follicular stomatitis.

GLOSSITIS.

Glossitis is an inflammation of the parenchyma of the tongue. It may be *acute* or *chronic*, and when chronic, is generally circumscribed.

Morbid Anatomy.—There is first intense hyperæmia, causing slight swelling and intense redness of the tongue. This is soon followed by so great an enlargement of the organ that it entirely fills the mouth and protrudes beyond the teeth. Its surface is covered by a thick secretion, and its substance assumes a pale or grayish color. The œdematous condition may rapidly subside and leave the tongue in its normal state, or the inflammation may be so intense that small abscesses form which leave deep cicatricial depressions, giving the tongue an uneven and lobulated appearance. In some instances the tongue may remain enlarged and hardened for life. There is a rare variety of glossitis which does not invade the deeper structure of the tongue, but is confined to its mucous and sub-mucous tissue, causing thickening and sloughing of its surface, with depressions similar to the cicatricial depressions of the parenchymatous variety.

Chronic glossitis occurs chiefly in patches along the edges of the tongue; the thickening, induration, and cicatricial depressions occur in circumscribed spots. When chronic glossitis is general, the tongue is uniformly enlarged and its color is much redder than normal, some spots being darker than others; its movements are interfered with, and its surface presents the appearance of eczema of the skin.

Etiology.—Acute glossitis may develop under the influence of mercurial poison, or as a consequence of direct injury. Croton oil and other acrid matters taken into the mouth may cause it; burns, blows, and the poison of insects have caused it. Chronic glossitis occurs in the old without any apparent cause. It may be produced by disease of the teeth, or of the maxillary bones, and may, in some instances, result from the action of the materials of which false teeth are made.

Symptoms.—With the enlargement of the tongue in acute glossitis, there is great restlessness and anxiety, accompanied by an increase of the pulse-rate, and an elevation of the temperature. In some cases, there is profuse salivation, and the swollen tongue protrudes between the lips. There is a sensation of heat in the mouth, and the swelling often causes severe pain. The glands at the angle of the jaw are enlarged and tender, and all movements of the tongue in talking, chewing or swallowing become exceedingly painful and frequently impossible. Dyspnœa and inability to lie down are sometimes caused by the obstruction to the free entrance of the air into the lungs. When the veins in the neck are compressed, cyanosis of the face is marked. The patient is anxious, and very much depressed, and may show signs of asphyxia; indeed death has occurred from suffocation in extreme cases. When it terminates in suppuration, the constitutional symptoms become severe, and all the oral symptoms are intensified. When clefts remain in the tongue after glossitis, the ulcers in them are painful, but otherwise there is no inconvenience. In superficial glossitis, which is apt to be protracted, any movement of the tongue is painful, and there is constant salivation. In chronic glossitis patients sometimes complain of a dull aching in the tongue, and in some cases movements of the tongue induce pain of a burning character.

Differential Diagnosis.—Chronic circumscribed glossitis may be mistaken for *cancer*. Cancer develops rapidly, and chronic circumscribed glossitis almost imperceptibly. Cancer tends to speedy ulceration, and hemorrhage is frequent, while glossitis passes on to induration and there is no hemorrhage. Fetor of the breath is present early in cancer, while it is slight or altogether absent in glossitis. In cancer the pain is sharp and lancinating, running along the branches of the fifth nerve, while there is only a dull pain in glossitis. In cancer the adjacent lymphatics are early involved, in glossitis they are uninvolved. In cancer, emaciation and cachexia are marked; these are absent in glossitis. A microscopical examination of a portion of the diseased tissue will establish the diagnosis.

Prognosis.—In acute glossitis, the prognosis is uncertain, for suffocation may occur unexpectedly; generally it subsides in from three to seven days. Of the modes of termination, that of thickening and induration is the most common, and is rarely entirely recovered from.

Treatment.—In acute glossitis, ice should be freely applied to the tongue and a mild cathartic administered. If the patient is not able to swallow castor oil, a turpentine enema may be given. If the swelling interferes with respiration, free and deep incisions on the upper surface must be at once made, and if abscesses form they should be promptly opened and washed out with some disinfectant fluid. The ulcerations occurring in glossitis should be treated in the same way as those of ulcerative stomatitis. In the chronic form, if possible, remove the cause. In superficial glossitis, the local application of carbolic acid will be found the best remedy. If suffocation becomes imminent in either variety, tracheotomy should be performed.

CANCER OF THE TONGUE.

The most common variety of cancer of the tongue is epithelioma.

Morbid Anatomy.—At some point that has been subjected to constant irritation, or in some ulcerative cleft in the tongue, there appears a small unhealthy ulcer or a small deeply seated nodule. When appearing on an otherwise healthy tongue, its locality is usually on its edge. In whatever way it may begin, an ulcer quickly forms, circular in shape with ragged everted edges, and a wide indurated base. The surface of the tumor has a dirty white or grayish-red appearance, is papillated and friable, and commonly of a firm consistency. As the disease advances, it may involve the whole tongue, which is then larger than normal, unevenly lobulated, and covered with small ulcerations. The mucous membrane on the floor and sides of the mouth may be secondarily invaded. As the deeper tissues are encroached upon, hemorrhages occur. The sub-maxillary and sub-lingual glands early take part in the cancerous development, and the oral cavity may be filled with the cancerous mass. On scraping the surface of an epithelial cancer, a grayish granular mass is found beneath, a portion of which under the microscope will show the characteristics of an epithelioma.

Etiology.—Cancer of the tongue is met with most frequently in middle life, between the ages of thirty-five and sixty, and occurs in men more often than in women. Its chief exciting cause is some local irritation, as from a projecting or carious tooth. It may develop in syphilitic fissures. Occasionally it appears on a tongue whose mucous membrane has, for a long time, been thickened and indurated. Usually there is an hereditary predisposition to cancerous development. It may develop without any discoverable cause.

Symptoms.—In most cases, from the onset there is a sharp pain at the seat of the disease. This pain is aggravated by any movement of the tongue, and generally runs along the branches of the fifth nerve. Salivation is profuse, and swelling of the lymphatics in the neighborhood is present early. Hemorrhages not infrequently occur, which increase the anæmia that attends the cancerous cachexia. The disease runs a very rapid course, the pain becomes agonizing, and a fatal termination may at any time occur from hemorrhage from the lingual artery, or suffocation may result from mechanical interference with respiration.

Differential Diagnosis.—This disease may be mistaken for *syphilitic ulceration.* A syphilitic ulcer is long and oval or irregular in shape, while cancer is circular. A syphilitic ulcer is developed slowly and with little or no localized pain, but cancer spreads rapidly and is accompanied by severe pain. The constitutional symptoms of syphilis are usually well marked, and the ulcer improves under anti-syphilitic treatment, while the evidences of syphilis are absent in cancer. A microscopical examination of a small portion of the ulcerating surface removes all doubt in diagnosis.

Prognosis.—The disease advances rapidly; its average duration is about fourteen months. I have known cases to last two years. Death results from the cancer, marasmus, exhaustion from hemorrhage, or from starvation, as the intense pain in eating causes the patient to refuse food. The constant and long-continued pain hastens the fatal termination. If, after removal, it does not reappear, death may result from cancerous developments in other parts of the body.

Treatment.—The relief of pain and the maintenance of the vital powers are the principal indications. The hypodermic use of morphia is the best means of relieving pain. Antiseptic gargles are grateful, and counteract the offensive odor of the breath and the unpleasant taste. The checking of hemorrhage, removal of the growth, removal of the tongue, ligation of the lingual artery and division of the gustatory nerve, belong to the surgical rather than to the medical treatment of the affection.

PAROTITIS.

Parotitis, or "mumps," is a catarrhal inflammation of one or both parotid glands; rarely are the other salivary glands involved. It is of two varieties, specific and non-specific. The latter is also called symptomatic.

Morbid Anatomy.—The left parotid is usually first affected. The disease commences in the gland-ducts, not, as was formerly supposed, in the intercellular substance. Both varieties begin as a catarrh, the intense initial hyperæmia of the parotid being followed by a serous exudation and a soft swelling of the gland. From the few cases that have been examined at this stage, we learn that there is œdema of the acini, and that the gland is reddened and injected, presenting a fleshy appearance. The connective-tissue about the parotid and often the parts beyond it are involved. The gland which is so enormously swollen may decrease to its normal size by the absorption of the exudation; or, when the process is very severe, the idiopathic variety may end in abscess; this termination is much less frequent than in metastatic parotitis. Occasionally, the gland remains permanently enlarged. It may atrophy.

Metastatic parotitis begins with a catarrh of the ducts; there is first hyperæmia, followed by an exudation into the ducts, which is semi-transparent or yellow. The acini are wholly or partly filled with pus, and there result numerous little spots of suppuration, but if the process is severe and rapid the interstitial tissue becomes involved, and the whole gland may be converted into an abscess. The suppurating process may extend to the neighboring bones, muscles, connective-tissue, or, rarely, to the cranial bones and meninges. As in "cancrum oris," if the adjacent veins are involved, pyæmia and multiple abscesses are the result. The Eustachian tube may be involved, and sometimes pus burrows under the muscles at the back of the neck.

Etiology.—Specific parotitis seldom occurs except as the result of contagion. It is both contagious and infectious, and seems to be favored in its development by dampness. Consequently, it is common in autumn and early spring, and among those who live in cold, damp cellars. It prevails most in crowded localities, such as asylums and foundling hospitals. It resembles the exanthemata, in that it attacks the same gland but once. The exact cause of metastatic parotitis is obscure. The reason of its development in small-pox, typhus, typhoid, measles, pyæmia, septicæmia, cholera, and rarely in pneumonia, cannot always be given. The period of incubation in specific parotitis varies from seven to fourteen days.

Symptoms.—In specific parotitis for a short time preceding the glandular enlargement there is chilliness followed by flashes of heat, frequently by dull pains in the limbs, general lassitude and loss of appetite. In nervous children headache, delirium, and often convulsions are its premonitory symptoms. In from thirty-six to forty-eight hours after these phenomena, there is a sensation of stiffness about the angle of the jaw, followed by pain and swelling in the parotid region. The pain is increased by speaking, swallowing, and by pressure. Both glands may be simultaneously affected but usually only one is involved at a time. The tumor is firmer over the centre than at its circumference, the outer rim being slightly œdematous and pitting on pressure. Moderate salivation occurs in a few instances. The disease reaches its height in from three to five days, and the swelling of the gland begins to subside on the seventh or eighth day.

In the non-specific variety, if abscess form, constitutional symptoms indicative of the formation of pus are developed, and the adjoining lymphatics become enlarged. These abscesses may open externally, into the mouth, pharynx, or external ear. During or after the decline of specific parotitis, metastasis to the testicles, mammæ, or ovaries may occur. Metastasis to the meninges of the brain (a rare occurrence) is indicated by delirium and active cerebral symptoms, which terminate in coma and death. Non-specific parotitis, developing during some severe constitutional disease, produces few symptoms of its own, excepting the physical evidences of the tumor, which shows a tendency to suppurate from its beginning; very soon a lobulated red swelling exhibiting fluctuation at various points is developed, which soon discharges laudable pus. In this variety, but one gland is usually involved.

Differential Diagnosis.—There is little difficulty in recognizing this disease by the situation of the swelling.

Prognosis.—In the specific variety the prognosis is favorable, the gland usually returning to its normal size and consistence in from ten days to two weeks. If an abscess forms and implicates the ear or Eustachian tube, permanent deafness may result; when orchitis, mammitis, meningitis and other metastases complicate the parotitis, the prognosis is more or less unfavorable. Death may result from meningitis with active brain symptoms. Non-specific parotitis, occurring in the course of any acute general disease, must be regarded as a very unfavorable symptom.

Treatment.—Specific parotitis is a self-limiting disease; during its active period the patient must be kept in an even temperature, and a mild saline cathartic may be administered. The diet should be non-stimulating. If the parts are painful, warm fomentations may be applied. If the patient suffers severe pain or is restless, the bromide of potassium or hydrate of chloral may be given. Inunctions of oil, when the swelling is disappearing, are said to aid in reducing it. When orchitis or meningitis occurs it is to be treated as a complication. During convalescence tonics are indicated. The non-specific variety requires no treatment except the use of means which hasten suppuration and support the patient.

DISEASES OF THE PHARYNX.

I. *Tonsillitis:*—(*a.*) *Acute* or *Quinsy;* and (*b.*) *Chronic.*—II. *Inflammations:*—(*a.*) *Catarrhal* which is either *Acute* or *Chronic;* and, (*b.*) *Membranous,* which is either *Croupous* or *Diphtheritic.*—III. *Retropharyngeal Abscess.*

TONSILLITIS.

"Quinsy sore throat," sometimes called phlegmonous pharyngitis, is an inflammation of the parenchyma of one or both tonsils, and may be acute or chronic.

Morbid Anatomy.—It must be remembered that beneath the external covering of the tonsils fibrous trabeculæ enclose numerous groups of lymph-follicles, and that this interstitial stroma is very vascular. In acute tonsillitis there is first hyperæmia and swelling of the gland and of the parts adjacent to it. The angry, red, lobulated surface of the enlarged gland and the back part of the tongue are covered with a tough, gelatinous or creamy secretion, and the uvula and anterior pillars of the fauces become swollen and œdematous. When suppuration is to occur a point on the surface of the inflamed tonsil becomes prominent, soft and fluctuating. The loose cellular tissue surrounding the tonsils may be involved in the suppurating processes. If chronic tonsillitis follows, subsidence of the acute hypertrophy of the stroma occurs, accompanied by enlargement of the supplying vessels. Chronic enlargement of the tonsils may also be developed slowly

Etiology.—The acute form occurs most frequently in children and in young adults. There seems to be a predisposition in some persons to this affection. One attack predisposes to others. The causes which predispose to quinsy induce acute pharyngitis. Chronic follicular pharyngitis may be produced by prolonged use of the voice in public speaking or singing, or by the excessive use of tobacco or of spirituous liquors. Weak, scrofulous persons, and those with chronic thoracic disease are frequently affected with it. Its chief cause is repeated acute attacks.

Symptoms.—Slight fever may usher in an attack of acute pharyngitis, or precede the development of its local symptoms. The throat first becomes dry and redder than normal, and movement of the parts produces pain in the direction of the Eustachian tubes, so that swallowing and speaking become painful. The elongated uvula may induce violent fits of coughing. The local symptoms are very severe; there will be more or less regurgitation of food through the nose. If particles of food do not readily pass into the œsophagus they may enter the larynx and cause severe fits of coughing. In these severe cases there is a nasal twang to the voice, and any movement of the throat, tongue, or mouth is carefully avoided on account of the pain it produces. If the inflammation invades the Eustachian tube, deafness may result, and not infrequently the tympanum is perforated by the pus which collects in the middle ear. The extension is more often forward, so that the mucous membrane of the tongue and mouth presents the same condition as that of the pharynx. These symptoms may gradually subside, after a few days, and the viscid secretion disappear from the tongue, mouth and pharynx. If it becomes chronic, the voice becomes hoarse, and there is a stridulous cough accompanied by a thick, tough mucous expectoration, often containing small firm, yellow masses. There is constant irritation of the throat, which is variously described as dry, tickling or tingling, and the secretion may be so much diminished that slight hemorrhage may occur from the membrane when pressed upon. All these symptoms are most marked in the morning. The symptoms in a long standing case may lead to anxiety on account of the supposed existence of pulmonary phthisis. These are all aggravated by "catching cold," atmospheric changes, and the prolonged use of the voice.

Differential Diagnosis.—Follicular pharyngitis may be mistaken for *pulmonary disease*, and the early stage of mild chronic catarrh often excites suspicion of *syphilis*. In the former case an exploration of the chest and an examination of the throat will at once decide, while the presence or absence of the constitutional signs of syphilitic infection will establish the diagnosis in the latter instance.

Prognosis.—Acute catarrhal pharyngitis is a very mild disease, subsiding completely in most cases within one week from its onset, while chronic pharingitis is the most persistent of all catarrhal affections.

Treatment.—In acute pharyngitis, ice-cold carbonated water affords the greatest relief during the first twenty-four hours. The throat and mouth should be frequently sprayed with a solution of alum, tannin, or sulphate of zinc, and at the same time the wet pack should be applied to the throat either hot or cold, but they should *not* be alternated. In chronic pharyngitis, the first thing to be done is to remove the cause and live an outdoor life. Spraying the parts two or three times a day with the astringent just named, or a mild solution of nitrate of silver, will generally afford temporary relief. In some cases capsicum or guiacum may be advantageously combined with the astringents, and in obstinate cases the local use of iodine or a twenty per cent. solution of carbolic acid may be resorted to. In chronic (follicular) pharyngitis a nutritious diet is especially important. German physicians recommend very highly the use of mineral waters, but alkaline gargles are as effective as a residence at some "spring."

MEMBRANOUS PHARYNGITIS.

Under this head are included both croupous and diphtheritic inflammations of the pharynx. Croupous inflammation may be primary, but diphtheritic inflammation is always secondary. This form of pharyngitis is considered in the history of membranous laryngitis and diphtheria.

RETRO-PHARYNGEAL ABSCESS.

Suppuration behind the pharynx, in the areolar tissue between it and the vertebræ, is known as retro-pharyngeal abscess.

Morbid Anatomy.—This is a cellulitis, and its morbid anatomy is the same as that of cellulitis terminating in an abscess elsewhere. It belongs properly to the province of surgery.

Etiology.—Retro-pharyngeal abscess occurs more frequently in children than in adults. It is developed during the progress of caries of the cervical vertebræ. It is rarely if ever due to the extension of inflammation from the pharynx. A strumous diathesis predisposes to it. Sometimes it appears late in pyæmia, septicæmia, typhoid, typhus, scarlet fever and measles. Now and then it occurs without any obvious cause.

Symptoms.—The first symptom is dysphagia. With this there is stiffness of the neck, slight difficulty in articulation, and a change in the tone of voice, which becomes nasal in character. On examining the pharynx its calibre will be found diminished by a bulging from behind and perhaps a little to one side; the pharyngeal mucous membrane is redder than normal, and there may be a slight swelling about the angle of the jaw. The head is thrown backward, and any attempt at flexion causes dyspnœa; the jaws seem to be partially locked. There is regurgitation of food through the nose. In young children there may be snuffling, choking, coughing and great dyspnœa, with a certain hoarse tone to the voice. The mouth is filled with a mucous secretion.

As the abscess increases in size the tumor may be seen nearly filling the space behind the soft palate. This swelling is soft, elastic and fluctuating, sometimes rupturing when pressed upon, and discharging an offensive pus. If it opens spontaneously the pus is vomited, swallowed, discharged through the nose, or is inspired into the trachea and may cause suffocation. Again, the abscess filling the pharynx may press on the rima glottidis and epiglottis and cause œdema glottidis. In rare instances the pus makes its way around to the opposite wall of the pharynx, and then breaks into the œsophagus or trachea, or burrows into the pleural cavity or even the pericardium. It may burrow between the tracheal muscles and appear at the anterior part of the neck.

Differential Diagnosis.—When fully developed, a careful examination of the pharynx will detect at once the existence of a retro-pharyngeal abscess.

Prognosis.—The prognosis is bad whenever caries of the spine has caused the abscess. The complications which may cause death are œdema glottidis, pleurisy, pneumonia, and pericarditis. Death may result from suffocation from pressure.

Treatment.—Open the abscess early, and never wait for its spontaneous rupture. The position of the child when the bistoury is used should be such that the escape of pus through the mouth is facilitated.

DISEASES OF THE ŒSOPHAGUS.

The following diseases of the œsophagus will be considered:

I. *Inflammation*, either *catarrhal* or *membranous*, including *Stricture of the Œsophagus*; and, II. *Œsophageal Cancer*.

ŒSOPHAGITIS.

Œsophagitis, or inflammatory dysphagia, is a catarrhal inflammation of the mucous membrane of the whole or a part of the œsophagus. It is an exceedingly rare disease.

Morbid Anatomy.—In the acute variety the mucous membrane is red, swollen, softened and covered with a layer of mucus containing epithelium and pus. In the chronic variety the mucous surface is of a dull pink or slaty blue color. The sub-mucous tissue is thickened, and a thick viscid mucus or pus covers its surface. It may cause dilatation of the œsophagus, which may affect the whole tube uniformly or form a pouch at its lower portion, or it may give rise to a hernial protrusion of the mucous membrane through the muscular coat. In all cases of œsophageal dilatation due to chronic catarrh, there is more or less thickening of the œsophageal walls. In some cases the thickening may diminish the calibre of the tube. Ulceration of the mucous membrane at the seat of the catarrh sometimes occurs. The ulcer may be superficial, or extend through the walls of the tube.

Membranous Inflammation of the œsophagus may be either croupous or diphtheritic. In either case the morbid changes are the same as in croupous or diphtheritic inflammation of other mucous surfaces.

Etiology.—Acute œsophagitis has its most common cause in the irritation produced by acrid fluids or solids in their passage to the stomach. Irritating drugs and corrosive poisons may excite it. Too frequent introduction of instruments into the stomach may cause it, and it may arise from the excessive use of alcohol. Extension of inflammation from the parts above or below it often induces acute œsophagitis. Chronic œsophageal catarrh may occur as part of a similar process affecting the whole alimentary tract. It may develop as the result of a strumous or phthisical diathesis, or follow an acute attack. Membranous œsophagitis is always secondary and results from, or occurs with similar processes in the respiratory or other portions of the digestive tract. It also may appear after some of the eruptive fevers, cholera, pyæmia and septicæmia.

Symptoms.—Varying with the intensity of the inflammation, an aching or severe burning pain is felt at the back, between the shoulders, or deep behind the sternum. Even the ingestion of fluids causes dysphagia, the pain being greatest as the fluids pass through the upper portion of the œsophagus. More or less febrile excitement and great depression and anxiety accompany the disease, and throughout its course the thirst is tormenting. In severe cases there are paroxysms of coughing, and perhaps slight dyspnœa with hoarseness. Vomiting sometimes follows attempts at swallowing. All these symptoms gradually increase in severity. If extensive ulceration is present, sudden rupture of the œsophagus may occur during the act of deglutition. In chronic œsophagitis there is dysphagia and pain only on swallowing solids. If ulcers exist, there may be vomiting of viscid mucus tinged with blood, accompanied by the symptoms of œsophageal stricture.

Stricture of the œsophagus is accompanied by gradually increasing dysphagia, emaciation and debility, which finally terminate in death from inanition. The most frequent seat of stricture of the œsophagus is at its cardiac extremity. It may be caused by structural changes in its wall, as in œsophagitis with or without ulceration, and cancer, or by compression from mediastinal and other tumors. As œsophageal strictures develop slowly, for a long time the only symptom is slight difficulty in swallowing solids, the patient usually referring the difficulty to a point behind the *manubrium sterni.* As the constriction increases there is difficulty in swallowing liquids. Food and drink collect in the œsophagus, and after a longer or shorter delay are regurgitated with the saliva through the mouth and nose. With œsophageal stricture there are usually painful eructations. Sometimes the pain is lancinating in character, shooting from the region of the œsophagus back to the spinal column. The introduction of a bougie will determine the seat, extent, and form of the stricture.[1]

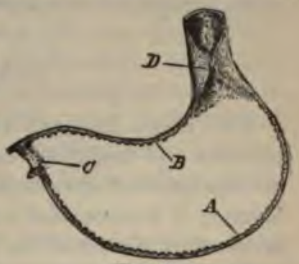

FIG. 53.

Diagram showing Stricture of the Œsophagus near cardiac extremity.

A. Greater curvature of Stomach.
B. Lesser curvature of Stomach.
C. Pylorus.
D. Point of the stricture.

Uniform dilatation cannot be recognized during life. When dilatation is partial, or when pouches exist, there may be vomiting of undigested offensive food some hours after eating. In all cases of alteration in the calibre of the œsophagus, the œsophageal bougie will determine the amount of narrowing and the locality of the pouches. Membranous inflammations of the œsophagus cannot readily be determined during life. A portion of membrane may be vomited, but it cannot be determined whether it comes from the œsophagus, or has been swallowed and regurgitated.

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[1] The treatment of stricture of the œsophagus belongs to surgery.

Differential Diagnosis.—This disease may be mistaken for *cancer* of the œsophagus; the diagnostic points will be considered in the history of œsophageal cancer. At the onset it may also be mistaken for *hydrophobia*, but the diagnosis is soon established by the development of the characteristic nervous phenomena of the latter disease.

Prognosis.—The immediate prognosis in acute œsophagitis caused by chemicals or mechanical irritants depends more on the changes which have occurred around the larynx and in the stomach, than upon the œsophagitis. The prognosis in croupous and diphtheritic inflammations of the œsophagus is also determined by the conditions of the primary disease. In chronic œsophageal catarrh without stricture the prognosis is good.

Treatment.—In acute œsophagitis, if the inflammation has been excited by foreign bodies lodged in the œsophagus, they must at once be removed; if corrosive chemicals have been swallowed, the proper antidote must be administered. In severe cases, all movement of the parts must be prevented. Ice in the mouth is grateful and does no harm. Nutrient enemata may be given, and, if the pain is severe, hypodermics of morphine must be given in sufficient quantities to afford relief. Hot anodyne fomentations applied locally are usually of service. In chronic œsophageal catarrh, if ulcers exist, spray the parts with astringent fluids, such as a solution of nitrate of silver. Surgery directs that if starvation seems imminent a gastric fistula should be made. In œsophageal stricture, bougies must be daily introduced for a long time, with the hope of dilating the stricture. No treatment is required in œsophageal dilatation.

CANCER OF THE ŒSOPHAGUS.

The most frequent variety of œsophageal cancer is epithelioma, but scirrhus and medullary cancer are not unknown. It occupies the upper and middle third of the œsophagus more often than the cardiac portion; in the former, it is associated with pharyngeal and laryngeal cancer, and in the latter with cancer of the cardiac extremity of the stomach.

Morbid Anatomy.—Epithelioma begins in the mucous tissue, and pursues the same course as cancer of the tongue. The ulceration may be limited to a circular patch an inch in diameter, or may involve the whole circumference of the œsophagus. The growth after a time invades all the tissues of the œsophagus, and causes stricture of its calibre. Above the stricture there is either uniform dilatation or a pouch, sometimes as large as an orange. If the cancerous mass involves the entire œsophageal wall, it may press upon and destroy one or both pneumogastrics, and lead to the development of pneumonia or pulmonary gangrene. If the œsophagus is ruptured, openings may be made into the trachea, through the diaphragm into the peritoneal cavity, or into the posterior mediastinum. Cancer has sometimes ulcerated into the aorta, pulmonary artery, and even into the right subclavian artery.

Etiology.—Two-thirds of the cases of cancer of the œsophagus occur in males between the ages of forty and sixty. It is generally primary; it may be secondary to cancer of the mouth, pharynx, mediastinum, or stomach.

Symptoms.—The first thing noticed in cancer of the œsophagus is difficult deglutition; soon well-marked dysphagia occurs. Pain is present early, and may be dull, burning, or lancinating in character; it is located about the centre of the sternum, in the throat, or in the interscapular space. It varies greatly in kind and degree, but it is greatly aggravated when food reaches that portion of the œsophagus which is the seat of the cancer. As the stricture grows narrower, flatulence, regurgitation of food and vomiting, with steadily increasing emaciation, become prominent symptoms, and a well-marked cachexia is developed. Cough, dyspnœa, and hoarseness sometimes result from pressure of the cancerous tumor. As the disease advances, the pain becomes more constant, the cachexia is better marked, and hemorrhages are frequent; the bloody fluid vomited often contains shreddy masses which contain cancer elements and show the character of the disease. The neighboring lymphatic glands may also be implicated, and, by pressure on a main bronchus, cause feeble respiration in the lung to which the bronchus is distributed. If rupture occurs, it is followed by a sudden sharp pain in the chest, fainting, and coldness of the extremities, followed almost immediately by death.

Differential Diagnosis.—Œsophageal cancer may be mistaken for *stricture of the œsophagus from chronic catarrhal inflammation.* In cancer, pain is constant and greatly aggravated by taking food, while in non-cancerous œsophageal stricture pain is absent, or is only present on swallowing. The glands about the neck are early involved in cancer, but are normal with chronic catarrh. Chronic pulmonary disease is rarely absent when œsophageal cancer is present, but is never induced by non-malignant stricture. Hemorrhage is frequent in cancer, and does not often occur with stricture from chronic catarrh. The bougie may bring up shreddy masses, with evidences of cancer in the one case, but merely meets with obstruction in the other.

Prognosis.—This is always a fatal disease. Its average duration is one year, but death may occur in a few weeks. The prognosis as to time varies with the presence or absence of complications. Death may result from any of the complications, from hemorrhage or septicæmia.

Treatment.—Early in cancer let the food be finely chopped and taken in a semi-fluid state; later, it should be *entirely* fluid, and when the patient cannot swallow, nutrient enemata must be given. The diet in all cases must be nourishing in the highest possible degree, and stimulants can usually be given with benefit. When the pain is intense morphia *per rectum* or hypodermically should be administered in doses sufficient to relieve it. Bougies should be used with great care; early, they should only be used to locate and diagnosticate the disease; later, tubes must only be used for the purpose of introducing food into the stomach, as fatal hemorrhage or rupture has followed their use. The subject of gastrotomy comes within the domain of surgery.

DISEASES OF THE STOMACH.

I. *Inflammations of its mucous membrane and its submucous or areolar tissue.*
  - a. *Acute or Toxic Gastritis.*
  - b. *Sub-acute Gastritis, or Acute Gastric Catarrh.*
  - c. *Chronic Gastritis, or Chronic Gastric Catarrh.*
  - d. *Phlegmonous Gastritis.*

II. *Dyspepsia.*
III. *Cancer and Ulcer.*
IV. *Neuroses or Gastralgia.*
V. *Hæmatemesis.*
VI. *Dilatation.*

ACUTE GASTRITIS.

Acute or *toxic* gastritis is a general inflammation of the mucous and submucous tissue of the stomach. It is of rare occurrence, unless the result of the introduction into the stomach of irritating poisons.

Morbid Anatomy.—On opening a stomach which is the seat of toxic gastritis a thick layer of tough, viscid mucus will be found spread over its mucous surface. Beneath this there will be found an intense redness of the membrane, which is most marked along the edge of the rugæ, near the cardiac orifice. The mucous and submucous tissues will be soft and œdematous. In some rare instances the whole or a portion of the mucous membrane will be found to present the appearance of a detached brown or black slough ; it may be entirely eroded. Fibro-plastic exudation may cause complete occlusion of its cardiac or pyloric orifices. When the muscular tissue is involved it becomes soft, easily torn, often gelatinous. In severe cases perforations may exist.

Etiology.—Acute gastritis is almost always caused by the entrance into the stomach of irritant poisons, such as sulphuric, nitric, and oxalic acids, arsenic, the chloride of zinc, and large quantities of concentrated alcohol. Mechanical irritation, such as results from the introduction into the stomach of knives, pins, false teeth, etc., may produce a *local* acute gastritis. Introduction into the stomach of boiling water, hot lead, or steam may cause a most intense gastric inflammation, with extensive sloughing of its mucous surface.

Symptoms.—Soon after the introduction of corrosive substances into the stomach, there will be a dull, uneasy feeling, sometimes one of warmth, over the epigastrium. This is rapidly followed by an intense burning pain shooting through to the back. The epigastric region becomes extremely tender. With, or preceding these symptoms, there is nausea, and vomiting of tenacious mucus ; the vomiting is very distressing ; the ejected mucus often contains blood, and, at first, portions or traces of the substance which has caused the gastritis. As the pain becomes more severe the vomiting is more distressing ; there is intense thirst, and frequent spasms of the abdominal muscles. The temperature rapidly rises, sometimes to 105° F.; the pulse reaches 120 or 140 per minute and is feeble and irregular. If the œsophagus is implicated, there is dysphagia. The urine becomes scanty and high colored, and is sometimes suppressed. These violent symptoms soon give place to a condition of general prostration, in which there is almost constant hiccough. The surface becomes cold and clammy, the radial pulse grows feeble and finally imperceptible, while the respirations are hurried, short and irregular, the mind remaining clear to the last. There are cases on record of poisoning by chloride of zinc and sulphuric acid, in which there was no pain in the epigastrium during the whole course of the disease.

Differential Diagnosis.—The diagnosis is not difficult. The history of its cause and the character of the vomiting establish it.

Prognosis.—The prognosis depends upon the cause. The more intense the pain, the more extensive the gastric inflammation. Death may occur in a few hours, or it may be delayed two or three weeks. Acute gastritis may be complicated by analogous conditions of the mouth, pharynx, or œsophagus, by enteritis, laryngitis, or œdema glottidis, and as sequelæ there may remain constrictions at the cardiac or pyloric orifices. Death may result directly from the shock of the gastritis, from the constitutional effects of the poison which produces it, or from resulting peritonitis.

Treatment.—The first thing is to administer the proper antidote to the poison which has caused the gastritis, and thoroughly wash out the stomach, after which the gastritis should be treated as a local inflammation. Four or five leeches may be applied over the epigastrium, followed by warm poultices or fomentations. Some prefer the application of ice to the epigastrium. Unless contra-indicated by the chemical constitution of the poison, the intense pain should be relieved by hypodermic injections of morphia. During the whole period the patient should be kept absolutely at *rest* in the horizontal position.

SUB-ACUTE GASTRITIS.

Sub-acute gastritis, or *acute gastric catarrh,* is always a secondary affection.

Morbid Anatomy.—The parts principally involved in this form of gastritis are the ridges between the depressions, the vessels which lie in immediate proximity to them, and the apertures of the tubules. The mucous membrane is mottled by red spots scattered over it in irregular patches; sometimes there are extensive ecchymoses and blood extravasations. The gastric juice is much diminished in quantity, and being mixed with much mucus loses its *acidity* and to a great extent its digestive power. The surface of the mucous membrane is covered with abundant, tenacious mucus ; there is also a moderate production of pus cells on the surface of the mucous membrane. The gastric tubules become filled with granular matter. Late in the disease the solitary and lenticular glands, especially about the pylorus, increase in size and stud the surface as small white specks. The inflammatory processes are superficial and do not involve the deeper tissue of the mucous membrane. Superficial sloughs are some-presents a well-marked granular appearance. The walls of the stomach are usually thickened and more or less indurated, especially about the pyloric orifice, which gives rise to more or less constriction, or "*pyloric stenosis.*" The thickened membrane is often "leathery" to the feel, and the induration may be so great that it tears with difficulty and can be stripped off the submucous tissue. The submucous tissue may also be thickened and congested, the color varying from an inflammatory blush to a livid, almost purple red. When the submucous tissue is involved, there is an infiltration of cells into it; and upon their organization into new connective-tissue, and the subsequent contraction of this tissue, there will be more or less interference with the peristaltic motion of the stomach. Besides this there will be hypertrophy and distention of the gastric tubules, for their secretion is retained by the tissue-increase in the intertubular structure, which will cause them to stand out as small granulations in the atrophied tissue, presenting an appearance denominated "*mammillation.*" This condition may also be the result of hypertrophy of the glandular layer, which thus becoming too large for the basement muscular layer is corrugated and gives rise to another form of *mammillation.* It is only in rare instances that there is any mammillation about the cardia.

In long-continued chronic catarrh of the stomach the muscular coat of the organ may become involved, and then the peristaltic movements will be still more impaired; finally, the peritoneum may become thickened and adhesions take place between it and the adjacent parts.

A *microscopic examination* of the gastric tubules in chronic gastritis will sometimes show that their epithelium has undergone granular degeneration, and in others there is a complete loss of epithelium, the tubules being filled with a granular detritus. Occasionally there will be found on the mucous membrane dirty white spots in irregular patches, which appear like depressions on the mucous surface. Under the microscope, there will be found in those spots some tubules completely filled with discrete fat spherules, and others whose epithelium has undergone fatty degeneration. If the tubules are constricted near their openings, cysts are formed from distention of the portion near the base by the secretion which cannot escape. In rare cases the fatty degeneration will involve the interstitial tissue as well. If hemorrhagic extravasation occurs into the gastric mucous membrane, the tubules will have their epithelium stained and their base blackened as a result of the sanguineous infiltration. Sometimes there is an increase in the intertubular lymphatic elements, with hyperplasia of the nuclei in the sheath of the vessels.

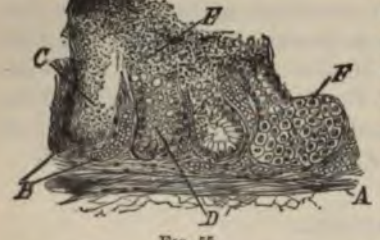

FIG. 55.

A Vertical Section of Mucous Membrane of the Stomach, showing changes in the Tubules in Chronic Gastritis.

A. Muscularis mucosæ.
B. Small blood-vessels around the follicles.
C. Tubule with granular epithelium and filled with granular detritus.
D. Another tubule, in which the epithelium is lost—filled with fat globules.
E. Granular matter filling the follicles.
F. A small cyst formed by closing of the mouth of the follicle, with proliferation of the epithelial wall. × 250.

In long-standing chronic gastritis there may be abrasions of the mucous surface and formation of ulcers (chiefly about the lesser curvature and the pylorus), circular in shape, varying in diameter from half an inch to an inch. These ulcers are very superficial, rarely extending beyond the mucous coat. They are pale in color, and their surface is covered with mucous cells, nuclei, and epithelium; between the ulcers the rugæ are congested. The intervening tissue is rarely normal. There may also be small follicular or punctate ulcerations, originating, it is supposed, in the enlarged solitary and lenticular glands. The base of these ulcers is infiltrated with lymph-cells and granular detritus; they are never present except in the advanced stage of chronic gastric catarrh. Chronic gastric catarrh may involve a large portion of the mucous surface of the stomach, and is generally associated with a like condition of the intestinal mucous membrane. *Waxy degeneration* may be associated with these morbid changes, but in such cases other organs, as the liver and spleen, will have been primarily affected by the amyloid infiltration. The size of the stomach varies: sometimes it is smaller than normal; at others it is dilated.

Etiology.—Chronic gastric catarrh is essentially a secondary affection; it is rarely the sequela of sub-acute, much less of acute, gastritis, unless the former has been caused by an abuse of alcoholic stimulants. In many persons there is an hereditary tendency, after middle life, to chronic gastric catarrh. The principal *general* cause of this affection is *anæmia*. The most common *local* cause is the daily use of alcoholic stimulants.

Mechanical obstruction to the capillary circulation of the stomach, inducing continued passive hyperæmia (congestion) will cause it, and hence we find it associated with cirrhosis of the liver and other chronic hepatic affections where the blood is dammed back in the formative branches of the vena portæ. In the same way, valvular and other cardiac lesions, and pulmonary diseases, such as emphysema, chronic bronchitis, and phthisis, which offer an obstacle to the venous return, will induce chronic gastric catarrh. Pressure on the walls of the stomach by tumors produces first congestion, and then chronic catarrh. Degeneration of the capillaries—"*arterio-capillary fibrosis*"—occurring in the cirrhotic form of Bright's disease, causes it, and it often accompanies ulcer and cancer of the stomach.

Those causes which may be denominated recent are rapid ingestion of food, improper quality of food, or food which is known "to disagree with the stomach," and the sudden arrest of the digestive process after hearty meals. The prolonged use of arsenic, mercury, cubebs, and purgatives often causes it. Finally, scrofula, syphilis, and gout seem to predispose to it, and I am inclined to regard the chronic gastritis which is so often found associated with these diseases as the result of some *degeneration of, or alteration in, the blood-vessels of the stomach.*

Symptoms.—The early symptoms of chronic gastric catarrh are chiefly those of *indigestion*. There is at first a sense of weight and fulness in the epigastrium, sometimes amounting to constriction, which comes on from half an hour to an hour after meals. Later there is actual pain and heat in the epigastrium ("heart-burn"). Pressure increases the pain and presents a well-marked granular appearance. The walls of the stomach are usually thickened and more or less indurated, especially about the pyloric orifice, which gives rise to more or less constriction, or "*pyloric stenosis.*" The thickened membrane is often "leathery" to the feel, and the induration may be so great that it tears with difficulty and can be stripped off the submucous tissue. The submucous tissue may also be thickened and congested, the color varying from an inflammatory blush to a livid, almost purple red. When the submucous tissue is involved, there is an infiltration of cells into it; and upon their organization into new connective-tissue, and the subsequent contraction of this tissue, there will be more or less interference with the peristaltic motion of the stomach. Besides this there will be hypertrophy and distention of the gastric tubules, for their secretion is retained by the tissue-increase in the intertubular structure, which will cause them to stand out as small granulations in the atrophied tissue, presenting an appearance denominated "*mammillation.*" This condition may also be the result of hypertrophy of the glandular layer, which thus becoming too large for the basement muscular layer is corrugated and gives rise to another form of *mammillation.* It is only in rare instances that there is any mammillation about the cardia.

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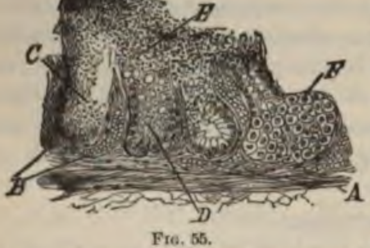

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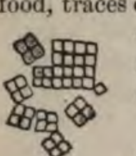


Fig. 56. *Sarcinæ Ventriculi.* × 750.

As the disease progresses, the feeling of malaise and uneasiness following meals changes to one of languor or exhaustion, and there is a sensation of heat in the epigastrium; *thirst* becomes a prominent symptom, one person craving cold, another hot drinks. The thirst is greatest in the evening, but the taking of fluids is usually followed by a sense of weight in the epigastrium, and by acidity and flatulence. The appearance of the tongue varies: it may be normal, paler than normal, florid and "beefy," or may be covered with a white or brown coating. The general symptoms which accompany the anæmic condition which attends this disease are headache, vertigo, cardiac palpitation, a gradual loss of strength and emaciation. Constipation and hemorrhoids are usually present, and the stools are often coated with mucus. In the chronic gastric catarrh of phthisis, diarrhœa is present. In cases of long standing, the hair becomes harsh and loses its lustre or turns gray; the skin is dry, sallow, and shrivelled, sometimes covered with an eczematous eruption; the nails are corrugated and exhibit a tendency to split, while in some there is premature caries of the teeth. Hypochondriasis, despondency, and irritability of temper are generally more or less marked. Hæmatemesis often occurs in that form of gastritis which accompanies cirrhosis of the liver, and the bleeding may for a time relieve the unpleasant gastric symptoms. Vomiting in the morning always accompanies the gastritis of Bright's disease.

The *urine* in chronic gastritis is cloudy, usually alkaline in reaction, depositing urates, phosphates and oxalates. Its specific gravity is highest at evening. The alkalinity is due either to imperfect gastric digestion or impaired function of the liver and pancreas. The greater the mental depression the more of earthy phosphates will be found in the urine.

If *hemorrhagic erosion* exist in a stomach which is the seat of chronic catarrh, the pain in the epigastric region is *constant*, frequently shooting back to the scapulæ. Vomiting occurs not only in the morning, on rising and after meals, but also in the intervals. The vomited matter contains traces of blood, and bile mixed with mucus; all of the gastric symptoms are augmented in *hemorrhagic erosion*. *Punctate* or *follicular ulcer-*

ation presents few, if any, symptoms differing from those of ordinary chronic catarrh. In most instances where a *post-mortem* has revealed this pathological state, there was vomiting of coffee-ground material during life.

Differential Diagnosis.—Chronic catarrh of the stomach is to be differentiated from *atonic dyspepsia*, from *cancer*, and *ulcer* of the stomach.

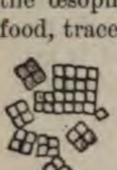
Atonic dyspepsia is associated with anæmic conditions dependent upon habits of life and an unhealthy occupation; while chronic catarrh is associated with the immoderate use of alcoholic stimulants, or is secondary to chronic thoracic, renal, or hepatic disease. In atonic dyspepsia there is little or no pain or tenderness in the epigastric region, which is always present in chronic gastritis. In atonic dyspepsia the tongue does not present the coated appearance so constant in chronic gastritis, but is broad, pale, and flabby. In atonic dyspepsia there is loss of appetite, but never the complete anorexia and constant thirst which are present in chronic gastritis. Spices and stimulating ingesta often relieve the gastric symptoms of atonic dyspepsia, while in chronic gastritis they aggravate the gastric symptoms. The constitutional symptoms in atonic dyspepsia are slight, while in chronic gastritis they are marked and severe. The urine is altered in atonic dyspepsia only during the attack; while it is cloudy and alkaline, and persistently deposits urates, oxalates, and phosphates in chronic gastritis. Nausea and vomiting are more apt to occur in chronic gastritis than in dyspepsia.

In its earlier stages cancer presents no symptoms other than those of a chronic gastritis; later the vomiting and other evidences of obstruction will be persistent, and the vomited matters will more certainly be coffee-ground in character. Dilatation of the stomach is more frequent with cancer. The recognition of an abdominal tumor alone can render the diagnosis certain.

The points in the differential diagnosis between *chronic gastritis* and *ulcer* of the stomach are given under the latter heading.

Prognosis.—The duration of chronic gastric catarrh is variable; it may last for months or years, and may terminate in ulcer or stenosis of the pyloric orifice. It is amenable to treatment except when associated with advanced hepatic, renal, or pulmonary diseases, or where stricture at the pyloric orifice exists. A not infrequent *complication* is disease of the suprarenal capsules, and the connection between the two diseases has by some been supposed to be a "sympathetic" one, but no rational explanation has yet been offered. Sub-acute gastric catarrh sometimes complicates chronic gastric catarrh and renders the prognosis unfavorable. Gastro-enteritis is a very rare complication. Death may result from hæmatemesis or from stricture of the pylorus. The general feebleness which results from long-standing gastritis predisposes to acute disease.

Treatment.—The most important thing to be accomplished in the treatment of chronic gastritis is the *removal of its cause.* Each case requires a special treatment suited to its special indications and to its complicating causes it to shoot backward and upward toward the scapulæ. Following, or with the advent of, these symptoms there is loss of appetite, first for solids such as meats; later there is complete anorexia. Nausea and eructations accompany the anorexia; the stomach, and often the intestines become distended with gas, but vomiting is not usually present unless pyloric stenosis exists. The most important of the dyspeptic symptoms are the *acid risings* after meals, and the vomiting or regurgitation of *acid mucus in the morning,* which may be regarded as characteristic, and without which the diagnosis is uncertain. It is this acid material belched up into the œsophagus that causes "heart-burn." If there is actual vomiting of food, traces of butyric acid are present, with the *sarcinæ ventriculi,* cuboid cells averaging 1-2500 inch in diameter, each being divided into four equal parts containing nuclei, usually heaped into large cubes.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Atonic dyspepsia is associated with anæmic conditions dependent upon habits of life and an unhealthy occupation; while chronic catarrh is associated with the immoderate use of alcoholic stimulants, or is secondary to chronic thoracic, renal, or hepatic disease. In atonic dyspepsia there is little or no pain or tenderness in the epigastric region, which is always present in chronic gastritis. In atonic dyspepsia the tongue does not present the coated appearance so constant in chronic gastritis, but is broad, pale, and flabby. In atonic dyspepsia there is loss of appetite, but never the complete anorexia and constant thirst which are present in chronic gastritis. Spices and stimulating ingesta often relieve the gastric symptoms of atonic dyspepsia, while in chronic gastritis they aggravate the gastric symptoms. The constitutional symptoms in atonic dyspepsia are slight, while in chronic gastritis they are marked and severe. The urine is altered in atonic dyspepsia only during the attack; while it is cloudy and alkaline, and persistently deposits urates, oxalates, and phosphates in chronic gastritis. Nausea and vomiting are more apt to occur in chronic gastritis than in dyspepsia.

In its earlier stages cancer presents no symptoms other than those of a chronic gastritis; later the vomiting and other evidences of obstruction will be persistent, and the vomited matters will more certainly be coffee-ground in character. Dilatation of the stomach is more frequent with cancer. The recognition of an abdominal tumor alone can render the diagnosis certain.

The points in the differential diagnosis between *chronic gastritis* and *ulcer* of the stomach are given under the latter heading.

Prognosis.—The duration of chronic gastric catarrh is variable; it may last for months or years, and may terminate in ulcer or stenosis of the pyloric orifice. It is amenable to treatment except when associated with advanced hepatic, renal, or pulmonary diseases, or where stricture at the pyloric orifice exists. A not infrequent *complication* is disease of the suprarenal capsules, and the connection between the two diseases has by some been supposed to be a "sympathetic" one, but no rational explanation has yet been offered. Sub-acute gastric catarrh sometimes complicates chronic gastric catarrh and renders the prognosis unfavorable. Gastro-enteritis is a very rare complication. Death may result from hæmatemesis or from stricture of the pylorus. The general feebleness which results from long-standing gastritis predisposes to acute disease.

Treatment.—The most important thing to be accomplished in the treatment of chronic gastritis is the *removal of its cause*. Each case requires a special treatment suited to its special indications and to its complicating causes it to shoot backward and upward toward the scapulæ. Following, or with the advent of, these symptoms there is loss of appetite, first for solids such as meats; later there is complete anorexia. Nausea and eructations accompany the anorexia; the stomach, and often the intestines become distended with gas, but vomiting is not usually present unless pyloric stenosis exists. The most important of the dyspeptic symptoms are the *acid risings* after meals, and the vomiting or regurgitation of *acid mucus in the morning*, which may be regarded as characteristic, and without which the diagnosis is uncertain. It is this acid material belched up into the œsophagus that causes "heart-burn." If there is actual vomiting of food, traces of butyric acid are present, with the *sarcinæ ventriculi*, cuboid cells averaging 1-2500 inch in diameter, each being divided into four equal parts containing nuclei, usually heaped into large cubes.

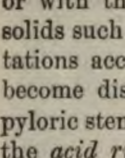
FIG. 56. *Sarcinæ Ventriculi.* × 750.

As the disease progresses, the feeling of malaise and uneasiness following meals changes to one of languor or exhaustion, and there is a sensation of heat in the epigastrium; *thirst* becomes a prominent symptom, one person craving cold, another hot drinks. The thirst is greatest in the evening, but the taking of fluids is usually followed by a sense of weight in the epigastrium, and by acidity and flatulence. The appearance of the tongue varies: it may be normal, paler than normal, florid and "beefy," or may be covered with a white or brown coating. The general symptoms which accompany the anæmic condition which attends this disease are headache, vertigo, cardiac palpitation, a gradual loss of strength and emaciation. Constipation and hemorrhoids are usually present, and the stools are often coated with mucus. In the chronic gastric catarrh of phthisis, diarrhœa is present. In cases of long standing, the hair becomes harsh and loses its lustre or turns gray; the skin is dry, sallow, and shrivelled, sometimes covered with an eczematous eruption; the nails are corrugated and exhibit a tendency to split, while in some there is premature caries of the teeth. Hypochondriasis, despondency, and irritability of temper are generally more or less marked. Hæmatemesis often occurs in that form of gastritis which accompanies cirrhosis of the liver, and the bleeding may for a time relieve the unpleasant gastric symptoms. Vomiting in the morning always accompanies the gastritis of Bright's disease.

The *urine* in chronic gastritis is cloudy, usually alkaline in reaction, depositing urates, phosphates and oxalates. Its specific gravity is highest at evening. The alkalinity is due either to imperfect gastric digestion or impaired function of the liver and pancreas. The greater the mental depression the more of earthy phosphates will be found in the urine.

If *hemorrhagic erosion* exist in a stomach which is the seat of chronic catarrh, the pain in the epigastric region is *constant*, frequently shooting back to the scapulæ. Vomiting occurs not only in the morning, on rising and after meals, but also in the intervals. The vomited matter contains traces of blood, and bile mixed with mucus; all of the gastric symptoms are augmented in *hemorrhagic erosion*. *Punctate* or *follicular ulcer-*

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Treatment.—The most important thing to be accomplished in the treatment of chronic gastritis is the *removal of its cause.* Each case requires a special treatment suited to its special indications and to its complicating causes. When alcohol is the cause, all stimulants must at once be prohibited, and the patient placed on a diet in which there are few fats or carbo-hydrates. The food should be taken slowly in small quantities, at shorter intervals than in health, and thoroughly masticated. I have found "underdone beef" and milk to be especially adapted to this class of cases. In catarrh induced by dram-drinking the best drug to allay morbid sensibility of the stomach and the morning sickness is opium, which also, by inducing sleep, relieves the nervous symptoms, which are always prominent. Strychnia and zinc in combination with mineral acids have a wide reputation in this class of cases, acting favorably on both the nervous and digestive disturbances. The vegetable bitters as tonics are often serviceable when the craving for alcohol is excessive.[1]

Thorough washing of the stomach with an alkaline fluid used by the syphon tube is of great service in severe cases. In milder conditions the patient may drink a large glass of very hot alkaline water half an hour before eating. Such treatment is often combined with an exclusive meat diet.

When there is marked anæmia, preparations of iron and pepsin may be given. When chronic catarrh is associated with cardiac disease, granules of digitaline, 1-50 of a grain each, may be given twice a day with advantage. When associated with pulmonary diseases, an out-of-door life in a suitable climate not infrequently effects a cure. In phthisical gastritis, a form that is very obstinate, hydrocyanic acid with the alkaline carbonates combined with bismuth is often of service. If hepatic disease exists, the portal congestion may be relieved by leeches about the anus and an occasional brisk mercurial purge; a course of mineral waters will in a large proportion of cases give temporary relief. The daily use of cold-water enemata will in these cases preclude the necessity of resorting to cathartics.

Scrofulous subjects should be treated with iodine and cod-liver oil. The Vichy waters in combination with colchicum are indicated in gouty patients. Free purgation and warm alkaline baths are also serviceable in this class of cases. There is, perhaps, no remedy which will for a time relieve the irritability, pain, and acidity after meals as certainly as bismuth. When it fails in cases of long standing, zinc, alum, tannin, or nitrate of silver may be tried. The habitual constipation which often complicates these cases will be relieved by daily use of aloes and strychnia, or rhubarb and soda. When there is evident deficiency of gastric juice, five or six drops of hydrochloric acid in a wine-glass of water, and ten or fifteen grains of saccharated pepsin, will greatly assist the digestive process. If there is an excess of gastric juice, alkaline waters should be freely used during, and after meals. When fermentation is very active, and flatulence is annoying, sulphite and salicylate of soda or creosote, given after meals, are serviceable. If the stomach rejects food as soon as it is taken, rest is essential, and the patient must be nourished for a time by the rectum and then placed on a milk diet. Minute doses of arsenic and of belladonna have been recommended as *curative* agents, but there is no evidence that they have any such power. Blisters, moxæ and issues over the stomach are sometimes of service in very chronic cases.

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[1] I have found the following to allay this craving:

℞ Tr. cinchonæ comp.................................℥ iv.
Tr. capsici.. ....ʒ ss.
Tr. nuc. vomicæ....................................ʒ ij.
M. A teaspoonful every two or three hours.

PHLEGMONOUS GASTRITIS.

Phlegmonous gastritis is a suppurative inflammation of the areolar (submucous) tissue of the stomach; it has also been called *"suppurative lenitis."*

Morbid Anatomy.—The suppurative process may be circumscribed or diffused. On removal of the stomach its wall is found thicker than normal, and its substance œdematous and very friable. The submucous tissue is distended by, and infiltrated with fibrin and pus, which not infrequently accumulate in large quantities in the muscular tissue as well. The entire mucous coat is, in rare instances, very much thinned and undermined by the purulent accumulation which perforates it at different points; the small openings thus formed give exit to the pus from the spongy, irregular shaped cavities, or *"abscesses,"* lying beneath. The mucous surface is reddened in patches, or is of a deep purple color; sometimes it is gangrenous. If the peritoneal coat is involved it presents the usual appearance of acute peritonitis. The abscesses in the sub-mucous tissue tend to open into the cavity of the stomach, although they may perforate externally and be discharged into the peritoneal cavity. In circumscribed phlegmonous gastritis these pus cavities *may* be the starting-point of ulcers of the stomach.

Etiology.—Phlegmonous gastritis is a very rare disease, usually occurring between the ages of twenty and forty years. It may occur idiopathically in previously healthy persons, without *any* assignable cause, or it may be secondary to pyæmia, septicæmia, puerperal fever, typhus fever, and diphtheria.

Symptoms.—Phlegmonous gastritis is ushered in by a distinct chill, followed or accompanied by intense pain and tenderness over the region of the stomach. Complete anorexia is an early symptom, and is accompanied by intense and constant thirst; there is persistent vomiting, which increases in severity with the advance of the disease; the ejected matters are sometimes purulent, but usually consist of a dark colored, bitter fluid. The pain increases in severity until it becomes as severe as in peritonitis. The temperature may reach 104° or 106° F. When the disease has reached its climax there is great depression and exhaustion; the patient is anxious and fretful, not infrequently passing into active delirium, but, whether the latter is present or not, typhoid symptoms with low muttering delirium, jaundice, stupor, and collapse are rapidly developed, and the patient passes into a state of coma and dies.

Differential Diagnosis.—The diagnosis of phlegmonous gastritis is only made by exclusion; it often passes unrecognized during life.

Prognosis.—The prognosis is always unfavorable. The majority die during the first week. When it is circumscribed its duration may be prolonged to two or three weeks. Its only complications are secondary abscesses in other organs (as the liver) and peritonitis. When primary, the disease reaches a fatal termination either from peritonitis or from exhaustion with typhoid symptoms.

Treatment.—When phlegmonous gastritis is secondary the primary disease will demand attention; in all cases the treatment is merely palliative; stimulants are indicated very early, and the sufferings of the patient must be relieved by morphia hypodermically.

GASTRIC DYSPEPSIA.

Dyspepsia and indigestion are terms used to indicate a train of symptoms caused by a functional derangement of the digestive processes. When these derangements are confined to the stomach they constitute *gastric dyspepsia.*

Morbid Anatomy.—Strictly speaking, gastric dyspepsia has no morbid anatomy. If it has continued for a long time the walls of the stomach may be found thinned, the mucous membrane atrophied, and many of the gastric tubules shrunken and in a state of fatty degeneration. Not infrequently the tubular structure of the stomach is replaced by a fibro-nucleated tissue. After death the power of self-digestion in such a stomach is markedly diminished or entirely lost. It is often met with as a part of *senile decay.*

Etiology.—Dyspepsia is often an inherited condition and accompanies the changes of advancing age. There is no affection in which individual idiosyncrasies are so strongly marked. Its etiology can best be considered under the following heads:—

First:—A class of cases in which there is a deficiency in the *quantity* of gastric juice secreted. Such deficiency often occurs in those disordered states of the blood which precede the onset of acute diseases. It occurs in enfeebled conditions, as the result of exhausting discharges, venereal excesses, masturbation, leucorrhœa and phthisis, and from the excessive use of narcotics, the tannin of tea, and the nicotine of tobacco.

Second:—There is a class of cases in which there is an *excess* in the gastric secretion. This is most apt to occur in those suffering with chronic hepatic and cerebral diseases and in gouty subjects. It is sometimes changed in quality and in quantity in young persons who have grown rapidly, and in females at the menopause.

Third:—There is a class of cases in which the gastric secretion is changed in *quality.* This occurs with ulcer and cancer of the stomach, gout, rheumatism, disease of the kidneys, uterus, and gall bladder. A lithic-acid diathesis is said to cause a change in the quality of the gastric juice.

Fourth :—There is a form of gastric dyspepsia due to impaired motion of the stomach, which may be the result of its adhesion to neighboring parts, to an omental hernia dragging it out of its normal position, to cicatrices and new growths at its pyloric extremity, to thickening of its walls, or to a weak, flabby, enfeebled condition of its muscular coat, and to pressure on the stomach from tight lacing and from positions assumed by shoemakers, needlewomen, writers, etc.

Fifth :—Mental emotion, prolonged mental labor, and anxiety rather than continuous and regular brain work, cause dyspepsia; in such cases it is the *sudden* arrest of the digestive functions, especially after eating too much, which is the main etiological factor. Organic cerebral disease and pressure on, or disease of one or both pneumogastrics act in the same way.

Sixth :—Deficient or excessive physical labor may be a cause of dyspepsia. Walking immediately after a full meal is a prolific cause of this variety, examples of which are frequently met with in *letter-carriers.*

Seventh :—Improper diet is a common cause of dyspepsia. It may arise from an excess of starchy materials, as potatoes; or from deficiency of meats. Under improper diet may be included decomposing food, impure water, badly cooked food, too rapid eating, the food not being sufficiently masticated, or taken at too short intervals and irregularly. Articles of food that may be suited to one climate, season, or age may in another be wholly indigestible and cause dyspepsia.

Symptoms.—The symptoms of dyspepsia are a series of phenomena which vary not only in different individuals, but in the same individual at different times; the most constant is an abnormal appetite: it may be lost, increased, or perverted. There is a weight, dull pain, and a sense of burning in the epigastrium after ingestion of food, accompanied by flatulence, heartburn, gastralgia, constipation or diarrhœa, a dull headache, languor, depression of spirits and irritability of temper. Indiscretion in eating or drinking, and exercise or exposure in dyspeptic subjects are apt to bring on an attack of sick headache. There is frequently a bitter taste in the mouth, bilious vomiting and sluggish bowels; this is called a *bilious attack.* If these symptoms immediately follow the taking of food, it is called "ingestive dyspepsia," or "morbid sensibility of the stomach." In some dyspeptics the breath and fæces have a very offensive odor.

Pyrosis, which is the chief symptom in another class of cases, is the regurgitation into the mouth of a large amount of thin, watery, saline fluid, preceded by a sense of constriction and pain in the epigastrium. This fluid consists mainly of saliva. Sometimes there is not only a feeling of oppression in the thorax, but a severe pain is referred to the heart, accompanied by palpitation and dyspnœa. In such cases the patient is very apt to imagine he has heart-disease.

Accompanying some cases there is *vertigo,* ringing in the ears, spots before the eyes, and other sensations which together have been called "stomachic vertigo." These patients hear a buzzing sound and feel as if a vapor were enveloping them; they grow pale, and grasp for support through fear of falling. When in any case the "indigestion" has lasted a long time, chronic gastric catarrh will almost always be developed, and evidences of mal-nutrition show themselves by anæmia, premature old age, corrugation of the nails, caries of the teeth, etc. At other times the patient will suffer from dyspnœa, with a short, dry cough, and occasional paroxysms of an asthmatic character. The skin becomes sallow, dry, and rough, while various eruptions appear on it, and the abnormal contents of the urine show that the functions of the kidneys are disturbed. Often in long-standing dyspepsia in females there will be a feeble pulse, leucorrhœa, and irregularities in the menstrual functions. There is no characteristic change in the appearance of the tongue; in one case it is white and heavily coated, in another it is clean, large, and indented. The *urine* often contains oxalate of lime ("oxaluria"). After the oxalates disappear, lithiates may appear for a time, soon to be followed by normal urine.

Differential Diagnosis.—The phenomena of dyspepsia closely resemble those of *chronic gastric catarrh.* Repeated attacks of dyspepsia are apt to result in a sub-acute or chronic gastric catarrh. Their differential diagnosis has already been considered. *Acidity from hypersecretion* may be confounded with *acidity from fermentation,* and *stomachal* may be confounded with *cerebral vertigo.*

The following are the principal points in their differential diagnosis: pain in *acidity* from *hypersecretion* either immediately follows the taking of food, and is accompanied by "heartburn," or, quite as often, it is felt most when the stomach is empty, and is *relieved* by taking food; but the pain from *fermentation,* due to obstruction to movements of, or to chronic inflammatory processes in, the stomach, comes on some time after eating, and is more a sense of weight or fulness in the epigastrium than pain. It is never present during the intervals between taking food. Vomiting is rare in acidity from fermentation, but if it does occur, the ejected materials will contain organic acids, torulæ, and sarcinæ; while with hypersecretion vomiting is a common symptom, and very frequently there is an excess of hydrochloric acid in the matter vomited. The constitutional symptoms, mental depression and emaciation, the sallow skin, etc., are much more marked in dyspepsia with fermentation than in dyspepsia with hypersecretion. In case of acid stomach from fermentation, flatulence is very common, while it rarely occurs with acidity from hypersecretion. The urine is alkaline or neutral in acidity from fermentation, while it is always acid with hypersecretion. Lastly, acidity from fermentation has a history of some cause or causes which interfere with digestion, while hypersecretion is usually a reflex symptom, or occurs with ulcer of the stomach, or in perfectly healthy persons.

With *vertigo* or dizziness from *stomachal* causes, there is a history of indigestion, and it usually occurs in middle life, while in *cerebral vertigo* the individual is beyond middle life, and there will be no history of difficult or impaired digestion. Vertigo from stomachal causes occurs during an attack of indigestion, or after some particular *kind* of food has been taken. Cerebral vertigo occurs wholly independent of the state of the stomach. Consciousness is never lost, nor are the special senses—sight alone excepted—involved in stomachal vertigo, while ringing in the ears, temporary deafness, and often complete loss of consciousness occur in an attack of cerebral vertigo. A person suffering with stomach vertigo *knows* that the apparent motion of the surrounding objects is unreal—while a patient with cerebral vertigo believes the apparent movement of the objects to be *real.*

Prognosis.—The prognosis varies with the etiology. Dyspepsia in most cases *can be cured,* but the cure depends for the most part on the will of the patient. The only danger is that the conditions induced by dyspepsia may predispose to organic diseases in other organs, as the lungs or kidneys, and that it may lead to a condition of melancholia.

Treatment.—First, if possible, remove the cause. When the gastric juice is *deficient in quantity,* hydrochloric acid and pepsin are indicated. In these cases, also, the vegetable bitters are especially beneficial ; indeed, in most cases of dyspepsia they are valuable adjuvants to the other remedies. Tea and tobacco are always to be avoided ; alcoholic stimulants in moderate quantities may sometimes be combined with the vegetable bitters with advantage. When acid risings occur after ingestion of food, and are due to active *fermentation,* the sulphite of soda or salicylic acid immediately after meals may prevent such changes. When fermentation is present, these remedies, with alkalies, will relieve the heartburn and other gastric distress. A course of saline waters will be found, in such instances, to aid the other remedies.

When there is great *irritability* of the stomach, bismuth acts almost as a specific, and should be given in twenty-grain doses before eating. Creosote, codeia, oxalate of cerium, and morphia may be employed to arrest vomiting. If there is pain in the epigastrium, the local application of heat by means of the hot-water bag will relieve. Dyspeptics should never wear corsets or belts about the abdomen ; they should retire and rise early, and eat slowly, masticating their food thoroughly. The meals should be small and taken at stated intervals. The diet must be determined for each case. Most patients do best on a diet free from hydrocarbons, but many cases of a lithæmic diathesis must be deprived of meats. No mental or physical work should be performed directly after or before eating. Horseback-riding and walking in the open air should be insisted upon. A change of scene and climate works rapid cures in many instances. Dyspeptics should take plenty of rest, have their sleeping-rooms well ventilated, and take a cold sponge-bath morning and evening. The general principles of treatment in gastric dyspepsia are similar to those given in chronic gastric catarrh.

CANCER OF THE STOMACH.

The stomach, next to the liver, is the most frequent seat of internal cancerous developments ; one third of all the cases of primary cancer have their seat in the stomach. The varieties of cancer of the stomach, in the order of their frequency, are as follows :

First, *scirrhus ;* second, *medullary ;* third, *epithelial.* Any one may undergo colloid degeneration, and thus may appear as either villous or melanotic cancer.

Morbid Anatomy.—Cancer has its seat at the *pyloric* extremity of the stomach in about three fifths of the cases. The next favorite seat is the *cardia* and the lesser curvature. When it is developed at the pylorus, it sometimes extends an inch or two into the duodenum; cancer at the cardia usually involves the lower part of the æsophagus.

Scirrhus of the stomach first appears as a small, grayish-white, opaque nodule in the submucous tissue, the normal structures of which are enclosed by the new growth. These nodules are developed from off-shoots of the gastric tubules which have pushed their way through the mucous membrane into the loose submucous tissue. The primary change is thus epithelial in character.[1] The fibrous stroma is far in excess of the cell-element; it develops rapidly at the exterior of the mass, causing induration and contraction of the surrounding structures. The mass sometimes extends inward toward the cavity of the stomach, causing flattened tumors which project into it. The contraction of these nodules puckers the *mucous surface*, which becomes immovably fixed upon them, and fibrous lines radiating from the growth penetrate the mucous membrane, which first undergoes a slight increase in thickness, and then becomes pale from compression of its vessels. The solitary glands are enlarged, and the gastric tubules are matted together in an indistinguishable bundle. A dark slough sometimes forms upon its surface and exposes the cancerous growth, which then ulcerates. The ulceration may extend so deeply as to destroy the new growth and invade the wall of the stomach underneath it, causing irregular cavities, bounded by a raised and indurated band of connective-tissue, and sometimes opening into the stomach. These polypoid tumors are sometimes as large as a hen's egg and develop *upon* the cancerous mass. The glands and villi are the longest to resist this encroachment of the cancerous development, the first change in them being an increase in the number of their epithelial cells. After a time the *muscular coat* becomes fused with the areolar,

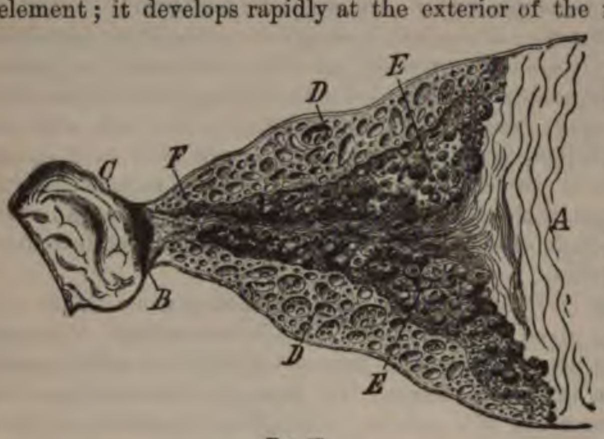

Fig. 57.

Cancer of the Pyloric Extremity of the Stomach.

A. Mucous membrane of the stomach beyond the seat of the cancerous infiltration.
B. Pylorus.
C. Commencement of the Duodenum.
D, D. Vertical section of the cancerous mass.
E, E. Internal surface of the cancerous infiltration encroaching on the pyloric orifice.
F. Small opening in the cancerous growth at the pyloric extremity.

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[1] Waldeyer.

so that at the seat of the neoplasm they cannot be distinguished from each other on section of the mass. At other times the parts affected are hard and fibrous, the stomach walls being so thickened that the disease is only differentiated from hypertrophy of its coats, by the glistening, pearly look and cartilaginous texture of the mass. After involving the muscular coat, the growth may involve the peritoneal covering; local peritonitis establishes adhesions between it and the diaphragm, liver, pancreas, and spleen.

The lesions which follow the development of scirrhus in the stomach are as follows:

Dilatation of the stomach is a frequent result of the obstruction at the pylorus caused by the cancerous development. Less common than dilatation of the stomach is the *gizzard* appearance caused by the same contraction that shrivelled the mucous membrane, inducing a shrinking of the whole stomach wall, which sometimes becomes an inch thick, the cardia and pylorus not infrequently being closely approximated, and the anterior and posterior stomach walls being almost in juxtaposition.

Chronic gastritis is developed when the new growth attains sufficient size to cause pressure, and in such cases the mucous membrane presents the characteristic appearances of that affection.

Perforation of the stomach sometimes occurs, causing a fatal peritonitis; a secondary opening may penetrate into the duodenum, liver, jejunum, or ileum, or through the anterior wall of the abdomen, and thus form an external opening. In rare instances openings are made into the pleural cavity, lungs, bronchi, or pericardium. Large branches of the pneumogastric may be destroyed by the new growths. In five per cent. of the cases of cancer of the stomach, secondary cancer is developed in other organs. The organ which is the most frequent seat of this secondary development is the *liver*, and after the liver the lymphatic glands in the immediate vicinity of the peritoneum, and various segments of the intestine, especially the rectum. The kidneys, bladder, spleen, pancreas, and ovaries may also be the seat of these secondary developments. The position of the stomach is sometimes *changed*, the weight of the tumor dragging it into the lower portion of the abdomen, and there it may be bound to the intestines, bladder, uterus or ovaries by firm adhesions.

Medullary or *acute* cancer of the stomach commences in the same tissues as scirrhus, in the form of nodules much softer than those of scirrhus.

On section, cancer-juice can be readily expressed from the cut surface of the cancerous mass; the proportion of the stroma being much less, and the cells more abundant. The growth is more vascular, and not infrequently contains small blood extravasations. It is much more rapid in its development than scirrhus, and while proliferation of the epithelial structure occurs at the periphery, fatty degeneration breaks down the centre, and it sometimes becomes diffluent. The mucous tissue is rapidly invaded. Large, spongy, "fleshy" excrescences project into the cavity of the stomach. Around the growth, which varies in size from that of a pigeon's egg to that of an orange, is a ring of tissue infiltrated with cancer, beyond which the solitary glands are enlarged, and the stomach follicles degenerated. "Villous" cancer of the stomach is a modification or variety of medullary cancer. If medullary cancer ulcerates, the slough is thrown off, and an excavated ulcer is exposed, surrounded by an elevated rim, from which projects the cauliflower-like growth, very friable and vascular. The surface exposed by such ulceration is often very large, even six or seven inches in diameter. Epithelial cancer is rare in the stomach. When present, it presents the same characteristics as when developed on other mucous surfaces.

Colloid or *alveolar* cancer has the same structure as colloid cancer occurring in other parts of the body. It appears oftener in the stomach than elsewhere, but *even here* it is rare. There are different opinions as to its starting-point; some state that it begins in the subserous, others in the submucous tissue. Recently a glandular origin has been ascribed to it, similar to epithelioma of the skin. Wherever commencing, it rarely appears as nodules, but commonly as an irregular mass of "gum-like" glistening material, contained in large and distinctly marked alveoli, in which are embedded polygonal nucleated cells. The walls of the stomach, the seat of colloid degeneration, are very much thicker than normal. Its tendency is to spread rapidly over a large surface. The contents of some of the alveoli are discharged into the stomach, thus giving to its inner surface an irregular, "honey-comb" appearance.

Etiology.—Cancer of the stomach occurs most frequently between the ages of forty-five and sixty-five. It is more frequent in males than females.

Hereditary predisposition is undoubtedly its most important etiological factor. Beyond this its etiology is obscure.

Symptoms.—The earliest and most prominent symptom of cancer of the stomach is anorexia, accompanied by a sense of uneasiness or distention in the epigastrium, with nausea and vomiting. Pyrosis is often present quite early. Patients describe the pain as dull, gnawing, or as a tightness or "soreness" over the stomach; after a time it becomes lancinating, fixed, and constant; the *locality* of the pain does not correspond to the seat of the cancer; when the growth has its seat in the lesser curvature, the pain is referred to the interscapular region; it is not usually increased by ingestion of food, and if it is, it does not cease at the end of the digestive process; it may become constant and severe. These symptoms usually exist *before the appearance of a tumor.* During the period of its growth, vomiting becomes frequent. There are three prominent causes of the vomiting: *first,* from *obstruction.* Vomiting from this cause comes on comparatively late; when the obstruction is at the cardia it occurs immediately after eating. If it is situated at or about the pylorus, the food is retained for one or two hours. *Secondly,* from *irritation.* This occurs independent of taking food and the time of its digestion. *Thirdly,* from *fermentation.* This occurs after a large accumulation of food in the stomach; and the matters vomited are dark and yeasty, not infrequently containing *sarcinæ ventriculi.* When free hydrochloric acid is persistently present in the gastric contents, cancerous obstruction may be quite certainly excluded. When this acid is persistently absent, the probabilities are in favor of cancerous disease being present; but no absolute deductions can be made.

Hiccough, flatulence, and constipation are often very annoying, sometimes very distressing symptoms; and emaciation, debility, and the haggard "cancer countenance," are often present. There is mental depression, anxiety, and the patient is morose or apathetic.

When ulceration of the free surface of the cancerous mass occurs, the most constant symptom is *hemorrhage*. This *may* be copious and bright red in color, but usually the blood is so altered by the gastric juice, and so mixed with food, that it is rusty, brown, or blackish in color ("*coffee-ground*" vomit). In the later and larger hemorrhages, the blood may in part escape by the bowels, and then diarrhœa occurs, caused by the decomposing blood; the stools have a dark tarry appearance, with a very offensive odor ("melæna"). The yellowish green color of the skin, usually present, may change to a jaundiced hue, due to pressure of the cancerous mass upon the bile ducts.

One may be deceived or puzzled, during the course of cancer of the stomach, by a remission of the anorexia, pain, hemorrhage, and vomiting, so that improvement seems to be taking place, and the patient believes he is recovering; but in a short time all these symptoms will return with increased severity, and the disease will progress more rapidly than before.

Again, there is sometimes a febrile reaction—not a definite hectic, but a symptomatic fever—which appears irregularly during the progress of the cancer, and often misleads on account of the belief that the temperature in malignant disease is normal or sub-normal. During the advanced stage in many cases the tongue becomes covered with aphthæ, typhoid symptoms develop, and death is often preceded by delirium and coma. The *urine* is scanty, high colored, and of a high specific gravity. It is loaded with urates, and deposits a *pink sediment* regarded by some as a diagnostic symptom.

Physical Signs.—By *palpation* a tumor may be discovered—sometimes large, hard, irregular, and nodulated; sometimes small, deep-seated, and elastic. In the former case it is easy, in the latter very difficult of recognition. If the cancer is situated at the cardiac extremity of the stomach, no tumor will be felt. The tumor is usually movable, except when adhesions have formed between it and the adjacent tissues. If the cancer is at the pyloric extremity of the stomach, the tumor is usually situated in the median line; it may, however, be felt at the lower part of the epigastric region, in the right hypochondrium, at or just above the line of the umbilicus, or it may be far over on the left side. It may receive and transmit the impulse of the aorta, that is, become a *pulsating tumor*. The epigastric region is prominent, hard, resisting, and tender. It is important, during the examination, to have the patient distend his stomach by drinking one or two tumblers of carbonated water.

Percussion over the tumor elicits circumscribed dulness with a tympanitic or a peculiar hollow quality; light percussion may give absolute flatness, when forcible percussion gives a tympanitic resonance.

Auscultation gives negative results.

Differential Diagnosis.—Cancer of the stomach may be mistaken for *gastric ulcer*, *abdominal aneurism*, *cancer of the pancreas*, *cancer* of the *left* *lobe* of the *liver*, and for *chronic gastric catarrh with hæmatemesis ;* the two latter are considered under those heads. It is hardly possible, after a careful study of a case, to mistake cancer of the stomach for *gastric dyspepsia*, or to confound a cancerous tumor at the pylorus with an *ovarian tumor*.

Ulcer of the stomach occurs most in young adults, especially females, while *cancer* is seldom met with in persons under forty. In cancer there is usually a history of hereditary cancer; while ulcer of the stomach is usually associated with anæmia, chlorosis, prolonged lactation, or prolonged compression of the stomach, as in the case of shoemakers and sewing-girls. The pain in cancer is continuous, and described as lancinating; while in ulcer the pain is intermittent, greatly increased by taking food, often referred to the lower dorsal vertebræ, and described as "gnawing" or burning. *Hæmatemesis*, in cancer, has a sooty or "*coffee-ground*" appearance, is small in amount, and appears late in the disease; while in ulcer it is bright red arterial blood, is profuse, and appears as an early symptom. Vomiting in cancer does not relieve the pain, is not very severe, and comes on late; but in ulcer it is severe, comes on early, and affords temporary relief from the pain. The cancerous cachexia and debility are present early and steadily progress in cancer; while in ulcer there may be pallor, but no characteristic cachexia. The presence of an epigastric *tumor* establishes the diagnosis of cancer.

An *aneurismal* tumor is smooth and ovoid; a *cancerous* tumor is hard and irregular. An expansive, dilating impulse is given to the hand on palpating an aneurismal tumor; but in cancer this impulse is lifting in character. In aneurism there is constant pain in the back corresponding to the position of the tumor, which is absent in cancer. There is a change in the femoral pulse in aneurism, which is not present in cancer. The gastric symptoms, the cachexia, and the debility of cancer are not present in aneurism.

In cancer of the pancreas, vomiting is less frequent, and when present is not coffee-ground, and the vomited matters contain free hydrochloric acid. There is no dilatation of the stomach, constipation is less severe, and the passages often contain fat. Jaundice is more frequent from implication of, or pressure on, the hepatic duct.

The tumor in pancreatic cancer is always high up and fixed; in gastric cancer it may be low in the abdomen or freely movable.

Glycosuria is occasionally present with pancreatic cancer, and not with gastric cancer.

Prognosis.—The prognosis in cancer is always unfavorable. Its shortest duration is seven weeks, and its longest three and one-half years, the average duration being one year. Early vomiting, with hæmatemesis, great and sudden emaciation, and complete anorexia, are especially unfavorable symptoms. The important *complications* of cancer of the stomach are the development of secondary cancer in other organs, peritonitis,—independent of or with perforation,—pleurisy and pneumonia, pericarditis, endocarditis and fatty heart, tuberculosis, coagula in the sinuses of the dura mater, phlegmasia dolens, non-cancerous ulcerations in the rectum and colon, ascites, and general anasarca. Death may occur from hemorrhage, peritonitis, exhaustion, marasmus, and from complications.

Treatment.—The treatment is altogether *palliative*. The indications are to make the patient comfortable by relieving pain and vomiting. The diet may be determined by the experience of each patient. In the majority of cases alcoholic stimulants in moderation are beneficial. When the pain becomes severe, morphia may be administered hypodermically. If at any time the stomach becomes overloaded, its contents may be carefully removed by means of a stomach pump. The constipation, which is often obstinate, is best overcome by aloes; the flatulence and painful eructations by sulphite of soda or oil of cajeput. During the whole course of cancer, subnitrate of bismuth may be administered, its combination with soda, conium, and stramonium being highly recommended by English physicians. Some assert that arsenic is effective in retarding the cancerous growth, and that its administration with iodine and carbolic acid may arrest its development. My experience does not confirm this statement. If the stomach entirely rejects food, rectal alimentation may be resorted to.

ULCER OF THE STOMACH.

Statistics show that gastric ulcers, or cicatrices caused by ulcers, are found in three out of every hundred cases of diseases of the stomach. They may be classed as follows:

I. *Superficial Ulcer*, or *Hemorrhagic Erosion*.
II. *Follicular Ulcer*.
III. The *Chronic, Round*, or *Perforating Ulcer*.
IV. The *Typhus Ulcer*.
V. The *Typhoid Ulcer*.
VI. The *Variolous Ulcer*.
VII. The *Diphtheritic Ulcer*.
VIII. The *Syphilitic Ulcer*.
IX. The *Tubercular Ulcer*.
X. The *Cancerous Ulcer*.

The first two have already been considered.

The specific ulcers which receive their names from the diseases in which they occur as occasional pathological lesions, will be considered in connection with the history of those diseases.

The chronic, round, perforating ulcer is by far the most frequent form of gastric ulcer, and is the one indicated when the unqualified term, *ulcer of the stomach*, is used.

Morbid Anatomy.—Chronic perforating ulcers most frequently occupy the posterior wall of the stomach near its pyloric extremity. They vary in size from half an inch to two or three inches in diameter; an ulcer one-half inch in diameter may exhibit all the clinical characteristics of one of large size. These ulcers are at first circular or elliptical in form; occasionally they become irregular when two or more are fused together. When oblong they have their axis either parallel with, or transverse to the axis of the stomach; sometimes they form a zone about the pyloric end of the stomach. The large ulcers are formed by the fusion of several small ones. They begin in the mucous membrane of the stomach; their boundary is nearly vertical, smooth and sharp, so that now and then at a post-mortem the mucous membrane will present an appearance as if a circular piece had been "punched out" with a sharp instrument. There is no evidence of an inflammatory process. The loss of substance may involve only the mucous layer, or it may extend to the submucous tissue, or penetrate deeper and involve the muscular and peritoneal coats; as it extends, smaller and less regular circles are formed, gradually diminishing in diameter, a small opening in the muscular coat, or a mere point upon the peritoneum, being the apex of the conical or "funnel-shaped" excavation. As the ulcer spreads transversely in the course of the vessels, this "step-like," bevelled appearance becomes more and more marked. The tissues around the ulcer are sometimes normal, especially when the mucous membrane alone is involved; at other times the mucous layer encircling the base of the ulcer is thickened and indurated. The mucous membrane in the vicinity of an ulcer is sometimes the seat of a circumscribed chronic catarrh; but more often *chronic catarrh* involves the whole gastric mucous membrane.

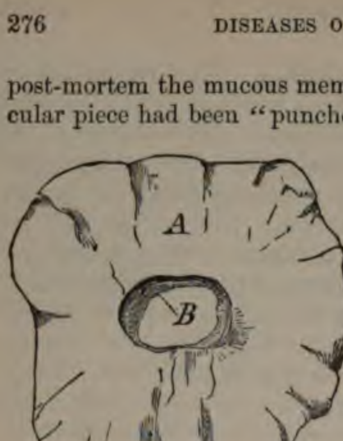

FIG. 58.

Perforating Ulcer of the Stomach.

A. Mucous surface.
B. Perforation, with clean cut edges, entirely through the gastric walls.

The *variations* from these usual pathological appearances consist, ***first***, in a mass of black blood adhering to the base of the ulcer; *secondly*, in petechial extravasations around the injected margin of the ulcer; *thirdly*, in the villous or "polypoid" vegetations springing up from the mucous membrane surrounding the base of the ulcer; and *fourthly*, in suppuration in the coats of the stomach with subsequent suppurative pylephlebitis. The progressive increase in the depth of the ulcer, which is part of its natural history, would always lead to perforation and discharge of the contents of the stomach into the peritoneal cavity, were it not for the establishment of a *local peritonitis* which causes the corresponding portion of the stomach to become adherent to the adjacent parts. These adhesions may join it to the pancreas, liver, mesentery or spleen.

The number of ulcers which may be found in a stomach varies from one to six; as a rule there is but one. Gastric ulcers, if not large or deep, may heal without producing deformity of the stomach. If they are large or deep, the resulting cicatrix, by its contraction, causes deformity of the stomach. When the mucous and submucous tissues are alone involved, the loss of substance is replaced by new connective-tissue, which does not contract; the resulting cicatrix is merely a white spot, with little or no puckering. The usual process of repair in *deep* ulcers is that of a local inflammation with lymph exudation. The connective-tissue formed at the base and around the ulcer contracts, and there remains a central, hard mass from which radiate bands of connective-tissue into the adjacent mucous membrane. The contraction of this cicatricial tissue *may* cause a stricture at the pylorus, or, if the ulcer extends around the central portion of the stomach, may give it an "hour-glass" shape. When there is stenosis at the pyloric orifice, the stomach is dilated and the walls are thickened in one subject and thinned in another.

Ulcers may extend by degeneration of the newly-formed tissue. With the extension of the ulcers, some of the larger vessels (as the superior pyloric) may become involved, and extensive hemorrhage result; usually, a "protective thrombosis" prevents this accident. Hemorrhages, the result of intense passive hyperæmia, or of erosion of small vessels, are of little consequence compared with those which result from the opening of vessels of large size or of the organs with which the stomach becomes adherent. In this way the portal vein, and the splenic, pancreatic and hepatic arteries have been pierced. Perforation of the stomach in gastric ulcer occurs only in about one-eighth of all the cases. Though the posterior surface of the stomach is the more frequent seat of these ulcers, the *liability* to perforation is greatest when the ulcers are situated in its anterior wall. If perforation and escape of the contents of the stomach take place into the peritoneal cavity a general, rapidly fatal peritonitis immediately follows; when adhesions prevent the contents of the stomach from escaping into the peritoneal cavity, a local peritonitis is developed and an abscess may be formed in the neighborhood of the ulcer, which abscess may communicate with the pleural cavity, duodenum, colon, or gall-bladder.[1]

Etiology.—Ulcer of the stomach occurs in females oftener than in males, the proportion being more than two to one. The liability to it is greatest between the ages of fourteen and thirty, although no age is exempt; it has been found in the new-born babe and in the octogenarian. Anæmia and chlorosis are the two great predisposing causes. Chronic and phlegmonous gastritis, cirrhosis of the liver, and obstruction of the gall-ducts may lead to ulcer of the stomach by inducing obstruction in the vessels of the walls of the stomach. Ulcer *may* result from an habitual stooping position, as in milliners, seamstresses, and shoemakers, or may come from the constant striking of the shuttle of the weaver against the epigastrium. *Miliary aneurisms* in the gastric walls may cause gastric ulcers. *Such ulcers are most frequently met with in connection with a cirrhotic kidney.* It may occur without any recognized cause.

Symptoms.—The symptoms of gastric ulcer are sometimes obscure, at others well marked.

Pain is one of its constant symptoms: at first it is dull and heavy; then it becomes burning and gnawing, causing a sickening sensation quite distinct from nausea. It usually comes on soon after the ingestion of food, and lasts during the entire period of stomach digestion; occasionally it is not present until an hour or so after eating. It is circumscribed to a spot rarely larger than a silver dollar, is accompanied by tenderness on *deep* pressure, and its intensity is usually greatest just above the umbilicus. The "dorsal" pain of gastric ulcer was first recognized by Cruveilhier. It comes on some weeks or months after the pain in the epigastrium; it is located at the eighth or ninth dorsal vertebra, and is constant, although it may sometimes alternate with the epigastric pain. In a few cases the pain is paroxysmal; there are intervals of freedom from pain, followed by severe attacks, resembling those of neuralgia. Relief from the pain of gastric ulcer is frequently obtained by a recumbent posture; this happens when the ulcer has its seat on the anterior wall of the stomach.

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[1] Many theories have been advanced in regard to the pathogenesis of gastric ulcers; the following are the principal ones: *perforating* ulcers may be the result of an inflammatory process, a sequel, oftentimes, of chronic gastritis. Rokitansky attributes them to congestion, extravasation, and subsequent necrosis of the tissue. Virchow maintains that embolism or a venous stasis deprives a portion of the stomach of its vascular supply, and that the stomach-tissue thus deprived of its nutrition, is acted upon by the gastric juice as dead tissue; as a result, there is a loss of substance and the formation of ulcers. He compares the funnel or cone-shaped appearance of the ulcer to embolic infarctions elsewhere, the capillaries always ramifying outwards from the main trunk.

Nausea, vomiting or regurgitation of food may accompany the pain; in some instances there is pyrosis, or "water brash;" usually the vomiting occurs when the pain is most severe. The matter vomited consists, first, of the food taken into the stomach, which has a strong acid reaction; later it is mingled with bile. The vomiting temporarily relieves the pain. After a time these dyspeptic symptoms are complicated by hæmatemesis, which may be regarded as essential to its diagnosis. In a few cases there is no vomiting. Some will vomit several times in the twenty-four hours, others once a day, and others every two or three days. As small bleedings do not cause vomiting, and as attention is rarely paid to the stools at this period, the exact date of the first hemorrhage is usually unknown. The symptoms which attend the hæmatemesis are a sense of unusual fulness in the stomach, accompanied by a feeling of faintness; the face is blanched, nausea exists for a varying period, and this is followed by vomiting of partially coagulated blood, which is so bright as to leave no doubt of its arterial origin. In rare instances the *first* hemorrhage causes distention of the stomach, syncope and painless collapse, followed by death. Sometimes the blood vomited has a dark, grumous appearance, looking like coffee-grounds. In young females the hemorrhage is usually preceded by a diminution or stoppage of the menses.

Preceding and accompanying the hæmatemesis there are usually dyspeptic symptoms similar to those of gastric dyspepsia. Cachexia is a late symptom, the appetite is rarely impaired, sometimes it is even increased; great debility and extreme anæmia result from the recurring hemorrhages. The face assumes an earthy pallor; when the pain is intense it is "drawn" and haggard, which by some is regarded as characteristic of ulcer of the stomach. Perforation of the stomach is attended by the symptoms of rapidly developed and extensive peritonitis. Pain in the right shoulder is a prominent symptom if an ulcer of the stomach involves the under surface of the liver. If cicatrization of a gastric ulcer takes place without adhesions or stricture all the above symptoms remit and complete recovery follows; if adhesions or stricture remain dyspepsia and cardialgia may continue for the remainder of the patient's life. Obstinate constipation is the rule in ulcer of the stomach, but hemorrhage may cause diarrhœa. The blood gives to the dejections a dark color and a "tarry" consistence, a condition called "melæna." The only *physical sign* of gastric ulcer is extreme tenderness on firm pressure over the epigastric and dorsal regions.

Differential Diagnosis.—The diagnosis in a typical case of ulcer of the stomach is not difficult; in the more obscure cases it may be mistaken for *cancer* of the *stomach, hepatic colic,* the second stage of *cirrhosis, cardialgia,* and *chronic gastric catarrh* with *hæmatemesis.* The diagnosis of cancer of the stomach, hepatic colic, and the second stage of cirrhosis are considered under these headings (q. v.).

In *neuroses* causing cardialgia, there will be a history of neuralgia in other parts of the body or a well-marked history of hysteria. The pain of cardialgia is not excited or increased by the introduction of food into the stomach, but often comes on when the stomach is empty; while in ulcer the pain is associated with ingestion of food. In cardialgia pressure over the epigastrium and the ingestion of food relieve the pain; the reverse is the case in ulcer. Again, cardialgia is relieved by the constant current and Faradization, which increase rather than relieve the pain of gastric ulcer. Dyspeptic and gastric disturbances are constantly present in ulcer; while these are absent in the intervals between the paroxysms of neurotic cardialgia. Hæmatemesis never occurs in cardialgia.

In *chronic gastritis* with *hemorrhage* there is the history of disease of the liver, heart, lungs or kidneys; while in gastric ulcer there is usually no such history. The pain in gastritis is not so intense or of the same character as in ulcer of the stomach. A coated tongue, great thirst, malaise, and pyrexia are prominent in cases of chronic gastritis, and absent in ulcer. The vomiting in chronic gastritis comes on in the morning, and the matter vomited is stringy mucus, sometimes streaked with blood; while in ulcer the attacks of vomiting usually follow the taking of fluids or solids, and the blood is vomited in considerable quantities.

Prognosis.—More than one half of the cases of ulcer of the stomach recover. The average duration cannot be determined. Some terminate fatally in a few weeks, others continue for a long period. In the protracted cases, the symptoms are probably due to the existence of more than one ulcer. Most of the cases that recover continue for more than a year. The prognosis is bad where ulcer occurs in the aged and in feeble women.

The *complications* of gastric ulcer are chlorosis and hysteria; thoracic complications, such as pneumonia, bronchitis, pulmonary tubercle, acute pleurisy, and empyema; abdominal, such as suppurative pylephlebitis and peritonitis. Death occurs from hemorrhage four times as often in the male as in the female. Exhaustion, either from pain or from vomiting or from starvation, causes death in 5 per cent. of the cases. Perforation, with peritonitis or without it, occurs in about 13 per cent. of all the cases. The liability to perforation is greatest in the female between the ages of fifteen and thirty; while in the male the liability is greatest about the fortieth year. Cicatrization of an ulcer at the pyloric extremity of the stomach is usually followed by dilatation of the stomach. When the cicatrix surrounds the pylorus and the obstruction is extreme, persistent vomiting and all the evidences of inanition may simulate cancerous obstruction.

Treatment.—The most important thing to be accomplished in the treatment of gastric ulcer is to give *rest to the stomach.* To this end the patient must be kept *in bed,* and the diet restricted to peptonized milk. From a tablespoonful to a teacupful may be given at intervals of two hours during the day and night. This makes it possible to keep the gastric contents persistently alkaline, and thus avoid the possibly destructive action of excessively acid gastric juice. When milk is refused, digested beef-juice may be given in its stead ; all vegetables, tea, coffee, starchy food and fruits must be prohibited. If all kinds of food are rejected, rectal alimentation must be practised, four ounces of defibrinized blood (containing four grains of chloral to prevent its decomposition) may be thrown into the rectum every six hours.

The remedial agents which have been found most useful in gastric ulcers are the sub-nitrate of bismuth, in twenty-grain doses, four times a day, sulphite of soda, oxalate of cerium, and hydrocyanic acid. Half a grain of nitrate of silver, three times a day, seems to exert a beneficial effect on gastric ulcers of long standing, as well as on the accompanying gastric catarrh. Several observers claim that arsenic retards the spread of gastric ulcers ; my experience does not confirm this observation. If the pain is severe, and there is no hemorrhage, warm poultices may be applied to the epigastrium ; but morphia hypodermically is far more reliable for the relief of pain, and may be used at regular intervals with benefit. Hypodermic injections of ergotin, and ice-bags to the epigastrium, will usually check the hemorrhages. The flatulence, which is often very distressing, may be mitigated by sulphite of soda, carbolic acid, or the alkalies. Constipation when present is relieved by ox-gall enemata, or saline mineral waters, the latter being especially useful when there are acid eructations. When the patient will bear it, castor-oil is a safe and efficient laxative. Stimulants must never be given by the stomach. They may be given by enemata in emergencies.

Rest in bed and a restricted diet should be enforced for at least three months. Then, if the symptoms indicate that cicatrization is well established, maccaroni, potatoes, stale bread and cocoa may be allowed ; still later oysters, soft eggs, and sago. The patient must not return to an ordinary mixed diet for at least six months. The anæmia which follows gastric ulcer must be overcome by fresh air, moderate exercise, iron, and quinine. It must be remembered that the higher the nutrition is carried, the more rapid and complete will be the repair of the ulcer. This end must be had constantly in view in the management of these cases. The establishment of nitric acid issues, and the employment of *moxæ* over the abdomen or epigastrium, as recommended by some, are of doubtful service, either for the relief of pain, or to hasten the healing of the ulcer.

NEUROSES OF THE STOMACH.

These are comparatively rare independent of a well-marked neuralgic diathesis. They are known as nervous gastralgia, or cardialgia, and as *erythism of the stomach*. They have no distinct pathological lesions.

Etiology.—Neuroses of the stomach are met with most frequently in females, and occur especially in those with an hereditary neurotic predisposition. Exhaustion, anæmia, and chlorosis, especially when accompanied by depressing influences, as grief, fear, anxiety, or great intellectual effort, play a most important part in its etiology. In the same way exhaustion from hemorrhage, insufficient food, venereal excess and masturbation will induce it. Central nervous diseases and disease of the pneumogastrics or sympathetic will sometimes cause it. The excessive use of tea or coffee has been cited as a cause, but those cases where some particular article of diet, as milk, brings on attacks of pain in the stomach, are not true neuroses. Reflex irritation caused by painful affections of the teeth, ear, kidneys, testicles, ovaries and disturbances in the alimentary canal, as hemorrhoids, constipation, worms and hernia, has been regarded as a cause of gastralgia. Diseases and displacement of the uterus, accompanied by disturbance of its functions, will very frequently give rise to attacks of cardialgia.

Hysteria and hypochondriasis are its two most frequent causes; statistics show that of 360 cases of these two diseases only 30 were free from gastralgia. Malarial gastralgia is accompanied by other forms of malarial neuralgiæ.

Symptoms.—Gastralgia usually begins with a sense of distention and tightness in the epigastrium, followed by a severe and agonizing pain, which will be described differently by different patients. In many instances the pain shoots through to the back. During an attack the abdomen is sometimes distended and rigid, sometimes flattened and retracted. The pain is often so severe that the heart's action becomes irregular, the extremities cold, the face pinched, and there is a tendency to syncope; in rare instances convulsions occur. The pain is relieved by firm pressure over the epigastrium, by the constant current, and by Faradization; the duration of these attacks is not at all regular, sometimes lasting only a few minutes, at other times an hour or two, generally terminating with gaseous eructations, or the ejection of an acid or an alkaline fluid. Sometimes before the first attack there will be complete anorexia. Vomiting, preceded by nausea, *may* be a part of an attack, and, though very severe, it does not depress the patient. Between the attacks, which occur at intervals of days or weeks, regularly or irregularly, the digestive functions are normal.

Differential Diagnosis.—Neurotic cardialgia may be mistaken for *ulcer* of the stomach, which has already been considered.

Prognosis.—This depends upon the cause; cardialgia may continue for years and not endanger or shorten life.

Treatment.—In the treatment of this affection, tonics are always indicated. Iron, arsenic, nitrate of silver, and oxide of zinc may be tried alternately. For relief when pain is intense, morphia may be given hypodermatically. Great care must be exercised not to repeat the hypodermatic too frequently, for this class of patients readily become addicted to the use of opium. Obstinate and protracted cases sometimes yield quickly to the constant current or to Faradization.

HÆMATEMESIS.

Hæmatemesis, or *blood vomiting*, is a symptom in a variety of diseases; it varies in amount and frequency with the morbid conditions which induce it. Rupture of a blood-vessel is one of its essential conditions.

Etiology.—I. *Injury* to the mucous tissue of the stomach by traumatism or poisons.

II. *Diseases* of the wall of the stomach, associated with diseased conditions of the blood-vessels.

III. *Obstruction* to the portal circulation, as in cirrhosis, acute yellow atrophy, aneurism of the hepatic artery and tumors compressing the vena portæ; gastric hemorrhage may remotely be produced by obstruction in the portal tissue, the result of cardiac and pulmonary diseases ("nutmeg liver").

IV. *Blood poisoning* may cause hæmatemesis, as scurvy, yellow, typhus, and relapsing fevers, snake-bites, and cholera. It also occurs in patients with the "hemorrhagic diathesis," and in "bleeders," or those affected with *hæmophila.*

V. *Cancer and ulcer* of the stomach cause it.

VI. *Passive* hyperæmia, stoppage of menses in the female, and the sudden arrest of hemorrhoidal discharges, are supposed to cause hæmatemesis by suddenly raising the blood-pressure in the portal system.

VII. Finally, this symptom appears nearly three times as often in females as in males, and usually between the ages of twenty and forty years.

Symptoms.—In hæmatemesis, if the hemorrhage is profuse, the patient experiences a sense of heat and distention in the epigastrium, with nausea and vomiting; he becomes pallid, and the surface cold and clammy, as the blood rushes up in a full stream through the mouth and nose, or is thrown up by successive acts of vomiting. When the blood comes up with a sudden gush, some portion of it may enter the larynx and excite coughing, and then it may appear to be coughed up. The appearance of the blood differs according to the length of time it has been acted upon by the gastric juice. If it is vomited in large quantities immediately after the bleeding has occurred, it will be partly fluid and partly coagulated; but if it has been retained in the stomach for a considerable time, it will be fluid and have a black, or brownish-black appearance, with an acid reaction.

Differential Diagnosis.—Hæmatemesis may be confounded with *hæmoptysis* or "blood-spitting."

Hæmoptysis is preceded by bronchial or pulmonary symptoms, and hæmatemesis by gastric symptoms. Hæmoptysis is preceded or accompanied by a sense of constriction across the chest, by dyspnœa and cough; while before hæmatemesis, there is nausea, with oppression and distention felt in the epigastrium. If cough is associated, it follows the expulsion of blood. Blood is coughed up in mouthfuls, bright red, frothy, alkaline and mingled with sputa in hæmoptysis; while it is vomited more or less profusely, is dark colored, mixed with food, coagulated, and often acid in hæmatemesis. In hæmoptysis there is a sense of "trickling" behind the sternum, and for a few days after the hemorrhage, small blood-spittings; and a physical examination of the chest readily determines the origin of the hemorrhage and establishes a diagnosis.

Prognosis.—Hæmatemesis, though a grave symptom, does not often directly cause death; the prognosis is determined by the diseased conditions with which it occurs. Hæmatemesis from cirrhosis of the liver or ulcers is always more dangerous than from any other conditions.

Treatment.—During the hemorrhage the patient must be kept absolutely quiet, in a horizontal position. Ice should be taken freely, and ice-bags applied to the epigastrium. Morphine and ergotin may be hypodermically administered, and the patient sustained by rectal alimentation. Astringents given by the stomach usually do more harm than good, and should not be employed. In severe cases the head must be kept low and brandy may be given by the rectum. If there is evidence of heart-failure, brandy and digitaline may be given hypodermically. After the hemorrhage ceases great care must be exercised in the diet of the patient; milk is the only nutritive article that should be allowed for the first week. The conditions which cause the hemorrhage must receive their appropriate treatment.

DILATATION OF THE STOMACH.

Morbid Anatomy.—The amount of dilatation is very variable; in one instance the stomach was found capable of containing ninety pounds of fluid; it may be either uniformly dilated, or there may be dilated, circumscribed pouches corresponding to ulceration or erosion of its walls. When stenosis at the pylorus exists, the walls of the stomach are first hypertrophied, and then a muscular paralysis is followed by atrophy, thinning of its walls, and dilatation of its cavity, which is usually to the left and upwards. The muscular coat may be so stretched and thinned as sometimes to be scarcely traceable; this is more frequently the case when the dilatation is independent of stenosis. In some few instances fatty degeneration of the muscular coat has been found, and with it the rugæ of the mucous coat have disappeared, and the mucous membrane has become pale and thin.

Etiology.—Dilatation results *first* from pyloric stenosis, and this may be caused by cancerous or non-malignant ulceration, by the effects of corrosive poisons (acute gastritis), by thickening from chronic and phlegmonous gastritis, and from fibroid indurations of the pylorus. Whether spasm of the pylorus is sufficient to cause dilatation or not, is as yet undecided. *Secondly*, dilatation of the stomach may be caused by *obstruction* of the pylorus by tumors external to the stomach and duodenum, as cancer of the liver, pancreas, gall-bladder, and lymphatics of the lesser omentum. *Thirdly*, paralysis and consequent impaired peristalsis will cause dilatation of the stomach. It may be the result of parenchymatous degeneration occurring in fevers and severe constitutional diseases. Suppurations about the stomach, as empyema and purulent pericarditis, may induce dilatation by diminishing its nerve supply. *Fourthly*, habitual over-distention of the stomach results from drinking inordinately and eating immoderately, especially of food which will ferment, and, by production of gases, will still more distend an overloaded stomach. *Fifthly*, hernia, by dragging down the stomach, and fibrous bands binding it to other organs, may lead to dilatation.

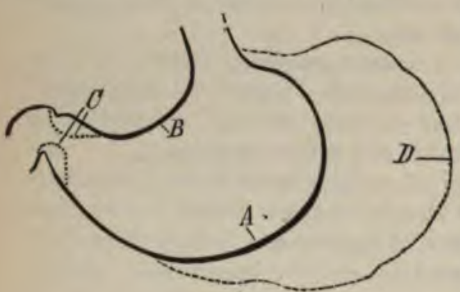

FIG. 59.

Diagram Illustrating Dilatation of the Stomach.

A. Greater curvature.
B. Lesser curvature.
C. Pylorus, the dotted line indicates the point of stenosis.
D. Line showing the usual direction of the dilatation, to the left and upward.

Symptoms.—Dilatation of the stomach may be acute or chronic. Acute dilatation usually occurs after exhausting diseases, though in some instances there is no discoverable cause. It begins with sharp pain in the epigastric region, with tympanitic distension and sometimes with tenderness on pressure. These symptoms rapidly subside, and the subsequent dilatation is revealed by a physical examination of the abdomen.

In *chronic* or *slow dilatation*, vomiting is the first important symptom. This sometimes occurs only after eating, but usually every two or three days there is ejected the accumulation of a portion of the previous day's food: the quantity vomited varies in amount from one to three gallons, and has a fetid odor, a black, "yeasty" appearance, and an intensely acid reaction. On a *microscopic examination* of the matter vomited, sarcinæ and torulæ are found in abundance. The eructations are offensive, consisting of sulphuretted and phosphuretted hydrogen, the results of the fermentation and putrefaction accompanying this condition; pyrosis is a very annoying and often painful symptom. There is progressive emaciation, with pain, and a sense of weight and distension in the epigastrium. Muscular *cramp* in the calves of the legs is often a painful attendant. The pulse is compressible: the appetite remains good, often amounting to "*bulimia.*" In some cases where there is complete paresis, there is no vomiting, but rapid emaciation, and anorexia from the commencement. The *bowels* are constipated, and the fæcal discharges hard and dry.

Physical Signs.—*Inspection* reveals a prominent rounding just above the umbilical region. In some cases there is a peculiar depression just at the epigastrium. When the patient takes an effervescing draught, the stomach is visibly enlarged and the epigastrium becomes prominent.

Palpation shows slight resistance at or below the epigastrium, the walls of the stomach being tense and elastic; sometimes the motions of the stomach and a prominence at the pylorus can be detected.

Percussion.—If the stomach is empty, percussion reveals an increased tympanitic area, by which the position and shape of the stomach can be quite accurately mapped out; or, on filling the stomach with fluid, an abnormal area of dulness indicates the enlarged stomach area.

On *auscultation,* a splash or "*succussion sound*" is heard on shaking the patient; and when fluid is swallowed, it can be heard dropping into the enlarged cavity.

Differential Diagnosis.—This condition may be mistaken for *ascites, distended urinary bladder,* or for *hydatids* of the *liver.* In *ascites* the fluid distends the lower and not the upper portion of the abdomen; the area of the abdomen broadens and flattens when the patient assumes the supine posture, and there is fluctuation on palpation.

An *hydatid tumor* does not change its shape or position when fluids are taken into the stomach; it is fixed in its position and is not accompanied by gastric symptoms.

The special diagnosis is of the cause. It is important to determine if this be pyloric obstruction, and the nature of such obstruction. In all cases careful examination should be made for external tumors causing pressure, or the presence of any pyloric growth. The two important ones are cancer and a cicatricial growth following gastric ulcer. The diagnosis by the history has been given; when the tumors have developed, cancer is more irregular, distinct in outline, and tender on pressure. With cicatricial contraction the vomited matters will contain free hydrochloric acid and be free from blood. With the history these points are usually sufficient for a diagnosis.

Prognosis.—The prognosis is determined by the conditions which cause the dilatation. Most cases are incurable. Surgical measures may afford relief in cases of cicatricial obstruction.

Treatment.—The most important thing to be remembered in the treatment of this affection is to adapt the diet to the condition of the patient; food must be taken in small quantities; as small a quantity of fluid as possible should be allowed. When the stomach is overloaded, its contents may be withdrawn by the stomach-pump, and thoroughly washed out with warm water. The daily use of the siphon in washing out the viscus in these cases has not proved to be as useful a therapeutic measure as was claimed for it when it was first employed. To overcome the paresis of the muscular coat, strychnine has been very extensively employed. Benefit may be obtained in some cases by the use of galvanism. To prevent fermentation, the sulphites or salicylic acid may be used. All saccharine and starchy foods must be abstained from, and a diet suited to each case must be persisted in. Resection of the pylorus for cancer has not given such results as to justify the operation. Loreta's operation of digital divulsion has been followed by quite invariably favorable results. It should be employed in all cases of undoubted cicatricial nature, and in many doubtful cases an exploratory laparotomy may be justified.

DISEASES OF THE INTESTINES.

I shall consider Intestinal Diseases under the following heads :—

I. *Enteritis or Intestinal Catarrh.*
II. *Diarrhœa.*
III. *Cholera Morbus.*
IV. *Cholera Infantum.*
V. *Dysentery.*
VI. *Typhlitis and Perityphlitis.*
VII. *Intestinal Ulcers.*
VIII. *Intestinal Hemorrhage.*
IX. *Intestinal Obstruction.*
X. *Waxy Degeneration of the Intestine.*
XI. *Cancer of the Intestine.*
XII. *Rectitis or Proctitis.*
XIII. *Intestinal Parasites.*
XIV. *Intestinal Colic.*
XV. *Constipation.*

ENTERITIS OR INTESTINAL CATARRH.

Enteritis is a general term applied to a catarrhal inflammation of the intestinal mucous membrane. It may be acute or chronic, circumscribed or diffused. The name "muco-enteritis" has been applied to it, when the mucous coat of the intestine only is involved ; when the inflammation involves the muscular and peritoneal coats, it is termed "phlegmonous enteritis." When situated in the *colon,* it has been called "inflammatory diarrhœa."

Morbid Anatomy.—At its onset acute enteritis is characterized by congestion, tumefaction and dryness of the mucous surface of the intestines ; this is soon followed by an abundant secretion of mucous and pus, which covers the inflamed surface. Peyer's patches and the solitary glands are congested and swollen, and stand out over the inflamed surface, causing it to present an appearance as if it were sprinkled with sand. After a time a thin serous fluid is copiously exuded into the intestinal canal. The layer of mucus and pus which covers the mucous membrane is either loosely attached or firmly adherent ; in the latter case it resembles in appearance a diphtheritic exudation ; when portions of the muco-purulent layer are removed, the mucous surface underneath is found eroded. This is the severest form of muco-enteritis.

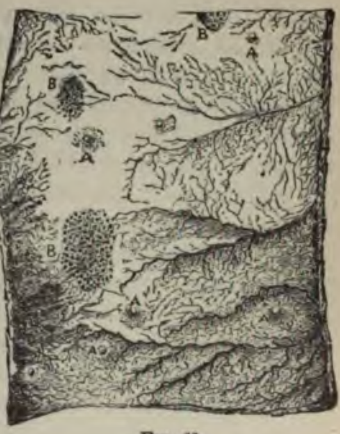

FIG. 60.

Acute Enteritis. Small Intestine, near middle of Ileum, showing hyperæmia.

A, A. Enlarged solitary glands.
B, B. Congested Peyer's patches, presenting the "shaven beard" appearance.

Simple or erythematous intestinal catarrh begins in the small intestine, and may rapidly spread over the entire alimentary tract. The membranous variety is usually confined to the *large intestine* and *rectum,* the portion near the ileo-cœcal valve being most extensively implicated. In severe cases the mucous membrane is often dotted with ecchymotic spots. These changes in the intestinal mucous membrane are not infrequently accompanied by changes in its lymphatic structures. The lymph follicles become greatly enlarged, and are surrounded by an inflammatory areola. Their contents soften, undergo necrosis, and a small, round, funnel-shaped ulcer is formed, called a "follicular ulcer." This ulcerative process may extend beyond the follicles and involve the submucous and muscular tissues. A dull pink flocculent substance, of a "porridge-like" consistence, is often found in the intestine at the seat of numerous follicular ulcers. In nearly every case of acute intestinal catarrh, the *mesenteric glands* are congested.

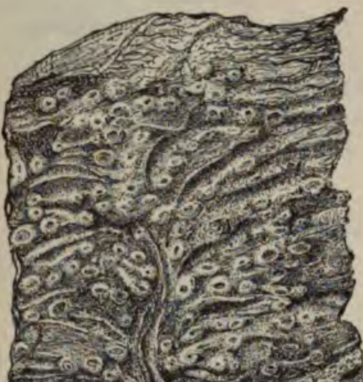

Fig. 61.

Acute Follicular Enteritis. Transverse Colon laid open, showing enlarged follicles, which have ulcerated at their apices.

In *phlegmonous enteritis* all the coats of the intestine are involved. It is usually limited to a small portion of the intestine, varying in length from two or three inches to one or two feet. The affected portion is excessively dilated; the mucous and submucous tissues corresponding to its seat are thickened, softened and congested, and are either of a dark color from blood extravasations, or pale from purulent infiltration. The muscular coat is thick, soft and œdematous. Upon the peritoneal coat are irregular patches of submucous extravasations, and the free peritoneal surface is covered with a thin plastic exudation. The intestine below the seat of the phlegmonous inflammation is contracted, empty, and its mucous membrane healthy, while above its seat the intestine is dilated and filled with fæcal matter, its mucous membrane remaining normal; sometimes at the seat of the inflammation numerous *ulcers* appear, extending in rings transversely about the intestine. It may be confined to the cæcum and ascending colon.

In *chronic intestinal catarrh* the mucous membrane has a slaty, blue or gray color; sometimes black pigment deposits are found in the villi, and between the follicles. The membrane is thicker than normal, and, as a result, the peristaltic motion of the intestine is impeded. Hyperplasia of the connective-tissue immediately beneath the epithelium, and perhaps of the solitary and agminated glands, occurs. The epithelium itself, in most cases, undergoes fatty or granular degeneration. The lymphatics are enlarged, and project from the mucous surface, as numerous distinct white nodules, covered with viscid gray mucus, which is sometimes purulent. The veins underneath the mucous membrane are enlarged and tortuous. If acute intestinal catarrh passes into *chronic*, the intestinal coats become thinned and pale, or hypertrophied, causing stricture of the intestinal canal; such stenosis occurs most frequently at the sigmoid flexure and in the rectum.

Follicular ulcerations occur more frequently in chronic than in acute intestinal catarrh. In prolonged cases, there may be developed polypoid cysts; similar changes in the stomach may accompany intestinal catarrh. Acute and chronic catarrh of the duodenum are attended by changes similar to those which take place in other portions of the tract; but in duodenal catarrh, the secondary catarrh of the ductus communis, by obstructing its opening into the duodenum, leads to catarrh of the hepatic ducts.

Etiology.—Intestinal catarrh is ordinarily caused by direct irritation of the mucous membrane by improper or decomposing food, impure water, or irritating medicines, or by exposure to wet or cold. Extensive burns will also cause it. Certain atmospheric conditions produce it in children during dentition, or during convalescence from one of the exanthemata.

Chronic intestinal catarrh is often a complication of chronic malarial infection and chronic Bright's disease. It may also be an accompaniment of hernia. Obstructed venous return, from hepatic, cardiac or pulmonary disease, as cirrhosis of the liver, chronic valvular lesions, and pulmonary emphysema, is a predisposing cause. It occurs at all ages; one-third of the diseases of children have intestinal catarrh as their primary or principal lesion. Its two great predisposing causes in children are dentition and bad hygiene, especially during the hot months, when there are the greatest variations of temperature. It may be epidemic or endemic.

The membranous and phlegmonous varieties occur as complications of the exanthemata, pyæmia, septicæmia, puerperal fever, and, in rare instances, acute tuberculosis, and acute Bright's disease.

Symptoms.—As the symptoms of acute enteritis vary with the portion of intestine involved, as well as with its severity, the symptoms will be considered without attempting a history of its course.

Diarrhœa is its earliest and most constant symptom. It is called mucous, bilious, or serous, according to the varying character of the discharges. In mucous diarrhœa watery mucus is mixed with ordinary fæces. In cases where the stools consist almost exclusively of mucus, rectitis and "colitis" may be suspected. In colitis, cylindrical casts of varying lengths and pus are often present in the discharges. In the so-called bilious diarrhœa, pain and cramps in the calves of the legs are not infrequent. There is vomiting, headache, sallowness of the surface, furred tongue, weight and fulness in the right hypochondrium, and more or less prostration. The color of the stools is more distinctly green than in any other variety. Serous diarrhœa is the most common, and when the unqualified word diarrhœa is employed this form is indicated. At first the dejections contain undigested food; after twenty-four or forty-eight hours the fæcal odor of the discharges is lost. At the onset of duodenitis, jejunitis, or ileitis, diarrhœa may be absent if the large intestine is not simultaneously involved. Diarrhœa may occur independent of intestinal catarrh.

Pain is another symptom which, although *not always* present, is so constant that its absence is the exception to the rule; sometimes it is colicky and griping in character, at others it is severe and paroxysmal, or dull and unremitting. In all cases it is rendered more severe by the ingestion of food. With the pain, there is a sense of fulness and distention of the abdomen, and tenderness on firm pressure ; yet moderate pressure sometimes relieves the pain. In local intestinal catarrh the pain is confined to the portion of the intestine involved.

Flatulence, gurgling, and tympanitic distention of the abdomen are usually present, and offensive borborygmi occur with the passages and with eructations which may give a sense of relief. When gurgling or borborygmi are prominent the *small intestine* alone is involved. When the passages contain unchanged ingesta, the large intestine is usually the seat of the catarrh. Nausea and vomiting indicate that gastric catarrh is associated with the intestinal, the combination being called *gastro-enteritis*. If severe intestinal catarrh has continued for a long time, there may be nausea, but rarely vomiting.

The local symptoms of enteritis are usually preceded and accompanied by a mild remittent type of fever. The skin is dry when the temperature is much elevated, and sweating often occurs at night. The change in the pulse corresponds to the elevation of temperature. Headache, thirst and loss of appetite accompany the fever, and are more marked the nearer the inflammation is to the stomach ; in one case there may be great restlessness, in another extreme lassitude and prostration. The tongue is usually dry and heavily coated ; in children it is often glazed and "beefy ;" in either case the breath is offensive. The urine is scanty and dark, almost black in the bilious variety.

When it occurs in children, it has been called gastric or *"infantile remittent fever ;"* in the evening the temperature may rise to 101° or 104° F., and be normal the next morning. The diarrhœa is severe in this class of cases, and the abdomen is usually very much distended. There is great restlessness, thirst is excessive, and the little patients are constantly calling for cooling drinks. The features become pinched, the lips pale and drawn, and the eyes deeply sunken. Vomiting is frequent and is "retching" in character. The papillæ of the tongue are elevated and covered with a yellowish coating ; all the other symptoms of acute enteritis attend it.

Duodenitis rarely occurs independently of gastric catarrh, in fact "gastro-duodenal catarrh" is much more common than simple duodenitis. The prominent symptom of duodenitis, independent of the symptoms of the accompanying gastric catarrh, is jaundice, which results from the secondary inflammation of the ductus communis, causing obstruction to the passage of the bile into the duodenum. A very acute duodenitis is liable to complicate extensive burns of the surface ; in obscure cases the urine should be analyzed for bile pigment, which may be found before the jaundiced hue of the skin is apparent. Some regard dyspeptic symptoms coming on three or four hours after meals as indicative of duodenal catarrh, but this symptom is only valuable in connection with the others.

Membranous enteritis has few distinctive symptoms, all the symptoms of acute simple catarrh are much exaggerated. The stools often contain bloody mucus and pus. If shreds of false membrane, or cylindrical casts of segments of the lower bowel, are voided in the diarrhœal discharges, the diagnosis is readily made, but without these it cannot be recognized.

Phlegmonous enteritis is a very grave disease. Pain and tormina are intense and come on in paroxysms. The abdomen is distended, tympanitic, and extremely tender, and the position of the patient is similar to that assumed in peritonitis. Vomiting is frequent, and severe; late in the disease it becomes fetid and even fæcal, although in some cases there is a mere regurgitation. The temperature rises to 103° or 105° F., the pulse keeps pace with the temperature, and is small and compressible. During a paroxysm of severe pain, the otherwise dry skin becomes covered with a profuse perspiration, and the distended intestine may rupture and gas escape into the peritoneal cavity. Constipation generally attends these cases, and the appearance of diarrhœa generally indicates the commencement of convalescence, during which the weakness increases for a time. With this convalescing diarrhœa the tongue is red, dry and glazed. But if a fatal termination is to be reached, the face becomes shrivelled, prostration becomes extreme, distressing hiccough occurs, the extremities become cold, and collapse closes the scene, the mind remaining clear to the last. The urine is very scanty, and frequently suppressed, a precursor of a fatal termination.

In *chronic enteritis* casts of mucus, already described, are passed with the stools, and are sometimes thought by the patient to be the mucous membrane of the intestine or a large intestinal worm. If the disease is long continued there is progressive emaciation, until the wasting is greater than in any other disease. The skin has a pale or dirty muddy hue, and the accompanying hypochondria may lead to a condition of melancholia. The tenacious layer of mucus which coats the intestine acts in the same way as in chronic gastritis, the contents of the intestine undergo decomposition, and gases are set free which distend the abdomen, interfere with respiration, and secondarily induce a passive hyperæmia in other organs. The passage of this flatus, and an occasional diarrhœal attack afford great relief to the patient. Chronic enteritis in *children* is marked by a diarrhœa which, though at first mucous in character, soon becomes serous and afterward dysenteric. The mouth shows evidences of "thrush," and emaciation is steadily progressive.

Differential Diagnosis.—*Acute intestinal catarrh* may be mistaken for *dysentery, hernia, acute* and *chronic poisoning, peritonitis,* or for *typhoid fever.* The diagnosis between intestinal catarrh and dysentery will be considered under the head of dysentery.

In *hernia,* the sudden onset of the symptoms with a history of previous good health, the localized pain, constipation, vomiting often following some sudden exertion or extreme muscular effort, and the existence of a hernial tumor establish the diagnosis.

Acute *poisoning* from certain articles of diet (as eating toadstools for mushrooms) can often only be differentiated in the first twenty-four hours by the history of the case. Poisoning from arsenic or any other chemical irritant causes severer gastric symptoms than are ever present in intestinal catarrh. The vomiting in arsenical poisoning is never stercoraceous, while phlegmonous enteritis of the same severity is usually attended by stercoraceous vomiting. A chemical analysis of the ejected matters will establish the diagnosis.

Peritonitis comes on rapidly, and at its onset the abdomen becomes exceedingly tympanitic and tender to pressure, while the advent of enteritis is comparatively slow, and excessive tympanitis is very rare. Vomiting rarely occurs in peritonitis until the peritoneum over the stomach is involved, and then it is "spinach-green;" in enteritis so severe as to be confounded with peritonitis, vomiting would be an early symptom and would not have a spinach-green character. There is constipation in peritonitis, while diarrhœa is the rule in enteritis. The pulse is tense and wiry in peritonitis, rapid and feeble in enteritis. The temperature is usually higher in enteritis than in peritonitis. As peritonitis becomes general, there are symptoms of collapse, and the anxious face, thoracic respiration, the immobility and the position of the patient are all characteristic.

Enteritis, particularly gastro-enteritis, is sometimes mistaken for *typhoid fever*, but in typhoid fever nausea, vomiting, and diarrhœa follow the febrile movement, whereas they precede it in gastro-enteritis. The temperature rarely rises to 103° F. in gastro-enteritis, while it may reach 104° or 105° in typhoid fever. The typical range of temperature during the first week of typhoid fever is characteristic, and is never met with in gastro-enteritis. In children the diagnosis is difficult without a complete thermometrical record.

Prognosis.—The prognosis in simple intestinal catarrh is generally good, particularly in children during dentition. The prognosis in *membranous* or *phlegmonous* enteritis is always bad, especially when it occurs with pyæmia, or Bright's disease. The duration in mild acute intestinal catarrh is ordinarily from three to five days. Chronic intestinal catarrh will persist as long as the cause which produces it is in operation. The signs which indicate recovery are subsidence of pain, the appearance of normal fæcal discharges, the clearing of the tongue, and a hopeful countenance. But when emaciation is progressive, the pulse irregular, or continuously rapid, constipation alternating with a serous diarrhœa, and profuse sweatings occurring at night the prognosis is unfavorable. *Death* may result from exhaustion, peritonitis, or from some of the more serious complications.

Treatment.—First, compel the patient to remain in bed until all active symptoms subside. If there is reason to believe that irritating substances in the intestine excite and keep up the catarrh, give a dose of castor-oil or calomel. It is safe to begin the treatment in *every case* of acute intestinal catarrh by the administration of castor-oil. The diet should consist of skimmed milk, or milk with lime-water, prepared meats, and light broths containing but little starchy matter. The yolk of eggs may be given with the milk. No fats should be allowed, or bread or any form of starchy food. Young infants should be immediately placed on a healthy wet-nurse. When prostration is marked stimulants may be carefully administered. The abdomen should be covered with flannel, and if pain is excessive warm fomentations of belladonna or opium may be employed. Opium is the most efficient agent in the treatment of all varieties. It must be given in sufficient doses to secure rest to the intestine, and to relieve the pain, half a grain every two or three hours is usually sufficient for an adult; its use must be continued until the diarrhœa ceases.

Rectitis is the only variety where astringents may be used. Here an anodyne and astringent plan may be combined, the best results having been obtained by enemata. When the catarrh is of *malarial* origin, quinine must be given in large doses. If it has been the result of exposure to wet and cold diaphoretics are indicated. In an intense form of enteritis three or four leeches may be applied to the abdomen, around the anus, or at the points of tenderness. Membranous or phlegmonous enteritis is to be treated the same as dysentery.

Chronic intestinal catarrh may be treated by astringents: the best are the nitrate of silver, the acetate of lead, and the sulphate of copper. A course of mineral waters will in many cases have a beneficial effect, and sea-bathing, cold sitz-baths, or sponging the abdomen with cold salt water may be of service in mild cases.

DIARRHŒA.

Diarrhœa is the frequent discharge of fluid or semi-fluid fæces (without tenesmus); it may be acute or chronic. It is a symptom of a variety of morbid conditions which will be considered under their appropriate heads.

The following are the principal varieties of acute diarrhœa:

Irritative diarrhœa includes those fluxes attended by pain and griping so often met with in children during the summer months in our large cities; those "brought up by hand" and those who have just been weaned are most liable to it. In adults, this form of diarrhœa may be caused by excess of food, improper and unseasonable food, improperly masticated food, foul water, tainted meats, etc. Personal idiosyncrasies play an important part in its causation. The diarrhœa produced by drugs causing hyper-purgation is "irritative." So also the pseudo-diarrhœa induced by hardened fæces, the result of long-standing constipation. The presence of worms, excessive discharges from the liver and intestinal surface, especially if they are inflammatory in character, are causes of irritative diarrhœa.

Symptomatic diarrhœa is part of the natural history of typhoid fever, waxy intestines, intestinal ulcerations, inflammation of the large and sometimes of the small intestine, Bright's disease, pyæmia, the exanthemata, Hodgkin's and Addison's diseases, leukæmia, and all forms of cholera. The diarrhœa of enteritis and proctitis is symptomatic.

Mechanical diarrhœa is that form in which the fæces are made fluid by a large amount of serum poured into the intestinal canal, the serous flow being induced by the action of salines, as Epsom or Rochelle salts. Hepatic, pulmonary, and cardiac diseases which retard the returning blood current from the superior and inferior mesenteric veins, will cause a transudation of serum into the intestine, which will dilute the fæces and wash out the intestine, causing diarrhœal discharges.

Nervous diarrhœa may be caused by fright, grief, great anxiety and severe pain. It is marked by profuse watery fæcal discharges, which, when once established, are apt to persist. It often comes on so soon after taking food that the food is passed undigested, and it is then called *lienteric diarrhœa*. The discharges usually are largely serous.

Choleraic diarrhœa precedes an attack of cholera, and is a prominent symptom in cholera morbus.

Vicarious diarrhœa is usually compensatory. When the functions of the skin, kidneys, or lungs are suppressed a flux from the bowels affords relief. Some regard a gouty diarrhœa as vicarious. Chilling the body suddenly produces a vicarious diarrhœa, provided enteritis is not established. In the latter case the diarrhœa would be symptomatic. Intense heat brings on a vicarious flux. Many fevers and acute diseases attended by an ushering-in chill cause diarrhœa, as much from chilling the surface (inducing a vicarious flux) as from the action of their specific poison (in which the flux would be symptomatic). Thus malarial, puerperal, and septic fevers are often attended by diarrhœa.

Some authors make different varieties of diarrhœa according to certain prominent symptoms, and speak of simple, fæcal, or stercoraceous diarrhœa (usually irritative), bilious diarrhœa,—when the dejections contain a large quantity of greenish-yellow fluid,—serous, mucous, and dysenteric diarrhœa. The discharges in the last variety contain mucus and blood.

Fatty diarrhœa is the result of faulty pancreatic digestion.

The term *crapulous* was formerly given to that variety caused by over-indulgence at table, or the ingestion of unwholesome food.

A diarrhœa is *critical* when it attends the crisis of a disease, not having existed before that time and ceasing directly after it.

A *colliquative* diarrhœa is a copious watery flux, occurring in wasting diseases towards their close, *e. g.*, phthisis, cancer, Bright's disease, etc.

The diarrhœa accompanying pyæmia and certain septic blood-conditions is by some called *eliminative*. It is a question whether the flux carries away the poison or the poison induces the flux.

Symptoms.—The symptoms of diarrhœa are too well known to need repetition; but cases vary greatly, not only in the kind of fluid dejections, but in their amount and frequency. A diarrhœa from over-eating may be harmless or even beneficial in relieving an overtaxed digestive system. Again, a profuse diarrhœa may be exhausting enough to cause anæmia, and in some chronic diseases hastens the fatal issue. Colicky pains and cramps in the limbs almost always accompany diarrhœa attended by profuse watery discharges. Thirst, anorexia, and febrile movement indicate that the diarrhœa has an inflammatory origin. In copious fluxes (serous diarrhœas) the urine becomes scanty, acid and albuminous. In fatty diarrhœa, free fat is found mixed with the fæcal masses.[1] Jaundice and melæna accompany some cases of fatty diarrhœa.

Chronic diarrhœa is always associated with some form of chronic organic disease, *e. g.*, chronic enteritis, intestinal ulcers, syphilis, malaria, scurvy, phthisis, etc. In India, chronic diarrhœa is called the *white flux*. Anæmia and exhaustion are its most constant symptoms. After (apparent) recovery there is a strong tendency to its return.

Differential Diagnosis.—Diarrhœa may be mistaken for *cholera, dysentery,* or a condition produced by the prolonged *retention of fæces.*

In *cholera,* the history of the epidemic, the watery stools resembling rice-water, the persistent vomiting, the cramps, suppression of urine, and the tormina will be sufficient to exclude a simple diarrhœa.

In *dysentery* there will be fever, rapid pulse, early and great exhaustion, tormina and tenesmus, scanty bloody stools having a dysenteric odor, and more or less tenderness along the line of the large intestine.

A diarrhœa dependent upon prolonged *retention of fæces* is recognized by the history of previous constipation and the presence of thin muco-feculent fæces accompanied by straining, a sense of soreness in the sacral region, and the detection of a fæcal mass by a rectal exploration. It is important to recognize this condition early.

Prognosis.—The prognosis in symptomatic and inflammatory diarrhœa depends upon the primary causative disease with which it occurs. The prognosis in simple diarrhœa is good, yet the disease is dangerous in the very young and very old. Nervous diarrhœa is apt to become chronic and often proves very obstinate. In fatty diarrhœa 50 per cent. die.

Treatment.—The treatment of diarrhœa will be determined by the causes which produce it, and the symptoms which attend it; if it depends on undigested food, the first indication is to remove the substances which are causing the intestinal irritation by a full dose of castor-oil, or rhubarb and soda. The diet should be restricted to milk and lime-water, and rest in bed should be enjoined. In the feeble a teaspoonful of brandy may be given every two or three hours. If the discharge continue, camphor, kino, bismuth or dilute sulphuric acid may be administered after each passage. If the discharges are accompanied by colicky pains and griping, opium may be combined with bismuth and camphor, or a simple diarrhœa mixture will be found efficacious.[2] In malarial diarrhœa, quinine must be given in combination with opium and capsicum. In bilious diarrhœa hydrargyrum cum creta may be combined with opium. In the summer diarrhœa of children, the treatment described under cholera infantum is indicated. It is often rapidly cured by enemata of chloral hydrate (gr. ij) in two or three drachms of starch water. In nervous diarrhœa I have found oxide

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[1] Dr. Bright stated that fatty diarrhœa probably indicated disorder of the pancreatic functions before Bernard discovered what these functions were.

[2] ℞

Spts. lavand. com	℥ ij.
Tr. opii	ʒ ij.
Tr. rhei	ʒ ss.
Ol. sassafras	gtt. x.

M.—Sig. Teaspoonful after each movement.

of zinc the most beneficial. Scorbutic diarrhœa is not influenced by drugs; lemonades, anti-scorbutics and fresh vegetables will usually check it readily. A vicarious flux frequently needs to be encouraged rather than checked, unless the patient is anæmic. As regards the treatment of fatty diarrhœa we have but few observations; large quantities of olive oil in one case, large quantities of whiskey in another, and a change from an indoor to an outdoor life in still another case, resulted in recovery.

In *lienteric* diarrhœa, arsenic is beneficial; it may be combined with bismuth or the alkalies. Hydrochloric acid is sometimes useful; astringents are not indicated.

In the treatment of *chronic diarrhœa*, bismuth is the most reliable drug. There should be great care in diet, and the body should be covered with flannel, even in warm weather. Sea voyages and change of climate are often of service. Tonics are indicated, and copper and silver salts are the best astringents. Hope's mixture—a well-known combination—will often control it when all other means have failed. In chronic nervous diarrhœa, arsenic and the bromides are indicated.

CHOLERA MORBUS.

Cholera Morbus, called also *cholera nostras*, *English cholera*, and *sporadic cholera*, is in reality a simple entero-catharsis.

Morbid Anatomy.—If any anatomical lesion exists, it consists in an acute gastro-enteritis; but the disease may occur without any discoverable lesions, thus simulating a functional disorder. It is so rarely fatal, that there has been little opportunity to study its morbid changes. In the few cases where post-mortems have been made, no adequate lesions have been discovered. Death may occur, and the intestinal tract may exhibit no morbid changes. Sometimes there is cerebral anæmia with serous effusion into the sub-arachnoid spaces.

Etiology.—Cholera morbus almost always occurs during the summer months. In this country it is most prevalent in July and August. Sudden checking of the perspiration, or suddenly chilling the surface of the body by external cold, or iced drinks, and sudden changes in the temperature after a heated term will produce it. Its most frequent cause is undigested food, as shell-fish, unripe fruit, cucumbers, etc. Sudden arrest of the digestive process from mental emotion is said to induce it. Some claim that malaria will cause it, especially in those greatly exhausted. Overdoses of tartar emetic and elaterium bring on attacks of vomiting and purging very similar to cholera morbus. Its prevalence during certain seasons seems to indicate a specific cause, perhaps some peculiar atmospheric condition. It is infrequent in old age. It attacks males oftener than females. In many cases its only discoverable cause is intense nervous disturbance, on account of which the peristaltic action of the intestines is greatly exaggerated.

Symptoms.—The symptoms of cholera morbus are familiar. An attack usually begins at night by vomiting and purging. The matters vomited are first, undigested food, gastric mucus and bile; afterward large quantities of acid or bilious fluid. The vomiting is projectile in character, and there is temporary relief after each attack. The bitter fluid ejected leaves a burning sensation in the mouth and throat. Although the thirst is intense, fluids as well as solids are immediately rejected. In some instances, instead of an abrupt onset, the attack is preceded for several hours or a day by nausea, general malaise, or sense of weight and uneasiness in the epigastrium and lower part of the abdomen, occasionally accompanied by colicky pains. Evacuations from the bowels follow each other in quick succession, the dejections becoming watery and profuse, and having a mouse-like odor. In some cases purging alone is present. After an attack has continued for some hours the discharges become watery and odorless, but they always contain bile. Pain generally accompanies or precedes every act of vomiting or purging, which either occur together or rapidly succeed each other. The larger the evacuations the lighter their color, and greater the thirst.

In all severe cases there are cramps in the lower extremities, especially in the calves of the legs and feet. Both vomiting and purging occur suddenly and without premonition. The skin is cool and covered with a profuse perspiration. The pulse grows weak and rapid as the vomiting and purging become more severe. The abdomen, at first distended, becomes retracted; sometimes the abdominal muscles are knotted by cramps. The urinary secretion, after the excessive watery discharges from the alimentary track, is greatly diminished, and traces of albumen and desquamated epithelium may be found in it. These severe symptoms, although seeming to threaten the life of the patient, usually continue only for a few hours, and the patient rapidly convalesces. If the attack is protracted, the pulse becomes flickering and imperceptible at the wrist, the countenance pale and shrunken, the voice feeble and the surface icy cold. This condition is called the *algid stage* of cholera morbus, and the patient may pass into a state of collapse, which may be followed by death. In all cases the mind is perfectly clear, and recovery or death occurs within twenty-four or forty-eight hours from the beginning of the attack. A fatal issue in adults is exceedingly rare. Sometimes a fever, attended by typhoid symptoms, follows the stage of collapse, called "the reaction fever." Generally, the stools become normal in character the day after the commencement of the attack, and the patient is simply weak. There are rarely any febrile symptoms during its active period.

Differential Diagnosis.—During a cholera epidemic, it is difficult to differentiate cholera morbus from either *cholerine* or true *Asiatic cholera*. When not prevailing as an epidemic, Asiatic cholera is differentiated by the absence of fæcal odor, by the color of the stools and by the duration of the attack. Cholera morbus rarely continues longer than twelve or eighteen hours. In cholera, collapse comes on early and the discharges have the distinctive rice-water appearance from its commencement.

Cholera morbus may be mistaken for the effects of *irritant poisons*. In cases of poisoning, the mouth and pharynx are usually intensely hyperæmic, and the pain is more intense and constant than in cholera morbus.

If there is diarrhœa, the discharges are blood-stained, and this never occurs in cholera morbus. In poisoning, the pain over the stomach is more severe, and an analysis of the vomited matters quickly decides the question.

Cholera morbus is differentiated from *typhlitis* and *perityphlitis*, by the absence of a tumor, the short duration of the attack, and by the intensity and character of the gastric symptoms. Typhlitis is, in the majority of cases, accompanied by constipation ; cholera morbus by diarrhœa.

Prognosis.—Cholera morbus is rarely a fatal disease. Its duration varies from two hours to two days. In the aged, and in the feeble, the prognosis is more unfavorable than in healthy adults. It is also more unfavorable when cholera and dysentery prevail epidemically, or when there is co-existing renal disease. When a patient passes into the algid stage or stage of collapse there is always danger.

Treatment.—In mild cases of cholera morbus, ice may be given to check the vomiting, and sinapisms applied to the epigastrium. In the severer cases, a quarter of a grain of morphine hypodermically will generally relieve the distressing symptoms. In all cases sinapisms should be applied over the abdomen, and if there is great coldness of the surface, *dry heat* should be applied to the extremities. If there is great prostration, with coldness of the extremities, alcoholic stimulants must be given with morphine, and if there is hepatic tenderness, one-half grain of calomel every hour for six hours will be of service. Small doses of the mineral acids are often beneficial after the vomiting is relieved.

If the diarrhœa is protracted, vegetable astringents may be given. All remedies should be given in small doses. After the subsidence of the attack, care should be exercised in the diet for several days, and the patient should be kept in bed.

CHOLERA INFANTUM.

Cholera infantum, or *summer complaint* in children, is a very common disease in cities and large towns during the heat of summer.

Morbid Anatomy.—Its principal lesions are found in the colon next to the ileum, but sometimes the whole intestinal tract is involved. Patches of arborescent injection are scattered over the intestinal mucous surface, which sometimes assumes a bright red color and becomes more or less tumefied. The most constant change is enlargement and softening of the follicles. Peyer's patches present the shaven-beard appearance, and in protracted cases the mucous membrane is studded with follicular ulcers. Over the inflamed patches the peritoneum may be reddened and covered with lymph. The intestines usually contain a thin rice-water fluid, more rarely fluid fæces. The mesenteric glands are sometimes enlarged and the liver congested. Death may occur and the intestinal tract exhibit no morbid change.

Etiology.—The prevalence of cholera infantum in summer is in direct proportion to the height of the temperature. Teething children are especially liable to it. Those over three years are less liable to it. Overcrowded and anti-hygienic surroundings predispose to it. It prevails extensively among the children of tenement-house districts and in asylums. The greatest mortality occurs during hot, still, sultry days. Gases from cesspools and malarial influences are powerful predisposing causes. The improper feeding of children which prevails in the densely packed tenement-house districts of New York City, where the death-rate from this disease is twice that of any other city in the world, has as much to do with its prevalence as the high temperature. Artificially fed children are more subject to it than those who nurse.

Symptoms.—It begins either with vomiting or diarrhœa, or both. There may be prodromata, but they are vague and inconstant. The child rejects all food, and becomes peevish or languid and apathetic. Purging is always present, and the passages are watery and greenish in color, rarely colorless, and contain curdy masses mixed with mucus. There is a peculiar odor to the discharges which is characteristic of the affection. Sometimes the stools contain particles of undigested food that have passed through the intestinal tract unchanged. The discharges are more or less slimy, sometimes frothy, and at first have a distinctly sourish odor. The child is constantly thirsty, although all liquids, even its mother's milk, are instantly rejected. Prostration and emaciation begin almost with the first discharges, and two or three days suffice to bring the healthiest child into an extremely exhausted condition. The reaction of the vomited matter varies; it may be acid or alkaline.

The patient becomes stupid, with a marked tendency to coma. Convulsions are not infrequent. The temperature is rarely above the normal, except during the first few hours, and then it is remittent in character. The urinary secretion is diminished, and uræmic symptoms often precede a fatal termination. The number of passages varies from six to seventy-five in the twenty-four hours; the abdomen at first may be distended and tympanitic; later, it is retracted, and always tender. The pulse varies from 120 to 160, and there is often marked dyspnœa. These little patients die from inanition, or rapidly recover after having seemingly been on the verge of death. Not infrequently they gradually pass into a condition where months will elapse before normal intestinal digestion is re-established. The disease usually lasts a week, at the end of which death or recovery takes place. Deceptive remissions may occur, only to be followed by graver and often fatal symptoms. Tenderness on pressure is generally marked along the whole line of the colon, and the diffuse erythema about the anus causes intense pain whenever a passage occurs.

Differential Diagnosis.—Cholera infantum may be mistaken for *Asiatic cholera*. The points already given for the differential diagnosis of cholera and cholera morbus will suffice to establish the diagnosis.

A *spurious hydrocephalus* sometimes follows cholera infantum, the symptoms of which, and the termination in coma, resemble very closely those of *acute hydrocephalus*. In spurious hydrocephalus there is diarrhœa and a history of previous vomiting and purging. In acute hydrocephalus, there is constipation. In spurious hydrocephalus, the pupils are dilated but regular, while in tubercular meningitis they are contracted and irregular. In spurious hydrocephalus the pulse is accelerated but regular, while in acute hydrocephalus it is slower than normal, irregular and intermittent. In acute hydrocephalus the abdomen is retracted; in spurious hydrocephalus it is distended and tympanitic. The hydrocephalic cry and convulsions, on the one hand, and the age of the patient and the mode of the attack on the other, will further aid in the diagnosis.

Prognosis.—The prognosis in a severe attack of cholera infantum is always unfavorable. Its duration depends upon the vigor of the child and the severity of the attack. It may continue a week, or death may occur in twenty-four hours. Children who are artificially fed are less likely to recover than those who receive the breast. The rate of mortality is greater in those living in badly ventilated tenements than in those with better hygienic surroundings. Continued vomiting, excessive purging, stupor, or great restlessness and convulsions, are unfavorable symptoms. The prognosis is favorable when the vomiting and purging are not excessive. Death may occur from exhaustion, or cerebral effusion causing convulsions and coma. Œdema of the lungs may result from heart-failure, and this, with hypostatic congestion, may cause death. In all cases the prognosis must be guarded.

Treatment.—The treatment of cholera infantum is mainly prophylactic; the diet and hygienic surroundings are the most important. Occurring as it does in large cities in the summer it is best treated by removing the children of the poor to the sea-shore. When this is impossible, the child must spend the morning and evening in the fresh air. The first indication, then, is change of air and location. The establishment of various seaside sanitariums for children during the summer months in the neighborhood of large cities is the most important advance that has been made in the management of this disease. At the same time great care must be exercised in the diet; fresh cow's milk with barley and lime-water added, is the best artificial diet; a good wet-nurse is always to be preferred. The amount of food taken should be regulated by the capacity of each case to retain it. At the onset of the attack a few drops of brandy in a teaspoonful of barley-water is all that should be allowed, and absolute rest in the horizontal position should be maintained as long as the vomiting continues. To relieve the intense thirst the child may suck pounded ice in a linen bag. The only drug that I have found efficacious in controlling the vomiting is calomel, which should be given dry on the tongue in minute doses, 1-12 of a grain every half-hour. Some claim excellent results from the administration of bismuth and carbolic acid; salicylic acid is also of value in arresting fermentation. Both bismuth and calomel are efficacious when the stools contain large quantities of mucus. If the intestinal symptoms persist after the vomiting is relieved, camphor and opium may be given—five or ten drops of the tr. opii camph. every two hours.

In malarial districts, quinine should be given as soon as the stomach will retain it. When vomiting is slight and purging is excessive with great prostration, benefit will be obtained from camphor and brandy. The vegetable astringents, such as hæmatoxylon, kino, and catechu, are of service in controlling the diarrhœa which so often follows a severe attack of cholera infantum. During convalescence, wine-whey may be given in connection with cod-liver oil, and the phosphates and oils may be applied to the surface as a means of sustaining the strength of the child. Seaside resorts and salt water baths are especially beneficial to this class of patients after the severity of the attack has passed. Spiced poultices wet with brandy and worn over the epigastrium are of service.

Flannel should be worn next the surface during convalescence, and great care should be exercised to avoid exposing the surface to changes of temperature, for capillary bronchitis carries off a large number of convalescents.

INTESTINAL DYSPEPSIA.

Closely connected with diarrhœa is a functional disturbance of the intestines which may be designated intestinal dyspepsia. It depends upon a derangement of the functions of the small intestine independent of any organic lesion.

Etiology.—Intestinal dyspepsia may be a primary disease, or it may be secondary to gastric dyspepsia, diseases of the liver, pancreas, or large intestine. Its causes are similar to those of gastric dyspepsia, such as structural changes in the mucous membrane, altered conditions of the secretions of the small intestine, the presence of undigested food, or the ingestion of improper food; an altered condition of the muscular coat of the intestine often accompanies general malnutrition.

Symptoms.—Pain, which is generally a constant symptom, is of a dull, aching character and not circumscribed, but radiates over the upper portion of the abdomen. It is rarely acute, like that of peritonitis, nor as sudden in its advent as the pain of colic, nor does it bear any relation to the ingestion of food. Nausea and vomiting, when present, depend more upon the accompanying stomach derangement than upon any intestinal disturbance. Constipation and gaseous distention of the large intestine are prominent symptoms. It is only after repeated attacks that the patient's health becomes impaired, so that he loses flesh and strength, and begins to worry about himself, fearing some serious organic lesion. It is a peculiar fact that the appetite is seldom if ever impaired.

Treatment.—The immediate condition can usually be relieved by five-grain doses of pancreatin taken two hours after eating. This also prevents fermentation and thus decreases intestinal irritation. Hygienic measures are the most important and should first be tried. If possible the patient should travel; if this cannot be done, out-of-door exercise, such as horseback riding and walking, will be most beneficial. This class of patients should abstain from all fats and starches, eat principally meat, and vegetables containing but little starch; of the drugs which yield the best results, ipecacuanha and cubebs in the form of a powder stand first, although some prefer bismuth combined with iron and quinine.

DYSENTERY.

Dysentery is a specific febrile disease, with a characteristic local lesion. Its *local lesion* is an inflammation of the mucous membrane, and of the solitary and tubular glands of the large intestine, which may be catarrhal or croupous in character. It has points of resemblance to acute infectious diseases, being attended by fever, and having a characteristic local lesion. It may be *acute* or *chronic, epidemic, endemic,* or *sporadic.*

Morbid Anatomy.—The difference between sporadic or simple dysentery and epidemic, often malignant, dysentery, is that in the former the anatomical changes *do not* pass to extensive ulceration, and it attacks comparatively few individuals in the same locality. In mild cases, the lesions are confined to the lower portion of the large intestine, while in severe cases the whole length of the large intestine is involved.

The first change, in the mild as well as in the severe types, is a more or less intense hyperæmia of the intestinal mucous membrane. Its color varies from a slight inflammatory blush to a purplish red; this change in color is never uniform throughout the affected portion. With the change in color, the mucous membrane becomes swollen and softened, from œdema of the mucous and submucous tissue; the latter being infiltrated with inflammatory products which give it a "cloudy" appearance. The thickening and softening are more marked at some points than at others. They are usually most distinct at the summit of the folds of the mucous membrane. In mild cases, only a small number of prominences indicate the localities which are involved, whereas in severe cases these prominences are so numerous that they give the membrane a lobulated appearance. In the first stage, the solitary follicles become distinct, enlarged, prominent, and vary in size from a millet-seed to a small pea. Each is surrounded by a zone of turgid and enlarged vessels. These glands enlarge, become distended with a whitish, albuminous exudation, having a dark central spot surrounded by a vascular ring. The next change, in most instances, is rupture of some of the distended capillaries in the wall, thereby filling the follicular cavity with blood. Molecular disintegration of the gelatinoid contents now occurs, and ulcers are formed after the contents of the follicles are discharged.

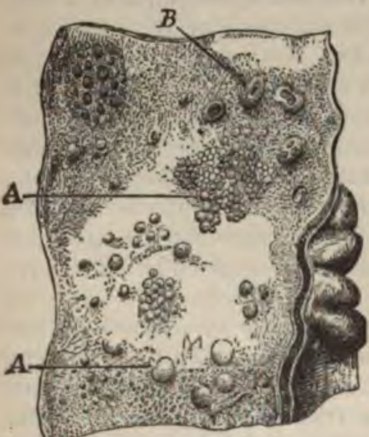

FIG. 62.
Mucous Surface of lower portion of Large Intestine in the First Stage of Acute Dysentery.
A, A. Enlarged follicles.
B. Small ulcers.

These ulcers are at first round; later they gradually enlarge, and two or more may coalesce, their edges becoming everted and flattened, and assume an irregular, serpentine, or rodent shape. The long axis of the ulcer usually corresponds to the fold of mucous membrane circumscribing the intestinal canal. The condition of the different ulcers in the same case varies; one is pale and superficial, another deep, angry, and irritable; again they may be covered by a more or less dense layer of lymph, or a thin film of serous fluid. The adjoining tubular glands are involved. Sometimes ulceration destroys *every* solitary follicle of the involved part. The floor of the ulcer may be on the muscular or on the peritoneal layer of the intestine. Between the ulcers is often found a polypoid growth, which is simply a flaccid, vascular tuft of mucous membrane.

As the mucous membrane of the large intestine has the poorest blood supply of any mucous membrane, a very acute dysenteric process may cause the whole length of the intestine to become a black, shaggy, gangrenous, pus-infiltrated slough. In high grades of dysentery, large tracts of the mucous membrane are converted into dark brown or black, ecchymotic, and nodular carbonified masses (sphacelus) more or less friable, and underneath them the submucous tissue is sometimes infiltrated with pus. When this occurs around the ileo-cæcal valve, invagination and obstruction may result. This purulent infiltration must be regarded as part of a "reactive inflammation," following the removal of the charred-looking mass. Some observers, however, are of the opinion that in rapidly sloughing dysentery the process begins by a submucous purulent cellulitis, which detaches the mucous membrane, this membrane then becoming gangrenous. Many pathologists regard coagulation-necrosis as the basis of all dysenteric processes in the follicles and tubular glands.

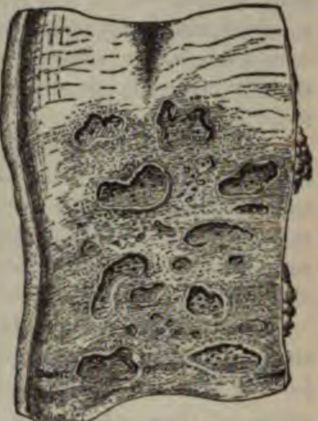

FIG. 63.

Mucous Surface of lower portion of Large Intestine in Second Stage of Acute Dysentery.

Ulcers are seen of varying size, from that of the single follicle to larger ones formed by their coalescence.

In some of the severe forms, the submucous cellular tissue becomes infiltrated with a sero-sanguinolent fluid, and its vessels become filled with black masses of altered blood. The muscular coat of the intestine is sometimes condensed, friable, and of a pale ashy-gray color. In severe epidemics, a thin dark-colored exudation covers the peritoneal investment of the affected portion of the intestine. In many cases the intestine becomes dilated, and is filled with dark blood and disintegrating inflammatory products. When the intestine contains a putrid, coffee-ground fluid, the case must be regarded as malignant. I have sometimes found the intestine collapsed and empty at the post-mortem of one who had suffered from severe dysenteric symptoms. In the mildest form of dysentery, cicatrization is accompanied by an exudation of lymph, which rapidly organizes. This lymph exudation first appears upon the floor of the ulcer; the edges become rounded, and are then drawn down towards the base, and so cicatrization is completed. When a large extent of the mucous membrane is removed, the edges of the ulcer are not approximated. The cicatrices which remain after the deep structures have been destroyed often cause "valve-like" or "annular" folds, which constrict the colon.

Perforation and peritonitis may result from an ulcer perforating the intestinal walls. If the cæcum is perforated, fæcal abscesses may form in the right iliac fossa. The liver is frequently congested, and it may be the seat of multiple abscesses. The mesenteric glands are enlarged, softened, and of a dark blue color. The lower part of the small intestine, in rare instances, may be involved in the dysenteric process. In the croupous variety, the mucous membrane of the large intestine undergoes changes similar to those which occur in croupous inflammation of other mucous membranes. It usually occurs in patches; the size of the patches will correspond to the severity of the inflammation. If the inflammation is mild in character, the membranous exudation will disappear after a few days, and the mucous membrane return to its normal condition. If it is severe, and the submucous tissues become infiltrated, there will be destruction of the mucous membrane and the formation of ulcers. These ulcers may involve the muscular and peritoneal coats of the intestine. They behave the same as those already described.

In *chronic dysentery*, the mucous membrane of the large intestine is studded either with slaty-blue cicatrices or pigmented ulcers. In the majority of cases, complete cicatrization of the ulcers does not occur. The edges of these ulcers are always made up of unhealthy tissue. These ulcerations are especially marked at the sigmoid flexure and in the rectum, while the mucous membrane in the remainder of the large intestine is thickened, tough, and pigmented. In some cases the intestinal walls atrophy and are thinner than normal, but generally, on account of the changes in the submucous connective-tissue, they are thickened and indurated; consequently there is more or less rigidity of the whole intestine, with narrowing of its calibre. Sometimes sinuses exist between the layers of the intestine; these are most often found about the rectum. In chronic dysentery, more frequently than in acute, are annular and valve-like constrictions formed, which cause subsequent constipation. Multiple abscesses in the liver are often met with in chronic dysentery. Small polypoid tumors sometimes form and project into the intestine.

Etiology.—Dysentery is especially liable to prevail in malarial districts. There are localities in which it is endemic, and others in which it is epidemic. It seems probable that there is a specific dysenteric miasm, and that the discharges from a dysenteric patient, when they have undergone certain changes, are capable of causing the disease. This may account for the occurrence of the disease in hot climates and in early fall in our own climate, under conditions similar to those which favor the development of typhoid fever. Impure air and water are recognized causes of its development; thus, seamen and those who live in crowded barracks are especially liable to it. In districts where it prevails, exposure to cold or chilling the surface is often an exciting cause. Bad or insufficient food, or a diet wanting in vegetables, alcoholismus, mental anxiety, and excessive fatigue are among its predisposing causes.

Symptoms.—Dysentery is preceded by loss of appetite, a furred tongue, constipation, or constipation alternating with diarrhœa, a dry skin, and a feeling of general malaise. The severer forms of acute dysentery commence with a chill or distinct rigor, followed by a slight rise in temperature, accompanied by anorexia and nausea. The temperature usually ranges from 101° to 103°; it may reach 105° F. The pulse is increased in frequency, small and compressible. With, or following these constitutional symptoms, there is a constant desire to go to stool, with tormina, both during and after a passage from the bowels. The evacuations are at first semifeculent mucus, watery looking, and contain lumps of hard fæces, "scybalæ." After and during stool, there occurs that painful straining with bearing down, called "tenesmus." The tenesmus is due to the abnormal sensibility of the lower bowel, and the involuntary action of the muscular fibres of the rectum. At the very onset of the attack, the nervous depression is very marked, the strength is diminished, and the face assumes a pale, anxious expression. The discharges soon become scanty and more frequent, containing blood and mucus (the "bloody flux"), and have the peculiar dysenteric odor. Twenty or thirty discharges from the bowels may occur in twenty-four hours, although they usually do not exceed eight or twelve.

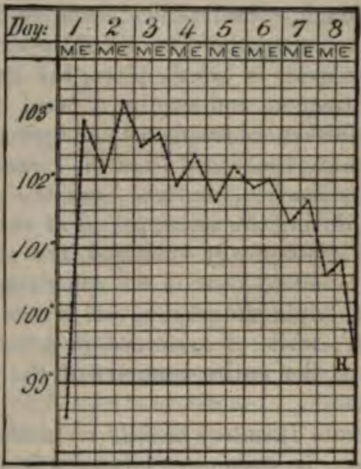

FIG. 64.
Temperature Record in a case of Acute Dysentery.

If the disease has its seat at the upper part of the large intestine, altered biliary secretions will be intimately mingled with the blood and mucus. If the dysenteric process is confined to the lower portion of the intestine, the blood will be separated from the mass and occur in streaks. As the disease progresses, the patient becomes more nervous and anxious, irritable and restless, and his countenance will be expressive of intense suffering. There is seldom much abdominal pain or tenderness on pressure during the first few days, but the slightest amount of solid food taken into the stomach causes tormina. The tongue is moist and covered with a thick whitish fur.

As the disease advances, in the severe type, the stools change in character; they contain sloughy shreds of exudative matter, looking like "washed raw meat," mixed with blood and purulent matter, or they are of a greenish color resembling spinach. The thickened intestine may now be felt through the abdominal parietes. The abdomen becomes tympanitic and tender; the tenderness is usually most marked at some point along the line of the large intestine. In some cases the stools become brownish, serous in character, and are often so copious as to cause extreme exhaustion. The pulse increases in frequency, and is extremely small and feeble; the tympanitis increases, the tongue becomes dry, its centre brown or black, and its edges red. The restlessness increases, with a mild delirium at night, which sometimes becomes violent. The *urine* is dark and scanty. There may be great difficulty in passing it, only a few drops at a time being voided,—strangury. If the case tends to a fatal termination, irregular febrile exacerbations and remissions occur, the stools have a cadaverous or gangrenous odor; hiccough, subsultus tendinum, cold perspiration, a flickering pulse, deeply sunken eyes, and cyanosis of the extremities usher in the fatal termination. If recovery is to take place, the discharges during the second week become less frequent and acquire a fæcal odor. The temperature falls, the pulse diminishes in frequency and gains in force. The tympanitis subsides and gases are discharged from the bowels with the fæcal discharges. As recovery is reached, the face loses the anxious, despondent expression which it had during the active period of the disease. Convalescence is slow.

Microscopically examined, the stools voided at the height of dysentery are found to contain fat spherules, blood globules, pus corpuscles, vibriones, triple phosphates, and traces of ingesta, all crowded into a molecular mass; while, if examined in a test tube, the amount of albumen present causes the entire contents of the tube to coagulate on the application of heat.

Acute dysentery in *children* is often accompanied by vomiting and convulsions. In some cases tenesmus is so great that prolapsus ani occurs.

A *malarial* dysentery is recognized by the periodicity of its febrile symptoms. The temperature is higher than in non-malarial dysentery, and exacerbations and remissions of all its symptoms occur at regular intervals; the hepatic, renal and splenic changes are the same as in malarial fevers. The stomach is very irritable, and the stools are likely to be serous from the onset, showing but slight traces of blood.

In *malignant* dysentery the typhoid state is present with the first dysenteric stool; the passages soon assume a gangrenous odor and contain gangrenous shreds of membrane, with abundant serum and blood. The countenance is not anxious, but listless and apathetic, the pulse is rapid and weak, the voice feeble, the stomach irritable, the skin cold and covered with a cold perspiration—indeed, a state of collapse is very quickly reached. The urine may be entirely suppressed, but, if passed, scalds, and has a fetid odor. Not infrequently, just before death, large amounts of blood are discharged from the bowels and also from the mouth and nose.

When all the causes of "scurvy" are added to those of dysentery, the symptoms of scurvy, namely, great prostration, emaciation, a pale muddy skin, darting pains in the limbs, the scurvy sore mouth, and the petechial spots, will be added to the dysenteric symptoms, and the stools will contain blood from the very onset, and are as fetid as in the malignant variety. In *scorbutic* dysentery, profuse and fatal hemorrhages are liable to occur after the first few days.

Chronic dysentery is, in most instances, the direct sequela of the acute form. In a few cases dysentery is chronic from the beginning, and its cause then lies in fatty or waxy degeneration of the liver or spleen, or both, or in a strumous or scorbutic taint. The temperature usually ranges above the normal. In some instances hectic fever may attend it. The evacuations are scanty and frequent, tormina and tenesmus are present in most cases, and the stools contain mucus. At times they are serous, pale, slimy, or frothy, but always fluid; occasionally they contain fæcal matter and are brown and watery. The patient progressively loses flesh and strength, although the appetite may remain good. Defective nutrition is shown by furuncles, a dry, scaly skin, a red, glazed, and often deeply fissured tongue, falling of the hair, and a worn and feeble expression of countenance.

Differential Diagnosis.—Dysentery may be mistaken for acute *rectitis* or *intestinal catarrh*, for *diarrhœa* complicated with *hemorrhoids*, and for *cancer* or *polypus* of the *rectum*. Acute *rectitis* begins with colicky pains and constipation; while dysentery commences with a distinct chill or rigor, diarrhœa, and a permanent elevation of temperature. Dysenteric discharges contain mucus, pus, blood, and scybalæ.

In *acute intestinal catarrh* there is no blood mingled with mucus in the discharges, and the latter do not have the dysenteric odor which is so characteristic of dysentery. Tenesmus is always present in dysentery, and never in intestinal catarrh. The discharges are profuse in intestinal catarrh, scanty in dysentery. The pain in acute enteritis is more intense and paroxysmal than in dysentery. The constitutional symptoms are much more severe in dysentery than in enteritis.

A simple *diarrhœa* in one suffering from hemorrhoids may be accompanied by bloody discharges and tenesmus, but the absence of constitutional symptoms and an examination of the rectum will readily establish the diagnosis.

In the same way *cancerous* or *polypoid* growths in the rectum, from which pus or blood is frequently discharged, can readily be differentiated from dysentery.

Prognosis.—The prognosis in simple, acute, and malarial dysentery is good. In the malignant, or asthenic and scorbutic varieties, it is exceedingly bad. The ordinary duration of acute dysentery is from eight to ten days; malignant dysentery may terminate fatally in two or three days. Sporadic dysentery in children usually lasts about a week. Chronic dysentery may continue for years. The favorable symptoms in any case are absence of a gangrenous odor to the stools, absence of great nervous depression and of an anxious, sodden expression of countenance, the gradual subsidence of the tenesmus and of the peculiar dysenteric stools. The unfavorable indications are a large quantity of blood in the discharges, a sunken aspect of the countenance, hiccough, vomiting, a distended, tympanitic abdomen, great nervous depression, a typhoid condition, great restlessness, suppression of urine, and marked cerebral disturbances. Dysentery may be complicated by extreme anæmia, by prolapsus ani, by hepatic abscess, by bronchitis and lobular pneumonia, by malaria, typhoid fever, purpura, scurvy, the hemorrhagic diathesis, enlargement of the spleen and liver, any one of which renders the prognosis unfavorable. *Death* may occur from exhaustion, from hemorrhage, from perforation and peritonitis, or from secondary pyæmic abscess. In all cases, however, the immediate cause of death is asthenia.

Treatment.—The preventive treatment of dysentery consists in disinfecting the evacuations as soon as they are discharged, in the same way as in typhoid fever, and the avoidance of all those conditions, such as bad hygienic surroundings, insufficient clothing, and improper food, which act as predisposing causes. A patient with dysentery must be kept in bed, and all irritating matter removed from the intestinal tract by a full dose of castor-oil; to accomplish this an enema of one to two quarts of warm water is recommended by East Indian physicians. The diet should be chiefly of milk with light meat broths; no solids should be allowed. If at the very onset there is great tenesmus, two or three leeches about the anus will often give great relief. Medicinally, opium is the drug almost universally employed, and should be given to semi-narcotism.[1]

Its direct action is three-fold—narcotic, sedative and astringent; secondly, it controls the inflammatory process by its action on the sympathetic nervous system. When the rectum is chiefly involved, it is best administered per rectum, but when the temperature is high and the tenesmus is intense, the rectal use of opium is contra-indicated. In such cases ipecacuanha in large or small doses has been found most efficacious. Some recommend that it should be given in thirty-grain doses,[2] but it seems to me just as efficacious when given in ¼-grain doses every half-hour. When larger doses are given, it must be administered when the stomach has been empty for some hours, and no fluid should be taken for some time after its administration; my own experience has led me to rely upon the ¼-grain doses of ipecacuanha with sufficient morphine hypodermically to relieve the pain and restlessness. Ipecacuanha is markedly beneficial in children, combined with bismuth, chalk, or bicarbonate of soda.

Hot fomentations or poultices over the abdomen are always grateful to the patient and are not contra-indicated. Finely pounded ice introduced into the rectum and ice-bags externally are recommended, but they have seemed to me to increase, rather than arrest the inflammatory process. In malarial dysentery, quinine must be given in full doses with the ipecacuanha, and when there is evidence of hepatic congestion one or two grain doses of calomel act beneficially. All treatment of malignant dysentery is unsatisfactory; it is summed up in the treatment of symptoms, and in supporting the patient with concentrated nutrition and stimulants. In the scorbutic variety, in addition to the opium and ipecacuanha, lemon and lime-juice, fresh vegetables and milk, and ripe fruit should be freely given. The "grape cure" has received a deserved reputation in the treatment of this variety. During convalescence iron, bark, and the mineral acids are indicated.

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[1] Opium may, by semi-narcotism, mask the true picture of the disease, and sudden death may unexpectedly occur when the friends regard the case as doing well.

[2] Ewart states that we possess in ipecacuanha "a non-spoliative antiphlogistic, a certain cholagogue and unirritating purgative, a powerful sudorific, and a harmless sedative to the heart and muscular fibres of the intestines." Quain's Dictionary, pp. 414–415.

Patients with *chronic dysentery* should reside in a mild, dry, equable climate, and wear flannel next the surface, especially over the abdomen. With some a sea-voyage will effect a cure. The diet should be most carefully regulated; each case is a law unto itself; and the articles of diet can only be determined by trial. Astringents, as the acetate of lead, sulphate of copper and nitrate of silver, combined with small doses of opium, are recommended, but I have found the greatest benefit from the prolonged use of cod-liver oil and the pernitrate of iron. Quinine is recommended by East Indian physicians.

TYPHLITIS.

Typhlitis, sometimes called "cæcitis," although a catarrhal inflammation of the cæcum and vermiform appendix, might properly be classed under the head of intestinal ulcerations, since the inflammation of the mucosa is commonly accompanied by ulceration.

Morbid Anatomy.—Typhlitis begins as an acute local catarrh of the mucous membrane of the cæcum, which soon involves the submucous tissue; the muscular coat loses its contractile power, so that the intestine becomes dilated and allows of large fæcal accumulations, which are usually attended by ulceration of the mucous and submucous tissue. The fæcal accumulation constitutes a typhlitic tumor. The catarrh may extend to the vermiform appendix, or an independent inflammation of the appendix may occur; in either case it becomes dilated and forms a sac with thin walls, which is filled with a semi-transparent fluid. The terminations of the ulcerations which occur in the cæcum are, 1st, rupture and general peritonitis; 2d, an extension of inflammation through the intestinal walls to its peritoneal covering, and a local peritonitis; 3d, extension of the inflammation to the connective-tissue at the posterior wall of the cæcum, where the peritoneum is wanting, and a consequent suppuration or adhesive cellulitis, which may bind the colon to the iliac fascia, and develop a suppurative "perityphlitis;" or, 4th, adhesions may form between the cæcum and small intestine, matting them together, and binding them to neighboring organs.

Etiology.—More than two-thirds of the cases of typhlitis are excited by the presence of foreign bodies, or by impaction of fæcal matter, from muscular atony of the intestine, the result of the habitual distention accompanying constipation; bilious and intestinal concretions and masses of lumbrici sometimes cause it. It may also result from acute or chronic intestinal catarrh. It is most frequently met with in males from twelve to thirty years of age.

Symptoms.—The premonitory signs of typhlitis are vague: sometimes there may be dull, uneasy pains in the right iliac fossa, usually of a distinct colicky character, or a dull, heavy, dragging sensation ; diarrhœa often alternates with constipation ; the abdomen is more or less distended and tympanitic. The fæces, for some time before the attack, will be described by the patient as hard round balls ; if it end in resolution, these symptoms gradually subside, and after a week or two the patient regains his former health. In some cases, these premonitory signs are absent. In either case, the actual symptoms begin with a severe pain in the cæcal region and right hip, increased by pressure and by motion of the parts. On account of a loss in the contractile power of the muscular coat of the intestine, there is nausea and vomiting. At first, the contents of the stomach are ejected, then bilious matters, and finally (in some instances) the ejected matter is stercoraceous in character. The pain is often remitting ; the patient is most comfortable on the right side. With these symptoms there may be a slight rise in temperature, but this is not constant, nor does it follow any rule ; the temperature may rise to 102° or 104° F., accompanied by a pulse of 130 per minute ; with these symptoms the patient often suddenly passes into a condition of collapse.

If recovery now occurs, the bowels will move spontaneously, and large quantities of fæces are discharged, accompanied by severe griping pains, and the typhlitic tumor may entirely disappear ; sometimes it remains, after copious discharges from the bowels, on account of the infiltrated state of the intestinal walls. This is called "chronic typhlitis." A local peritonitis may remain after the typhlitis has disappeared, but if peritonitis has occurred, it usually subsides with the typhlitis. When peritonitis occurs it will mask the ordinary symptoms of typhlitis.

Physical Signs.—*Inspection* may show a swelling in the right iliac fossa.

Palpation discovers a superficial, sausage-shaped tumor just above Poupart's ligament, its long axis pointing inwards and downwards, and sometimes reaching laterally to the median line, and vertically to the free border of the ribs. It is tender, slightly movable, and may give a "gurgling" on pressure. It is not to be forgotten that in chronic typhlitis with peritonitis, the tumor loses its characteristic sausage-shape and grows broader.

Percussion.—There is dulness on slight percussion over the tumor, and the limits of the tumor can be well defined.

Differential Diagnosis.—If a typhlitic tumor develops *slowly*, it may be confounded with other *abdominal tumors*, as ovarian, renal, cancerous and aneurismal. But in all cases the latter are covered by the intestine, whereas typhlitic tumors are superficial. The perforating peritonitis of typhlitis may be mistaken for *peritonitis* from other causes, when it comes on suddenly, and a diagnosis is only reached by careful study of the previous history, which in typhlitis is characteristic.

Prognosis.—The prognosis is always doubtful, but in the majority of cases favorable, statistics showing 75 per cent. of recoveries. The duration in severe cases may be only three days, and again it may continue for three or four weeks ; the average duration is from eighteen to twenty days. The peritoneal changes may be permanent. Typhlitis may be complicated by perityphlitis, peritonitis, periproctitis, perinephritis, fæcal fistula, pylephlebitis and thrombosis of the femoral vein. Death always occurs from some of the complicating conditions.

Treatment.—Typhlitis demands prompt treatment. Leeches should be applied over the cæcum, and followed by hot fomentations over the tumor. If there are no evidences of peritonitis, large enemata of tepid water may be administered, preceded by a full dose of castor oil; drastic purges should never be employed. If free evacuations from the bowels are obtained, the case soon terminates in recovery, for the swelling, which often goes on increasing afterward, is *not* due to fæcal accumulation, but to a local inflammation. If there are evidences of local or general peritonitis, opium must be administered in sufficiently large doses to give complete relief from pain, and it should be continued until all signs of peritonitis have disappeared. If the bowels do not then move spontaneously, a full dose of castor oil may be given, followed by an enema of warm water.

APPENDICITIS.

Morbid Anatomy.—Primary inflammation of the appendix may be catarrhal, ulcerative, or gangrenous. In the former case the cavity is distended by catarrhal or purulent inflammatory products; its walls are tense and thinned. Simple ulceration seldom follows: the inflammation subsides or the appendix sloughs. Localized ulceration more commonly depends upon the presence of a foreign body, and is often followed by perforation. In either case a peritonitis is developed which agglutinates the adjacent peritoneal surfaces. When perforation or sloughing has occurred, the resulting peritonitis may be local; but if it become general the inflammatory products are often encysted. Simple appendicitis may subside and leave no evidences save in adhesions of the appendix to the colon or pelvic wall.

Etiology.—Catarrhal appendicitis may attend typhlitis or develop without obvious cause. The ulcerative and gangrenous forms are quite invariably, and the catarrhal often, excited by foreign bodies, small masses of inspissated fæces, fruit seeds, foreign bodies, worms, gall-stones, etc. Traumatism is occasionally the exciting cause, and straining—as in lifting or jumping—is the etiological factor in about 20 per cent. of the cases. It is most frequent in young male adults from fifteen to thirty years of age.

Symptoms.—The earlier stages often pass entirely unnoticed. If any symptoms are present, they are either a slight localized pain and tenderness in the iliac fossa, or the symptoms just given as those of typhlitis without the sausage-shaped tumor.

In such cases it cannot be distinguished at first from typhlitis.

The symptoms which are diagnostic are, first, a sharp, sudden, intense pain localized in the iliac fosssa, but possibly extending to the navel, liver, or into the back. This is usually attended by nausea, vomiting, and chill. These symptoms mark the time of perforation or the advent of an acute peritonitis. Fever is the next most constant symptom, but the temperature is seldom above 103° F. and averages from 101° to 102° F. Micturition is often disturbed, and there may be complete retention, possibly dependent upon the opium given to control pain.

On physical examination a circumscribed tumor is detected, which is composed of inflammatory exudation. It is distinctly different from the tumor of typhlitis, being more oval, doughy, or fluctuating, and not sausage-shaped. Usually located in the iliac fossa, it may extend towards the median line or to the iliac crest. When the appendix has become adherent to the pelvic wall, it will be situated deeper, and best recognized by rectal examination. The tenderness of the parts renders an examination with an anæsthetic advisable in all cases. Fluctuation is a late rather than an early sign, and often can be obtained only by rectal examination. The tumor is dull as a rule, but gives a tympanitic note when covered by distended intestines, or when the contents of the abscess are partly gaseous. The inflammatory products become fluid, and abscess is formed in from two to four days. The purulent products are usually encysted, and seldom invade the peritoneal cavity unless rupture has occurred. The course of these cases is exceedingly acute. Over 50 per cent. of deaths occur in the first week.

Differential Diagnosis.—This is principally from simple typhlitis and perityphlitis. An early diagnosis is not always possible. Appendicitis, however, is usually insidious, and attended by few if any symptoms before its sudden and severe explosion. Then all the symptoms are intense, and the nervous shock is prominent. Typhlitis begins with mild but distinct symptoms of constipation, iliac lameness, and tenderness, and the sausage-shaped tumor, and there is no sudden and severe exacerbation until the condition is well established. When abscess has formed in appendicitis, rectal examination may differentiate the tumor from that of perityphlitis. Quite frequently the diagnosis cannot be made.

Prognosis. — Resolution may possibly take place, but is exceedingly rare. When perforation has occurred and is followed by general peritonitis, then death is almost inevitable, even when operative procedures are resorted to.

Treatment.—When an exact diagnosis of appendicitis can be made before perforation, antiphlogistic measures locally, and opium internally, may be employed with the hope of arresting or limiting the inflammation ; but my experience leads me to the opinion that it is better to make an explorative incision as soon as there is reasonable evidence that appendicitis exists. After the advent of sudden pain, shock, and other symptoms of perforation, surgical measures employed before general peritonitis has developed offer the only efficient means of relief.

PERITYPHLITIS.

Perityphlitis is an inflammation of the connective-tissue which attaches the ascending colon to the iliac fascia, rarely extending beyond the region of the cæcum. By some it is regarded as a form of peritonitis, and it has also received the name of *pericæcal abscess.* In nearly all cases the inflammation is propagated from the cæcum, the vermiform appendix, or the ascending colon. It may occur as a primary inflammation from traumatic causes.

Morbid Anatomy.—The ulcerative processes within the cæcum are usually accompanied by localized congestion of the peritoneum over it. Frequently the congested peritoneum is covered with a thin layer of partially organized lymph. The new connective-tissue formation binds together the appendix vermiformis and the cæcum, and attaches either or both of them to the adjacent parts. When typhlitic ulceration extends into this new tissue a perityphlitic abscess is formed. In rare instances recovery takes place without an abscess. If an abscess forms, its seat is in the cellular tissue between the colon and quadratus lumborum, or in the cellular tissue between the iliac fascia and the cæcum. Many perityphlitic abscesses are undoubtedly peritoneal abscesses. These abscesses are deep and irregular in shape, owing to the great resistance of the fascia in this neighborhood.

The pus may infiltrate the connective-tissue as far up as the level of the eleventh rib, and reach the under surface of the liver, or may extend as far down as the rectum. It may burrow and point near the anus, or it may make a direct external opening in the groin or loin, or, as most frequently happens, it may perforate the adjoining wall of the cæcum and be voided through the bowel and anus. After the pus has burrowed it may form a sinus or a series of sinuses which never become obliterated, although they grow smaller and smaller as time advances; fæcal matter may at times escape from such sinuses. In many instances a perityphlitic abscess opens into the bladder. The pus may escape through the skin of the thigh, or it may perforate the peritoneal sac and induce general and quickly fatal peritonitis. In most cases peritoneal adhesions prevent the opening of the abscess into the peritoneal cavity. When the veins are pressed upon by the abscess there will be more or less œdema of the extremity. In some cases the inflammation does not assume an intense or acute form, but is rather *sub-acute* in character, and then its area is limited, and the accompanying adhesions are firmer and more extensive.

Etiology.—Perityphlitis is usually the result of extension of inflammation from the vermiform appendix, or is secondary to a typhlitis. It may be due to the extension of tubercular, typhoid, or dysenteric ulcers in the cæcum, and to the lodgment of foreign bodies in the vermiform appendix.

Caries of the spine or of the pelvis has induced it. Traumatism is an occasional cause; it is rarely of spontaneous origin.

Symptoms.—In a few instances of perityphlitis, especially those supervening on typhlitis, there will be a history of colicky pains which radiate outward from the cæcal region, with more or less irregularity in the action of the bowels. There is pain in the thigh, accompanied by numbness and a sense of formication in the right lower extremity, due to pressure of the tumor upon the nerves. This pain is deep-seated and much increased by flexing the thigh upon the abdomen. Rigors and febrile movement are usually slight. In extensive perityphlitis the patient cannot raise the right thigh, either on account of the pain, or from interference with functions of the nerves from the pressure. When the abscess is of large size there may be œdema of the limb. The parts in the vicinity of the cæcum are very tender to pressure, and the patient usually lies on the right side, with his thigh semi-flexed so as to relax the psoas and iliac muscles. As the abscess increases in size, there is in adults constipation, and a tendency to vomit. In children the bowels are commonly loose, and pain in the stomach will have been an early and prominent symptom. When a perityphlitis arises as a typhlitis is disappearing, a painful tumor, more deeply seated than in typhlitis, will make its appearance.

Physical Signs.—*Inspection* reveals a tumor in the right iliac region, which may extend upward and to the left, as far as the umbilicus. In children the tumor is often elongated, reaching from the ramus of the pubis to the free border of the ribs.

Palpation.—If suppuration has occurred, palpation may show the existence of fluctuation over, and to the right of the tumor. In children these tumors sometimes have a brawny hardness. Careful manipulation shows the tumor to be deeply seated.

Percussion may give a tympanitic resonance on account of the gaseous distention of the cæcum, the tumor being behind this portion of the intestine. More often the percussion note has an obscure tympanitic resonance, for the cæcum is either tumefied, or contains fæcal matter.

Differential Diagnosis.—Perityphlitis may be mistaken for *typhlitis.* In typhlitis there will be a history of colicky pains, dyspepsia, irregular action of the bowels, and tympanitis, all of which are present before a tumor is developed; in perityphlitis a tumor is present *before* any other symptoms are developed. In typhlitis the pain is superficial and unaffected by the motion of the thigh, in perityphlitis it is deep-seated and increased by motion of the right thigh. There is no sense of numbness or formication in the right lower extremity in typhlitis, but this is more or less marked in perityphlitis. There is no evidence of suppuration (fluctuation), etc., in typhlitis, while evidence of fluctuation is present in perityphlitis as soon as suppuration occurs. The tumor of typhlitis is superficial and sausage-shaped, that of perityphlitis deep and irregular. A typhlitic tumor gives a flat percussion sound, a perityphlitic tumor an obscure tympanitic resonance.

A *psoas abscess* has no intestinal symptoms, and the purulent discharge is of a very different character, lacking the fæcal odor. *Renal* and *ovarian* tumors will not be mistaken for perityphlitis if the early history of the case is carefully analyzed.

Prognosis.—In the majority of cases in which the peritonitis is localized, and intestinal perforation does not occur, recovery takes place. If a perityphlitic abscess opens externally, or into the ascending colon, or if the pus burrows and points in the region of the thigh, buttock, or scrotum, the prognosis is more favorable than when it opens into the peritoneal cavity, or into the bladder. If chills, hectic, emaciation, and extreme exhaustion are present the prognosis is unfavorable. The average rate of mortality is about 25 per cent. If recovery from the primary attack occurs, there still may be narrowing of the intestine, or such alteration in the relative position of the parts that there will be more or less intestinal obstruction for the remainder of the individual's life. One of the most unfortunate sequelæ of perityphlitis is the formation of a fæcal sinus.

Treatment.—The first and most important thing, after aspiration has determined the character of the tumor and shown the presence of pus, is to make an incision into the abscess, cutting cautiously through the abdominal wall at the seat of the swelling. Free drainage must be kept up by means of a drainage tube. Previous to the evidences of suppuration, leeches may be applied over the tumor, followed by warm poultices. Absolute rest is all important to the successful management of these cases. Some prefer to open at once, *i. e.*, before fluctuation occurs. After an artificial or spontaneous opening, or after the occurrence of absorption, care must be taken to avoid any pressure upon that part of the intestine for some time. The exhaustion from the suppuration must be combatted by iron and the vegetable tonics. The diet throughout should be highly nutritious and easy of digestion. Opium should be employed when the least indication of peritonitis, local or general, appears, and laxatives and purgatives must not be administered until the reparative processes are well established.

INTESTINAL ULCERS.

The Duodenal Ulcer.—The round or perforating *duodenal* ulcer may be regarded as the analogue of the peptic gastric ulcer.

Morbid Anatomy.—This ulcer in its nature and appearance closely resembles the gastric ulcer, and its subsequent changes are the same. Its most frequent seat is the ascending portion of the duodenum, it is rarely found in its descending or transverse portions. Not infrequently duodenal and gastric ulcers coexist. The cicatrix which results from the healing of a duodenal ulcer may cause dilatation of the stomach, and of that portion of the duodenum between the cicatrix and the pylorus. This constriction and subsequent dilatation will induce chronic gastric and duodenal catarrh. In rare cases the vena portæ is completely obliterated by the constriction of a duodenal cicatrix, or the ductus communis may be occluded, thus giving rise to obstructive jaundice. Atrophy of the pancreas has resulted from occlusion of the pancreatic duct, and in this connection it may be mentioned that duodenal ulcers near the head of the pancreas rarely, if ever, perforate. Another result is the formation of a fistula communicating with the gall-bladder. A duodenal ulcer may perforate the duodenal walls into the peritoneal cavity and cause peritonitis, or having perforated the walls of the duodenum it may involve the liver, pancreas or gall-bladder, which becomes adherent to it at the seat of the ulcer. It may perforate directly outward at the seventh intercostal space, or indirectly outward after having first opened into the loose cellular tissue behind the bowel. Abscesses resulting from such perforation have burrowed through the mediastinum into the tissues of the neck and opened posteriorly near the shoulder-blade.

Etiology.—Compared with gastric ulcers, duodenal ulcers stand in the proportion of one to thirty. The cause of the round duodenal ulcer is the same as that of round ulcer of the stomach. An *embolus* obstructs a vessel, or blood is extravasated, necrosis results, and the action of the intestinal juices rapidly establishes the ulcerative process, just as the gastric juice causes a gastric ulcer ; an ulceration resulting from *burns* is much more liable to be duodenal than gastric. Duodenal ulcers occur more frequently in males than in females ; occasionally they are met with in children.

Symptoms.—The symptoms of duodenal ulcers are obscure, the first and only symptom, in many cases, being a sudden and fatal peritonitis. When perforation occurs, it ordinarily takes place after a hearty meal, or from efforts made in vomiting or defecation. The patient rapidly passes into a condition of collapse, the face becoming "Hippocratic," the pulse small or imperceptible, the extremities cold, and suppression of urine often precedes the fatal issue. A fatal gastric hemorrhage has been the only sign of duodenal ulcer. Pain is less pronounced than in gastric ulcer. The pain may be limited to the right hypochondrium, or it may be localized over the duodenum. It commences from two to four hours after eating. The location of the pain and the time of its occurrence, are the diagnostic signs of a duodenal ulcer. It gives rise to intestinal hemorrhages more frequently than to peritonitis. If jaundice occur with these symptoms, it aids in its diagnosis. If the pancreas is secondarily enlarged and indurated, there will be a tumor in the right hypochondrium.

Prognosis.—The prognosis is not as good as in gastric ulcers, for cicatrization is not so likely to occur. Duodenal ulcers are to be treated in the same manner as gastric ulcers.

Follicular Ulcers.—Though the morbid anatomy of this variety of intestinal ulcers is almost inseparable from that of enteritis (q. v.), its symptoms are distinct, and are better considered under a separate head.

Symptoms.—Following a long-continued intestinal catarrh, especially in cachectic subjects, where the diarrhœal discharges have been distinctly muco-purulent, and have alternated with natural fæcal discharges coated with blood and mucus, there will appear in the dejections transparent masses of mucus, looking like "boiled sago," and having the form of the conical-shaped ulceration of the follicles of Lieberkühn. With follicular ulceration there is great emaciation and marasmus. In children, where this form of ulceration is often extensive, death often results from inanition. If long continued, there may be tormina and tenesmus, while the stools become uniformly opaque, puriform and yellowish white. If recovery takes place, the resulting cicatrices give rise to obstinate constipation. Death may occur from perforation, peritonitis, or from marasmus.

Diffused Catarrhal Ulcers are never met with apart from acute enteritis.

Morbid Anatomy.—Acute intestinal catarrh may be accompanied by a suppurative process, which will cause the destruction of an irregularly circular portion of the intestinal wall, including the mucous, submucous, and often the muscular coats, and lead to perforating peritonitis. The margin of these ulcers is usually well defined. Their centres, which are irregularly depressed, contain a grayish shreddy mass; between the ulcerated patches the mucous membrane is congested. If cicatrization takes place rapidly, stricture of the intestine at the corresponding point may result. If cicatrization takes place slowly, portions of the intestine will be matted together in coils, and will become more or less adherent to the adjacent organs.

Etiology.—The etiology of catarrhal ulcers is the same as that of catarrhal enteritis. They are especially liable to be induced by foreign bodies and fæcal impaction. The condition occurs most frequently when an *acute* becomes ingrafted on a chronic intestinal catarrh. The symptoms, prognosis and treatment are identical with those of chronic enteritis.

Tuberculous Ulcers.—Under this head I shall consider the so-called "follicular" or *strumous enteritis.*

Morbid Anatomy.—The cæcum is the region which is most frequently the seat of these ulcers; next the lower portion of the ileum. The large and small intestines are often equally affected. Peyer's patches and the solitary follicles are the primary points of invasion. At first they become swollen and congested, hyperplasia of their lymphatic elements causing numerous projections on the mucous surface. These projections are small, gray, translucent, elastic nodules, "the gray miliary tubercle." They usually occur in isolated patches, but may become confluent over a considerable portion of the intestine. After a time these gray nodules become yellow, dry, and cheesy, increase in size and constitute the "yellow tubercle," and afterward soften and form ulcers.

The primary tubercular ulcer is a small, round, crater-like cavity, with indurated base and walls. Several primary ulcers in one Peyerian patch may be separated from each other only by thin septa, and then the patch presents a honeycomb-like appearance; or several follicles form an elevated patch, which ulcerates in points. Diffuse inflammation of the submucous tissue occurs in the vicinity of these patches. The villi are matted together at their base, and free at their apex. Tuberculous ulcers spread by the development of small fresh nodules in the walls of the blood-vessels, so that they extend transversely, sometimes forming girdles half an inch wide around the whole internal surface of the intestine. In this way the oval tuberculous ulcers *outside* of Peyer's patches have their long axis transversely. As these ulcers increase in size, they cause contraction and narrowing of the calibre of the intestine. These ulcers rarely cicatrize and rarely cause hemorrhage or perforation of the intestine. If a tuberculous ulcer extends to the serous coat of the intestine, the latter becomes thickened, reddened, and somewhat clouded, and is covered with a fibrinous exudation which mats the intestines together. It is at these points that the peritoneum is covered with minute tubercle granules. A more or less severe intestinal catarrh accompanies tubercular ulceration. The process may extend to the mesenteric glands, or these may be primarily involved, constituting what is called "tabes mesenterica."

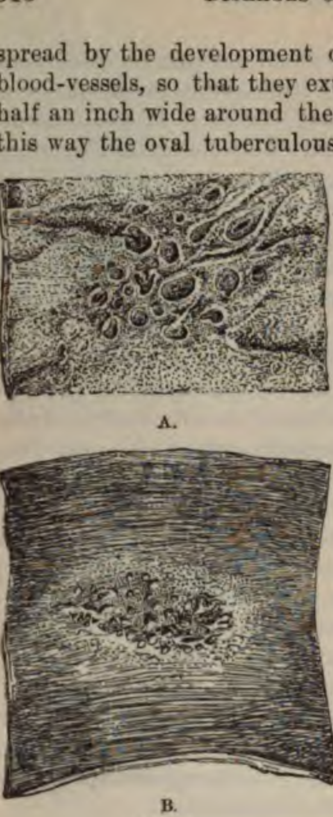

FIG. 65.
Tubercular Ulcers of the Ileum.
A. Mucous coat.
B. Peritoneal coat.

Etiology.—As the primary manifestation of tubercle it appears almost exclusively in children under five years of age, while tubercular inflammation of the intestines and mesenteric glands of adults is always *secondary* to tubercle elsewhere, especially pulmonary tuberculosis. The causes which in non-tubercular subjects would excite simple enteritis may in this class predispose to tubercular development.

Symptoms.—The symptoms of tubercular intestinal ulceration are never diagnostic. Diarrhœa is its most constant symptom. Pain in many cases precedes the diarrhœa, which consists of thin green mucus, or more rarely is yeasty in character. Blood is not often present in the discharges. An inordinate desire for food is often a prominent symptom. There is early tympanitic distention of the abdomen, which causes it to become round and protuberant, and this pot-belly presents a marked contrast to the wasted chest and limbs. Ascites is sometimes present, and enlargement of the abdominal veins is quite common. The general health is much impaired, and there is progressive emaciation, although there are intervals of apparent improvement. The sleep is disturbed, and muscular twitchings and convulsions occasionally occur in children. During the whole course of this disease there is a continuous rise of a degree or two in temperature. When the end is near, the diarrhœa which has continued through the disease in many cases gives place to obstinate constipation.

Physical Signs.—*Palpation* may reveal localized tenderness, especially about the cæcal region. The enlarged mesenteric glands may sometimes be felt through the abdominal walls, although tympanitic distention often so interferes with the examination that they cannot be detected.

Differential Diagnosis.—Tubercular disease of the intestine may be mistaken for *tubercular meningitis* and *tubercular peritonitis*. The abdomen is distended in tubercular intestinal ulceration and retracted in meningitis There is constipation in tubercular meningitis, and diarrhœa in intestinal ulceration. Vomiting, projectile in character, is a marked and constant sign of tubercular meningitis, while vomiting is rare in intestinal ulcers, and when present is retching in character. The pulse is slower than normal early in meningitis, while it is accelerated in tuberculous ulcer of the intestine. The pupils are normal in size in intestinal disease, and contracted or dilated in meningitis.

Tubercular *peritonitis* is often associated with intestinal ulceration. If peritonitis exists, the abdomen is more rigid than in intestinal ulceration, the tenderness and paroxysmal pain are much more severe, and emaciation is not so prominent a sign or so progressive. Enlarged cervical glands are more frequent with ulceration than with peritonitis.

Prognosis.—This is always unfavorable. It is essentially a chronic disease. Death may occur from exhaustion incident to the diarrhœa and marasmus, or from intercurrent tubercular complications.

Treatment.—The prophylactic treatment is similar to that of general tuberculosis. If the diarrhœa is copious and exhaustive, astringents with opium may be employed. Inunctions of cod-liver oil and iodine may be made over the abdomen, which should be covered with a flannel bandage. When pain is severe anodyne poultices may be applied locally to relieve it.

INTESTINAL HEMORRHAGE.

Intestinal hemorrhage may be a symptom of local or of general disease. The bleedings may be slight when they are capillary, profuse when they come from vessels of considerable size. Blood from the stomach which is passed with the dejections cannot strictly be regarded as intestinal hemorrhage.

Morbid Anatomy.—At the post-mortem of one who has died during or soon after an intestinal hemorrhage the intestinal mucous membrane may be found either hyperæmic or anæmic, according as the hemorrhage has been slight or profuse. The intestinal canal will contain dark grumous blood or small clots. If the hemorrhage is caused by ulcers in the intestine, coagula generally adhere to the ulcers, and the edges and base of the latter are suffused with blood. In a few instances the mucous membrane appears normal, especially when the hemorrhage is due to obstruction of the portal circulation.

Etiology.—Any disease in which there is extensive obstruction to the portal circulation may be a cause of intestinal hemorrhage; with the exception of intestinal ulcers, cirrhosis or atrophy of the liver is its most frequent cause. It is a very frequent attendant upon typhoid and dysenteric ulcerations. Foreign bodies and traumatism act mechanically in producing it, and it may be induced by powerful chemical and mechanical irritants acting directly on the intestinal mucous membrane. It occurs in waxy degeneration of the intestine, and may be caused by the rupture of an aneurism into the intestinal canal.[1] It frequently attends the development of intestinal cancer, and is a common symptom of internal hemorrhoids. It occurs in yellow fever and in the pernicious malarial fevers, and a vicarious hemorrhage may take the place of the menstrual discharge. Subsidence of the fiery-red tint of the face in erysipelas has been followed by remittent intestinal hemorrhage. Chronic constipation and the pressure of large tumors may cause it by impeding the venous return. It has occurred in a few cases of intestinal invagination and with embolism of the mesenteric artery. It is a common symptom in purpura hemorrhagica, and in that condition known as hæmophilia. Acute yellow atrophy and splenic leucocythæmia may be attended by intestinal hemorrhage. Those affected with a scorbutic or syphilitic taint are always predisposed to it. It occurs oftener in men than in women. The aged are subject to a passive intestinal hemorrhage, without apparent cause.

Symptoms.—If the hemorrhage is slight it frequently passes unnoticed, for it gives rise to no symptoms. When it occurs in dysentery, typhoid and yellow fevers, the symptoms which attend it will vary with the amount, number and persistence of the bleedings as well as with the stage of the disease in which it occurs. Its color will be determined by its seat and the length of time it remains in the intestinal canal. When it comes from the upper portion of the intestinal tract, it is of a dark red color, grumous and tarry, when its seat is near the rectum it is of a *bright* red color and fluid. The tarry color and consistence given to the fæces by the presence of blood is called "melæna."

The symptoms which attend a large intestinal hemorrhage are a feeling of faintness, a feeble pulse, a deadly pallor, ringing in the ears, bright flashes before the eyes and coldness of the surface, followed by syncope which may end in death. Immediately following or preceding these symptoms there may be a large discharge of blood from the bowels. In most instances, after a few hours the patient recovers from the shock of the hemorrhage. There may be abdominal pain at the time of the hemorrhage, accompanied by a sensation as if warm water was being poured into the abdominal cavity. A continuation or succession of large hemorrhages will rapidly give rise to the characteristic signs of anæmia.

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[1] Bamberg places diphtheritic inflammation of the intestines as its second most frequent cause, dysentery being the first.

If the normal dejections are coated with blood, the origin of the hemorrhage is within the large intestine. When the hemorrhage is from intestinal ulcers, pus will be mingled with the blood. Intestinal hemorrhages may cause death when there has been no discharge of blood from the anus.

Differential Diagnosis.—The diagnosis of intestinal hemorrhage is not difficult, it is usually made by the patient. Its source can only be determined by a careful history of the case. In acute infectious diseases its seat is in the small intestine; in chronic disease it usually has its seat in the large intestine. When diseases of the liver can be excluded, local causes in the rectum should be sought for.

Prognosis.—The prognosis will depend upon the seat and cause of the bleeding. In yellow, typhoid and malarial fevers, in dysentery and cirrhosis, it is always unfavorable. Arterial hemorrhages are far graver than capillary. The condition of the patient at the time of the hemorrhage influences the prognosis. It is less dangerous in vigorous than in feeble and anæmic subjects. Death may occur from a single large hemorrhage, or from the asthenia produced by repeated smaller bleedings.

Treatment.—The first, and perhaps the most important, indication is absolute rest. The patient should be kept in bed in a cool, quiet room, and should not be allowed to change the position of the body. Peristalsis must be arrested by opium; fluid nourishment should be given at short intervals and in small quantities. Cold compresses or ice-bags should be placed over the abdomen, and styptics should be administered by the mouth or by the rectum, according to the supposed seat of the hemorrhage. Alum-whey, perchloride of iron, acetate of lead, tannin, or any of the hæmostatics may be given by the mouth or rectum. Ergot internally or ergotine hypodermatically should be administered in sufficient quantities to produce the physiological effect of the drug. When the bleeding has been excessive, stimulants—alcohol, ether or musk—should be administered, and it may be necessary to practice transfusion. Everything taken by the patient by the mouth or rectum must be ice-cold. The use of the mineral acids as beverages has been advocated. No irritating food or drinks should be allowed for many days after all signs of hemorrhage have ceased. If intestinal hemorrhage occurs in one who gives the evidence of purpura or hæmophilia, the salts of iron or potassa, combined with vegetable acids, are serviceable.

INTESTINAL OBSTRUCTION.

By the term intestinal obstruction is meant a narrowing, closure, invagination, or twisting of some portion of the intestine, so that its calibre is diminished and the passage of its contents retarded or prevented. In all cases there is a mechanical impediment situated either within or without, or in the wall of the intestine. When a foreign body prevents the onward progress of the contents of the intestines, by becoming an actual plug, the obstruction is said to be *within* the intestine ; when some abdominal tumor presses on, and narrows almost to closure, some portions of the bowel, it is said to be *without* the intestine ; and when a deep ulcer, involving the whole wall of the intestine, contracts and forms a stricture, or other changes take place in its walls which diminish or obliterate its calibre, it is said to be *in* the intestinal walls. It is impossible to separate the etiology of intestinal obstruction from its morbid anatomy, and they can best be considered together.

Morbid Anatomy.—I shall first consider intestinal obstruction from causes which have their seat *in the wall* of the intestine. In this group stricture and intussusception are the chief lesions. Intussusception, or invagination, consists in the descent or prolapse of a portion of the intestine into that which immediately succeeds it, like a glove-finger drawn partially within itself. It always occurs from above downward. In this condition the two mucous and the two serous surfaces are opposed to each other, so that a section exhibits three cylinders, one within the other, called respectively—the receiving layer, the entering layer, and the returning layer. Sometimes the intussusception is lateral and partial. The mesentery belonging to the invaginated portion lies between the middle and internal layers. By the traction of the mesentery, the central part of the intestine is curved laterally. Invagination which occurs during the last moments of life is unimportant ; it is chiefly found in children who die of brain disease. The occurrence of intussusception is regarded by most as the result of the sudden passage of a spasmodically contracted portion of the intestine into a flaccid or paralyzed portion. An exaggeration of the normal peristaltic wave can produce it, especially when there is great gaseous distention of the intestine. Some observers state that it is the paralyzed portion which is thrust forward by a sudden increase in peristaltic action of the intestine.

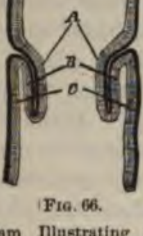

FIG. 66.

Diagram Illustrating Intussusception.

M. Mucous surface of intestine.
A. Entering layer.
B. Returning layer.
C. Receiving layer.

Its most frequent seat is in the neighborhood of the ileo-cæcal valve. It may occur in any portion of the intestine. The whole colon may be filled by the ileum, and the latter may even project outside of the anus. It is more common in the young; 50 per cent. of all the cases occur in childhood. Males are more subject to it than females.[1] The mucous membrane of the invaginated portion is intensely congested, its villi are swollen, softened, friable, and sometimes united by a pseudo-membrane. The single and the agminated glands are hypertrophied, infiltrated with fluid, and ulcerated at the centre. The sub-serous cellular tissue is infiltrated with blood, and is ecchymotic. The intestine above the obstruction, when the latter has existed for some time, is dilated. In many cases there is more or less twisting besides the invagination. Sometimes blood extravasates into the mucous tissue and into the mesentery. The invaginated portion may become gangrenous, slough, and be discharged per rectum. During the sloughing the peritoneal cavity may be opened.[1] In the neighborhood of the obstruction the mucous membrane may be the seat of intense catarrh. Sometimes the intussusception is restored. Polypoid growths in the rectum or sigmoid flexure, besides inducing intussusception, may themselves cause intestinal obstruction.

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[1] In the Croonian Lectures, 1857, intussusception appears to be ileo-cæcal in 50 per cent., iliac in 28 per cent., jejunal in 4 per cent., and colonic in 12 per cent.

Other common causes in the intestinal wall which induce obstruction are cancerous and non-cancerous stricture. These diminutions in the calibre of the intestine may be due either to contraction of cicatrices, or to infiltration of the intestinal walls. Such obstructions are developed gradually, and are rarely complete. Intestinal stricture may be merely a thin fold of mucous membrane, or four or five inches of intestine may be included. Above the stricture there is well-marked dilatation and hypertrophy ; below, atrophy of the intestine. The accumulation of matter above a stricture often leads to ulceration and perforation. About seventy-five per cent. of all strictures are in the large intestine, and most of these are in the rectum. About half of all intestinal strictures are of cancerous origin. Congenital stricture is a surgical disease.

Intestinal obstruction may, *secondly*, have its cause *outside* the wall of the intestine. Folds of the intestine may be caught in bands of adhesion formed by an old peritonitis, or may be forced through openings in the abdominal walls and become strangulated, causing internal or external hernia. The small intestine is strangulated by herniæ, diverticula, and bands of new formation, more frequently than the large intestine. Internal strangulation occurs oftener in men than in women. It may occur at any age, although it is met with most frequently in those about thirty. *Volvulus* or twisting of the intestine may occur, so as to produce intestinal obstruction ; the mesentery or a coil of intestine may be the axis about which certain parts revolve. Again, a coil of intestine may, with another, form a knot. Both of these occurrences are most frequent about the sigmoid flexure. An abnormally relaxed mesentery predisposes to it. Half a turn is sufficient to cause obstruction. In the old the sigmoid flexure may become twisted, by shrinking of the mesentery. Any abdominal tumor may induce by pressure more or less diminution in the calibre of the intestine and intestinal obstruction.

The *third* class of causes which give rise to intestinal obstruction are situated *within* the intestinal canal. Under this head are included obstruction from gall-stones, impacted fæces and foreign bodies of various kinds. Gall-stones are most frequently impacted in the ileum near the ileo-cæcal valve and in the duodeno-jejunal region. The larger and thinner the calculus, the more liable is obstruction to occur. In some cases gall-stones cause a fatal obstruction, but usually the obstructions continue only for two or three days. Gall-stones of immense size have been found completely plugging an intestine. These obstructions rarely have their seat in the large intestine and are met with more frequently after fifty years of age, and in females oftener than in males.

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[1] In one instance the whole of the cæcum and ascending colon sloughed away, and recovery took place.—*Guy's Hosp. Reports.*

Hardened fæcal masses mixed with the phosphate of magnesia and lime, concretions of chalk and magnesia, ammonio-magnesian phosphate, inspissated mucus, and large oval masses consisting chiefly of cholesterin, are among the commoner forms of the so-called "*enteroliths.*" Concretions of hardened fæces rarely cause fatal obstruction. The list of foreign bodies which have caused intestinal obstruction is very large, but they all act in the same way.

The changes which follow all forms of intestinal obstruction, are dilatation of the intestine above, and atrophy below, the seat of the obstruction. The peritoneum over the site of the obstruction is the seat of acute or chronic peritonitis. Gangrene may occur at the point of greatest pressure. There is always more or less extensive intestinal catarrh in every case of intestinal obstruction.

Symptoms.—The symptoms of intestinal obstruction vary with its seat, extent, and cause. The symptoms which are common to all varieties are obstinate constipation, and vomiting. The matters vomited consist first of the contents of the stomach, then mucus, and after a time bile and stercoraceous matter. The accompanying pain varies in character and intensity: sometimes it resembles that of a colic, sometimes that of peritonitis. When the obstruction is low down there is tympanitis. When the upper part of the small intestine is obstructed there is hiccough. Accompanying these symptoms there is prostration and often collapse. The skin is cold and the countenance assumes an Hippocratic expression. In a few cases, portions of the intestine that have become invaginated can be seen projecting from the anus. A careful analysis of these prominent symptoms will often enable one to determine the seat and character of the obstruction. When gall-stones and other foreign bodies or intestinal worms obstruct the intestine, constipation will come on suddenly.

In intussusception, constipation does not occur suddenly, for thin liquid fæces are able to pass the narrowed orifice. When invagination occurs in the small intestine, the discharges are accompanied by a copious flow of blood. But when the lower bowel is invaginated the blood is mingled with the discharges, and they are dysenteric in character. When intussusception becomes chronic, diarrhœa may develop and become exhausting, especially in children. When sloughing occurs gangrenous masses mingled with mucus and blood will be discharged.

If thin bands of fæces are passed, it indicates the existence of an incomplete stricture of the large intestine. Slow but steadily increasing constipation, the bands of the fæces becoming gradually smaller, indicates the growth of a stricture or the enlargement of a tumor compressing the intestine. If obstruction occurs suddenly, the rectum retains its contractile power and is empty, while if the obstruction comes on gradually it is patulous. Vomiting is present in most cases; when the obstruction is high up the vomiting is bilious, and occurs within an hour or two after its occurrence; when near the cæcum, the obstruction is accompanied by fæcal or stercoraceous vomiting; when low down, two or three days may elapse before the vomiting occurs. In the so-called chronic forms of obstruction, vomiting occurs at intervals, and is more persistent the higher the obstruction. Copious stercoraceous vomiting is evidence that the obstruction is at the ileo-cæcal valve.[1] In children vomiting occurs very readily, and, with the pain, is the first well-marked sign of the obstruction. When gall-stones are lodged high up, vomiting comes on early and continues until death occurs, or the stone is dislodged.

Enteroliths usually give rise to typhlitic symptoms. The pain which accompanies or precedes the vomiting is colicky or paroxysmal at first, afterward it becomes constant and severe. Sometimes patients can locate the spot where the pain originates, at other times it resembles a stitch in the side. When the constriction has come on slowly the pain resembles that of ordinary colic. If the small intestine near the cæcum or jejunum is strangulated, there is pain in the region of the umbilicus. When the colon is obstructed the pain is located at the seat of the obstruction. Pain in the groin or in the left iliac fossa indicates obstruction at the sigmoid flexure. There is usually no tenderness at the onset. When tenderness, local or diffuse, is extreme, peritonitis is indicated.

In all cases except those where the obstruction is near the duodenum, the abdomen gradually becomes distended from gaseous accumulation in the intestine above the seat of the obstruction,—*tympanitis*. It occurs first near the obstruction. When twisting of the intestine occurs the portion constituting the loop often forms a tympanitic tumor. The tympanitic note is readily elicited as the abdominal muscles become rigid. Vomiting relieves the tympanitis to a greater or less degree. The higher the obstruction the greater the relief from the vomiting.

Intestinal cancer, large gall-stones, fæcal masses, and other abdominal tumors which gradually compress the intestine may usually be accurately located and definitely mapped out. Invagination sometimes gives rise to a soft, sausage-shaped tumor, which can be distinctly felt, especially when it occurs at the cæcum. In intussusception of the small intestine a central tumor may often be felt near the umbilicus. If invagination occurs low down, the slit-like opening in the invaginated portion may be felt per rectum. Fæcal tumors along the line of the colon are quite distinct on *palpation*, and, while firm, continued pressure gives no pain, their situation and form may gradually be altered; attention to these points will prevent mistaking fæcal for malignant tumors.

Percussion over these tumors elicits dulness corresponding to their extent. If the obstruction is in the duodenum, there is an almost total suppression of urine; when in the jejunum or ileum, there is marked diminution; but when the lower bowel is occluded the flow is abundant and limpid. In many cases of intestinal obstruction there is a sudden shock at the time of its occurrence, similar to the shock in peritonitis; this is often followed by symptoms of collapse. During the whole course of intestinal obstruction, the temperature is rarely elevated. If the obstruction is complete, the face becomes "drawn," the extremities and the surface cool, the pulse rapid and small, the patient lies on his back with the knees flexed, and carefully avoids movements which induce pain and vomiting. Later on symptoms of collapse are developed, the breathing becomes rapid and superficial, thirst is intense, the voice is husky, the pulse becomes imperceptible, and the patient dies as in collapse, from peritonitis which will be found in most cases after death. If the obstruction is in the small intestine hiccough is a constant and annoying symptom. The mind is undisturbed to the last. In slowly developed intestinal obstruction, as in cancer-strictures and compression from tumors, the patient loses flesh and strength, becomes anæmic and melancholic, and the countenance bears the aspect of one suffering from malignant disease. Torsion or twisting of the intestine is attended by acute and rapidly fatal enteritis.[1] In these cases all the symptoms of severe enteritis are present.

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[1] Brinton suggests that a double current is produced; the intestinal contents are propelled along the wall of the intestine until they meet the obstruction, and then a return current passes up in the centre of the intestine. This retrograde movement continues until the vomited matters are the same as those at the seat of the stricture.

Differential Diagnosis.—It is important in every case to determine the *seat* and cause of the intestinal obstruction. *Intussusception* occurs most frequently in children, it begins suddenly with intense colicky pains, and there is blood mingled with the scanty mucous discharges. Fæcal vomiting occurs early, and there is commonly a distinct tumor, firm pressure on which sometimes relieves the pain; in a few cases the invagination may be determined by a rectal examination. The patient rapidly passes into collapse. When this train of symptoms occurs suddenly in a child, previously healthy, and without appreciable cause, intussusception may be suspected.

Internal hernia occurs suddenly. The pain is fixed at one point and paroxysmal in character, fæcal vomiting comes on after a few hours, accompanied by obstinate constipation and rapidly developing tympanitis; in a day or two the patient may pass into a state of collapse. The large intestine is usually empty. In a few cases the patient will have "felt something give way in the abdominal cavity." Its symptoms resemble those of an external strangulated hernia and intussusception combined.

Twisting, *volvulus*, and the sudden incarceration of loops of the intestine under bands of adhesion or diverticula are attended by similar symptoms.

Foreign bodies usually have their seat in the cæcal region and give rise to typhlitic tumors. When gall-stones are the cause of the obstruction there is usually a history of hepatic colic, and the seat of the obstruction is high up; this will be indicated by the vomiting and urinary suppression.

Obstruction from cicatrices, and from the pressure of tumors is of slow growth, there will be the history of frequent attacks of constipation, gradually increasing in duration and severity, coils of distended and displaced intestine are easily detected, the centre of the abdomen has a doughy feel, and a tumor can often be made out.

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[1] Bristowe supposes the enteritis may occur first, may weaken the parts, and that the volvulus is a secondary phenomenon.

Intestinal obstruction may be mistaken for *colic* (or *enteralgia*) *peritonitis, external hernia, acute poisoning* (as from arsenic, antimony, etc.), *hepatic* or *renal colic*, and *enteritis*. In *colic* the discharges from the bowels will be normal, or there may be diarrhœa. In intestinal obstruction there is obstinate constipation. The pain of colic is of short duration and is usually relieved by pressure, while in intestinal obstruction the pain is persistent and as a rule not relieved by pressure. Fæcal vomiting, tympanitis, and symptoms of collapse are present in obstruction and absent in colic.

Peritonitis is attended by a rise in temperature, by great tenderness on pressure, by a tense, hard, wiry pulse, and by rigidity of the abdominal walls; while obstruction, if it begins with colicky pains, is soon attended by fæcal vomiting, the pain is localized, there is a sub-normal temperature and more distention of the abdomen.

Internal hernia may be confounded with *femoral* or *inguinal hernia*; a careful examination of the inguinal regions and the history of the case are usually sufficient to establish a diagnosis.

In cases of *acute poisoning* there will be evidences in the mouth and pharynx of the action of an irritant poison; the gastric symptoms, especially the sense of heat in the epigastrium, will be marked; there will be diarrhœa, no fæcal vomiting, no tympanitis, and the vomited matters will contain traces of the poison.

In *hepatic colic* the pain is persistent and radiates from the region of the gall-bladder to the back. There is no fæcal vomiting and no tympanitis, but the stools are clay-colored, and the calculus may be detected in the fæces after the attack ceases. The urine contains bile, and if the attack is prolonged jaundice occurs.

In *renal colic* the bowels are normal, the pain shoots from the back down the ureter to the end of the penis, and the testicle on the affected side is retracted. Relief immediately follows the passage of the calculus into the bladder, which is followed by a copious flow of bloody urine; in intestinal obstruction there is no hæmaturia, no symptoms referable to the urinary organs, the bowels are constipated, and there is tympanitis and fæcal vomiting.

Enteritis is distinguished from intestinal obstruction by copious mucous discharges from the bowels, by the rise in temperature, and the absence of fæcal vomiting, excessive tympanitis, and the symptoms of collapse.

Prognosis.—The length of time for which an intestinal obstruction may exist before causing death varies with its seat and its character; a weak child may die in eight or ten hours from the shock of intussusception, and an adult whose intestine is gradually being occluded by the presence of some slow-growing tumor, may live for months. As a rule, the nearer the stomach the obstruction, the more rapidly death ensues. Volvulus, strangulation, internal hernia of the small intestine, and obstruction by large gall-stones and enteroliths induce death more rapidly than stricture, compression, and intussusception, especially of the large bowel. Intussusception may be recovered from when a gangrenous process throws off the invaginated portion, and it is possible for it to slip back into its normal relations. The prognosis is favorable in intussusception when a portion of the invaginated bowel is discharged. It is better, the lower the seat of the obstruction. Of all forms of obstruction, fæcal tumors are the least grave. The complications of intestinal obstruction are enteritis, with or without perforation, septicæmia, phlebitis, ulceration and gangrene within the canal, and perforation of the intestine above the stricture. After recovery from the primary obstruction, the attending peritonitis may cause permanent constriction of the intestine. Another sequela is the formation of internal fistulæ.

Treatment.—Whenever there is reason to suspect intestinal obstruction of a non-fæcal origin, free catharsis should be avoided. It is better not to relieve a simple constipation, than to attempt to force fæces through an internal hernia. Whatever may be the seat or character of the obstruction, the therapeutical indications are the same, whereas a knowledge of the site and variety of the obstruction is demanded before surgical interference should be resorted to. Rest is demanded in every case; hence opium is to be given in sufficient quantities to relieve pain. The more sudden and severe the onset, and the more urgent the symptoms, the more serviceable is opium. The condition of the patient alone regulates the quantity to be administered.

Nourishment should be given per rectum in the form of defibrinized blood, and peptonized fluids. Each injection should contain from three to five grains of chloral, to retard decomposition. Ice may be given to relieve the thirst. If the tympanitis is excessive, it may be relieved by aspiration, or by the introduction of a tube into the colon. Instead of ice, warm compresses seem to relieve the pain and soreness, but they have no other value. There is no objection to saline laxatives in stricture or in compression, where, though not wholly occluded, the intestine is gradually narrowing, and only a small opening remains through which liquid fæces can pass.

When the obstruction is fæcal, opium should not be given; the bowels should be acted on by those drugs which produce copious watery evacuations without drastic action; at the same time the rectum must be emptied by mechanical means, or by enemata of warm water and glycerine. Much patience is often required to remove these fæcal obstructions. The most efficient method of mechanically overcoming obstruction is to make large injections of warm water through long rubber tubes, which must be inserted as far as possible. In order, however, to overcome by enemata an obstruction situated high up, the patient must be brought under the influence of ether or chloroform, and during the administration of the enema careful manipulation of the bowels must be practised. Taxis is to be practised according to the rules which surgery lays down for the reduction of hernia. Instead of warm water, air and gas have been injected in order to distend the intestines. Whatever injection is employed, it should be thrown in very slowly, and in very large quantities. It has been suggested to develop gas inside the patient by successively injecting solutions of soda bicarbonate and of tartaric acid ; rupture of the intestine may result from such a procedure. Before injections are given or taxis practised, it is well for the patient to take a prolonged and moderately hot bath. As an aid to the reduction of an intestinal obstruction, the hips may be elevated or the patient may assume the knee-elbow position. The use of the constant current is advocated by some, to produce active peristalsis ; this must not be resorted to unless there is no danger of exciting peritonitis by the active peristalsis. If the long tube is used for the purpose of giving injections, it must be introduced with the utmost care, for perforation has been caused by its careless introduction.

If at any time symptoms of collapse come on, alcoholic stimulants, musk, ammonia, etc., should be freely administered. As regards surgical measures, colotomy and laparotomy are the proceedings which have been proposed. The mortality after laparotomy is not exactly known, some statistics showing 68 per cent., others 73 per cent., and still others 75 per cent. of deaths. Laparotomy is especially applicable in acute intussusception, and should be performed with as little delay as possible. The dangers from colotomy seem to be less than those of any other operation. The statistics of lumbar colotomy exhibit the low rate of 33 per cent. of deaths. Laparo-enterotomy has a mortality-rate below sixty, but concerning laparo-colotomy, laparo-typhlotomy, etc., the number of operations is too small to give reliable statistics.

WAXY DEGENERATION OF THE INTESTINES.

Statistics show that after the kidney, spleen, and liver, the intestines are most frequently the seat of waxy degeneration.

Morbid Anatomy.—The primary seat of amyloid change in the intestines is in the arterioles. The small intestine is more often involved than the large. The mucous membrane is pale, shining, and slightly œdematous ; on the application of the iodine test small maroon colored spots appear in the villi, where the earliest changes occur ; later the muscular coat is involved, and finally the entire wall of the intestine is fused into a homogeneous mass. Peyer's patches are less affected than the surrounding tissues, but there is annular infiltration about the solitary glands.

Etiology.—Its causes are all those conditions which predispose to waxy changes in other organs. It is usually a late complication of waxy kidney and liver.

Symptoms.—Its symptoms are masked by those of waxy liver and kidney, with which it is always associated. When *general* amyloid degeneration of the entire intestinal canal exists, the nutritive disturbances are great ; exhaustion, emaciation, and anæmia are more marked than in any other condition. The countenance, the appearance of the skin, and the other constitutional symptoms are identical with those of waxy liver and kidney, *but in addition to these there is a serous diarrhœa* which is persistent and exhausting.

Differential Diagnosis.—The diagnosis is made by the presence of diarrhœa associated with the evidences of waxy changes in other organs.

Prognosis.—This is very unfavorable; more so than when the amyloid change is confined to other organs, for it indicates that the changes which usually precede a fatal termination have already occurred in those organs.

Treatment.—The treatment is altogether palliative; the diet should be restricted to meat and milk taken in small quantities and at short intervals. In addition to the general constitutional treatment of waxy degeneration, the diarrhœa may be checked by the vegetable astringents—hæmatoxylon, tannin, and catechu, or when these fail, the mineral astringents can be given. Iodide of potassium and iron will be found especially beneficial in these cases.

CANCER OF THE INTESTINE.

Carcinoma is the most common variety of intestinal neoplasm. It is less frequent than carcinoma of the stomach, and is almost always primary. In rare instances, it may be secondary to cancer of the peritoneum, uterus, or bladder. It almost exclusively affects the large intestine; the rectum is its most frequent seat, then the anus, the cæcum, the sigmoid flexure of the colon, and lastly the duodenum and jejunum.

Morbid Anatomy.—The primary development of intestinal cancer is commonly in the mucosa and extends into the submucous connective-tissue; the infiltration extends in a ring around the intestine. Sometimes its primary development is in the epithelium of the follicles. It may involve an inch or three or four inches of the intestine. In any case it causes more or less diminution in the calibre of the intestine; the intestinal wall becomes infiltrated and ulceration may be established, which will destroy the mucous membrane covering the cancerous mass, and temporarily remove the intestinal obstruction. Frequently cancerous masses project from the anus looking like cutaneous growths about its margin, or they may project from the anus in the form of fungous masses.

Before ulceration occurs scirrhus cancer presents a smooth, nodulated surface; encephaloid is soft, vascular, and often forms a tumor or series of tumors projecting into the intestine; these tumors are round, lobulated or villous. Often where the upper part of the rectum seems to be the seat of cancer, the disease will be found to have its seat at the sigmoid flexure, which has been pushed down into the pelvis, as the result either of the obstruction which it has caused, or of its own weight.

When ulceration occurs, fungoid masses may spring up upon the elevated surface and lobulated tumors may rapidly develop, or a smooth excavation may be formed, with hard, well-defined edges. Cancerous ulcerations may extend through the intestinal walls, and cause peritonitis or fæcal abscess, or establish communication with the bladder, urethra, uterus, vagina, or with other portions of the intestine. Scirrhus, more often than either of the other varieties, produces stricture of the intestine. In a few cases both large and small intestines are studded with small cancer-nodules, whose favorite locality seems to be the Peyerian patches. In any form of cancer, especially scirrhus, there may be great distention of the intestine above the seat of the cancer. There may be catarrh of the intestinal mucous membrane above and below the seat of the cancer. In cancer of the rectum the disorganization may be so rapid and extensive that dilatation occurs at the site of the cancer. Stricture, hemorrhage, perforation, fistulæ, and matting together and deformity of the organs in the neighborhood, are common pathological sequelæ of intestinal cancer. The neighboring lymph glands are always more or less involved. If cancer commences outside of the intestine and extends inward, it will be most extensive along the line of the attachment of the mesentery. The loose tissue around the rectum, cæcum, or duodenum may be so extensively infiltrated that the intestine within is merely a narrow rigid channel.

Etiology.—Intestinal cancer is rare before thirty, and more frequent between forty and sixty. Sex has no marked influence over its development; statistics give cancer of the rectum as occurring three and one-half times as often in males as in females. Its etiology is obscure; cancer of the colon seems to develop most frequently in the cicatrix of an ulcer or after traumatism.

Symptoms.—The symptoms of intestinal cancer vary with its seat. It comes on insidiously, with vague abdominal pains, a sense of unrest, and a marked decline in health and strength.

Duodenal cancer simulates hepatic and gastric cancer; it is often attended by coffee-ground vomiting coming on several hours after taking food. Sometimes there will be jaundice from pressure of the tumor on the common bile ducts. The tumor may be felt near the cartilage of the right tenth rib.

Cancer of the *cæcum* is attended by pain in the right iliac fossa and a tumor will be felt in the region of the cæcum, usually much larger than the cancerous mass and formed by the accumulation of fæces above the stenosed portion. Manipulation in these subjects causes movement, and diminution in size of the fæcal tumor, leaving distinct the cancer nodule, which is tender. In colloid cancer of the cæcum, the tumor is large, hard, and smooth. In most instances there is "tympanitic dulness" on percussion, over the tumor.

Cancer of the *rectum* first causes the symptoms due to a stricture, the bowels are constipated and the stools are not cylinders, but narrow bands. "Sacral" pains darting down the limbs, of a stabbing, lancinating character, giving rise to most intense suffering, are often present. When the bowels move, there is a sensation as if the parts were being burned, accompanied by more or less tenesmus. The bowels usually are at first constipated, but sometimes an irregular diarrhœa is present from its commencement; later in all cases, there is diarrhœa, the thin stools containing blood, pus, mucus, and shreds of sloughing and gangrenous matter. The invasion of the sphincter is followed by loss of power to retain the fæces, and then a brownish, watery, offensive fluid continually oozes from the anus. Communications with the vagina or urethra are followed by the escape of liquid fæces through these channels. A physical examination of the rectum (the patient being ether-ized) reveals numerous hard nodular masses, with a cartilaginous feel, or a soft, fungoid, friable mass having a hard base. A portion of the mass removed and examined microscopically, will usually decide its character. At times, an irregular, angry red, fungoid mass protrudes from the anus, and on inspection it is readily recognized as cancer. The finger may detect a septum thrown across the gut, or, in colloid disease, a large, round, smooth tumor projects forward, to occlude the rectum. The symptoms of epithelial cancer of the rectum are the least urgent and serious in appearance of all forms of rectal cancer. It may exist for a long time without producing either constipation, pain, or cachexia.

The prominent symptoms, which are common to *all* varieties of intestinal cancer, are pain, cachexia, constipation, and the presence of a tumor. As soon as the cancerous development has reached sufficient size to cause pressure, there is constant pain, which may be dull, vague, and dragging in character, or sharp and lancinating. In upper rectal and sigmoid cancer, the seat of pain is in the left iliac fossa and loins; in lower rectal cancer, it is in the loins, upper part of the thigh, and sometimes in the testes. In other situations the pain is at the site of the cancer. A cachexia usually develops with the commencement of the cancerous development, accompanied by emaciation, loss of strength and flesh; the skin is dry, "scaly," and assumes the dirty greenish "cancer color," the hair and nails become harsh and dry, and easily split. With the growth of the cancerous mass, exhaustion and cachexia gradually and steadily increase, and in some cases are the direct cause of death. As with cancer elsewhere, the disease may run its entire course without pain, anæmia, or marasmus. Constipation is the rule: at first there is flatulence, nausea, and vomiting; later these symptoms vary according to the locality of the obstructions, as, for example, if the cancerous growth is high up in the rectum, or at the sigmoid flexure there is marked distention of the colon. A free evacuation of the bowels temporarily relieves the distress; diarrhœa may alternate with the constipation, attended by rapidly increasing exhaustion. In some instances intestinal cancer is attended only by the symptoms of intestinal obstruction. Cancer high up in the colon is more often attended by diarrhœa than in the other localities. In long-standing cases of intestinal cancer, the lymphatic glands in the neighborhood of the cancer will be found enlarged, and there will be occasional intestinal hemorrhages, dropsy, and thrombosis of the neighboring veins. The discovery of a tumor along the line of the intestine is essential to the diagnosis of intestinal cancer. When it is developed in the ascending and descending colon and cæcum, the tumor is always felt in the normal position of the intestines, but when it is developed in the small intestine and transverse colon, the mobility of the parts and the weight of the tumor may cause it to occupy an abnormal position.

Differential Diagnosis.—Cancer of the duodenum cannot at first be distinguished from *hepatic* or *gastric* cancer; later on, however, the situation of the tumor, the character of the vomiting, and the time of its occurrence after meals will often enable one to recognize its seat and character.

Cancer of the *pancreas* cannot be distinguished from that of the duodenum. *Abdominal aneurism* may be distinguished from a pulsating duodenal cancer by the alteration which it causes in the pulsation of the femoral artery.

Cancer of the intestine is to be distinguished from *floating kidney* by the absence of the cancerous cachexia in the latter, and from the fact that the kidney tumor is *behind* and the cancer tumor in *front* of the intestine. Cancer may be distinguished from *enteritis, colic* or *intestinal ulceration* by the cachexia, tumor, and constipation. The age of the patient and a history of gradual development will aid in the diagnosis. The diagnosis of cancer of the rectum from other growths in it, or from *proctitis* or *hemorrhoids*, is made by a digital and ocular examination of the rectum and by a microscopical examination of a portion of the mass.

Prognosis.—The prognosis is always unfavorable. When the disease is situated in the rectum or at the anus, surgical interference may prolong life. After it is possible to recognize the existence of cancer of the intestine a fatal termination will generally be reached within a year.

Duodenal cancer gives rise to more distressing symptoms, and is more rapidly fatal on account of its situation, than any other variety. Death may result from exhaustion and anæmia ("cancer marasmus") from small hemorrhages, or from a single large hemorrhage, from rupture of the intestine and peritonitis, and from secondary complications. Death sometimes occurs with all the symptoms of sudden intestinal obstruction, attended by large accumulations of fæces above the site of the cancer. Pyæmia, thrombosis, and embolism are sometimes the immediate causes of death.

Treatment.—The treatment of intestinal cancer is only palliative. The diet should be restricted to such articles as will produce the least fæcal matter, such as milk, nutritive broths, and eggs; saline waters should always be taken freely to keep the fæces semi-fluid, without at any time causing diarrhœa. Pain must be relieved by the hypodermatic use of morphine; hemorrhages may be checked by balsams and astringents. If at any time hardened fæces collect at the sigmoid flexure, warm water injections are to be given through a long tube. The formation of an artificial anus, the operations of colotomy, typhlotomy, etc., are surgical means for prolonging life. Extirpation of the rectum for cancer has been performed with success.

RECTITIS: PROCTITIS.

Rectitis, or proctitis, is a localized catarrh of the rectum; it rarely occurs except as the result of traumatism, or from the pressure of foreign bodies. It may be acute or chronic.

Morbid Anatomy.—The morbid changes in rectal catarrh are similar to those which occur in catarrh of other portions of the intestinal canal. The colon is distended, and there may be fæcal impaction above the sigmoid flexure. The results of chronic rectal catarrh are periproctitis, peritonitis, abscess, fistulæ into the adjacent tissues and organs, cicatricial contractions, and thrombosis of the hemorrhoidal veins, with subsequent *embolic hepatic* abscess.

Etiology.—Acute catarrh of the rectum may result from blows, the presence of foreign bodies, irritation produced by hardened fæces, and the long continued use of purgatives which act on the lower bowel. It may arise from an extension of inflammation, as in enteritis or dysentery; in the latter case it will be accompanied by ulceration; some regard rectitis as a mild form of dysentery, but it has none of the constitutional symptoms of dysentery, although it is attended by tenesmus, and blood may appear in the stools. Hemorrhoids may excite it, and then it is often called "*the hemorrhoidal catarrhal flux.*"

Syphilitic disease of the anus or rectum, fistula in ano, mucous patches, ulcerations of tertiary syphilis, and exposure to cold, as sitting on the damp earth or on wet sand-bags, may cause it.

Symptoms.—The first symptom of proctitis is tenesmus,—a feeling of fulness and heat in the rectum with straining at stool, which gives rise to burning, scalding pains that shoot from the anal region into the loins and back. The stools contain gelatinous mucus, and frequently there are quite profuse hemorrhages. There is spasm and excessive tenderness of the sphincter ani, and, after violent efforts to expel *supposed* contents, rectal prolapse occurs, and causes the most intense suffering. At no time does the patient feel that the rectum has been completely emptied. There is frequent urination without relief. After these symptoms have existed for some time, scybalous masses are mingled with the muco-purulent discharges, and strangury, hemorrhoids, headache, nausea and restlessness may be present. Hard fæcal masses can often be felt along the line of the colon. It may terminate in recovery in from four to eight days, or it may become chronic.

Chronic proctitis is attended by purulent or sero-purulent discharges, in which are scybalous masses and shreds of sloughing mucous tissue. The discharges are foul smelling. It is usually accompanied by constipation. A digital examination of the rectum gives the sensation of a rigid cicatrical tube.

Differential Diagnosis.—Proctitis may be mistaken for *dysentery, hemorrhoids,* or *cancer* of the *rectum,* and, in women, for *displacement* of the *uterus. Dysentery* is an acute febrile disease, attended by severe pain in the abdomen and great exhaustion, and the discharges have a characteristic dysenteric color and odor. On the other hand, the symptoms of proctitis are local, and a digital examination of the rectum readily establishes the diagnosis. The presence or absence of *hemorrhoids* is also determined by a digital examination; the two conditions, however, may frequently be associated.

Cancer is accompanied by the characteristic cachexia; it develops slowly, and the form of the stools is for a long time modified by the constriction. A small portion of the cancerous mass may sometimes be removed, and when examined microscopically will exhibit the characteristics of the cancer-tissue. In mal-positions of the *uterus,* symptoms analogous to those of rectitis are often present, but the introduction of a uterine sound at once determines the condition.

Prognosis.—The prognosis in acute proctitis is good; its average duration is about a week, and its only dangers are chronic proctitis, peritonitis, fistulæ, and abscess. When proctitis is the result of cancer, or of tumors pressing on the rectum, the prognosis is unfavorable. Chronic rectal catarrh is difficult to cure, and cicatrices following attendant ulceration may lead to intestinal obstruction. When any disease of the liver, lungs, or heart is present which interferes with the venous return, recovery is rarely reached.

Treatment.—A patient with acute rectitis should be kept in bed; a mild laxative, castor-oil, should be given, and the intestine thoroughly evacuated; a milk and farinaceous diet only should be allowed. Sedative enemata, opium and belladonna, cocaine, or morphine, alternating with copious warm water enemata, are the most successful methods of treatment. If there is intense pain, with tenesmus and local engorgement, a hypodermatic of morphine may be given, and leeches applied about the anus. Hot hip baths often give marked relief. Chronic rectal catarrh, if mild, is to be treated by the local application of any of the vegetable astringents; and when severe, the tough and ulcerating membrane should be brushed over every few days with a forty-grain solution of nitrate of silver. Constipation should be avoided, and aperient enemata should be employed rather than cathartics. Local treatment is always more successful than internal medication.

PERIPROCTITIS.

Periproctitis is an inflammation of the connective-tissue surrounding the rectum, and is usually suppurative in character; the resulting abscess may open either into the lower bowel or internally.

Morbid Anatomy.—The manner of the extension of the inflammation through the coats of the intestine to the adjacent connective-tissue, and the course of the morbid processes excited, are identical with those of perityphlitis following typhlitis. The infiltrated tissue forms a tumor which can readily be detected through the rectum. After fluctuation occurs in the tumor, its subsequent course varies. A spontaneous cure by absorption and induration may take place, or the abscess may open and a complete fistula be established, having an internal opening communicating with the rectum. These fistulous tracts are very tortuous, and are always accompanied by a suppurative cellulitis in the adjacent cellular tissue. These fistulous openings in the rectum are high up, and the tracts are separated from the rectum by indurated connective-tissue. Fistulæ may also be established with the bladder or vagina. Suppurating granulations surround the irregular sinuses, and in cases of long standing they may have an epithelial lining similar to that of the anal mucous membrane. Chronic periproctitis may lead to stricture and intestinal obstruction.

Etiology.—Periproctitis is very often a result of proctitis. It also occurs with cancer, intestinal ulcers, and other structural diseases which may involve the rectal mucous membrane. It may be traumatic in origin. It is especially liable to develop in phthisical subjects at a point remote from the rectum, and it may be one of the changes in metastatic pyæmic inflammation.

Symptoms.—The existence of periproctitis is determined by a physical examination. There is local pain, heat, and tenderness; a tumor develops, which soon fluctuates and either gradually disappears or opens externally. If extensive, the formation of the pus-cavity will be attended by hectic, rigors and irregular sweats. There may be a well-marked febrile movement, with nausea and vomiting. If a recto-vesical fistula form, then the urine will infiltrate the adjacent tissues, and septic symptoms will be developed. In all cases, defecation causes intense suffering. The tumor produced by the abscess has been, in some cases, so prominent and resistant that symptoms of intestinal obstruction have resulted. These patients cannot sit erect; and all pressure about the pelvic region is attended by pain. When the abscess opens internally, foul-smelling, purulent masses will be mixed with the fæcal discharges, and the tumor gradually diminishes.

Differential Diagnosis.—Periproctitis may be mistaken for any of those affections of the mucous membrane of the rectum which cause constipation, local pain, and tenesmus; but a careful examination of the parts will show disease of the mucous membrane, and palpation with one finger over the rectal region will discover a fluctuating *tumor* that is found in no other disease.

Prognosis.—When occurring with structural and malignant disease of the rectum, the prognosis is determined by the primary disease. Its prognosis is often unfavorable, on account of its painful and annoying sequelæ, such as *fistulæ* and *stricture*. Fistulous openings communicating with any other parts than the skin or intestine, are very grave results; when they occur in phthisical or enfeebled anæmic subjects the prognosis is always unfavorable. In idiopathic and traumatic periproctitis, the prognosis is good. The lower down the suppurative process, the better is the prognosis.

Treatment.—Before fluctuation occurs, the rules of treatment are the same as in perityphlitis—rest and opium. When fluctuation occurs, the abscess must be opened at its most prominent point; subsequent fistulæ should be freely opened. The abscess should be opened early, to prevent its being discharged into the bladder, rectum, or vagina.

HEMORRHOIDS.

Hemorrhoids, or *piles*, are tumors formed at or near the anus by distended hemorrhoidal veins, or by connective-tissue and skin, which have been distended by blood and indurated by local inflammation. The anastomoses of the superior, middle, and inferior hemorrhoidal veins about the anus form a channel through which venous blood flows, either to the liver or to the cava ascendens. Hence any obstruction in the liver, or cava, may cause distention of these veins.

Morbid Anatomy.—Hemorrhoids are internal or external; the former are within the rectum, the latter are at its anal margin. Piles are "dry" or "bleeding;" internal piles are usually bleeding. External piles are usually dry; they may be large, smooth, tense, dark blue tumors, congested and painful, or smaller, shriveled tabs of skin, quiescent and usually painless. The latter represent a later form of the former after partial spontaneous cure.

On section these tumors exhibit a congeries of dilated veins, sometimes a central cyst containing a blood clot, and sometimes free extravasated blood. Their size varies from that of a pea to that of a walnut. Internal hemorrhoids are sometimes merely flat patches of the mucous membrane with dilated capillaries, and bleed at the slightest touch. Any internal pile may be extruded during defecation, and, if not directly replaced, become congested and inflamed through partial strangulation by constriction of the sphincter. Thrombi forming in the large varicose tumors may lead to ulceration and obliteration. A strangulated hemorrhoidal tumor may slough, and pyæmic symptoms may follow, or a hemorrhoidal ulcer may form. Again, the process may cause periproctitis, abscess, or a rectal fistula.[1]

Etiology.—Hemorrhoids are oftenest met with after the fiftieth year. There is often an hereditary tendency to their development. A sedentary mode of life, luxurious living, and a tropical climate are predisposing causes. Anything retarding blood-return from the rectum, such as impacted fæces, habitual constipation, a gravid uterus, or pelvic tumor, leads to their development. Cirrhosis, atrophy, and passive hyperæmia of the liver, or obstructive hepatic disease will cause hemorrhoids. In diseases of the heart or lungs, causing obstruction in the venæ cavæ, hemorrhoids will develop. Proctitis, cancer, ulceration about the rectum, and the excessive use of drastic purges are causes of hemorrhoids. They may be produced by prolonged attacks of diarrhœa or dysentery. Excess in venery is a frequent cause, and they often develop just *after* the menopause.

Symptoms.—The symptoms of hemorrhoids vary with the size, number, stage, and seat of the tumors. At first there is a feeling of weight and fulness in the rectum, or a sensation as if a foreign body were present. During and after a hard stool, there is a throbbing, aching or burning pain, radiating to the loins or down the limbs. There is heat, soreness and tingling about the anus, and as the tumor increases in size, sitting becomes uncomfortable, and the individual grows restless, depressed, and anxious. The pain soon becomes constant, and is always more severe after a passage or after a moderate walk.

Internal hemorrhoids have, as their chief symptom, *bleeding*, when the bowels are evacuated. From this symptom the name is derived. Slight internal piles may exist for years and only produce local itching and heat. Large internal piles are almost always extruded during a passage, but at first are easily replaced. Later on, standing or walking may cause them to

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[1] Among pathological sequences of hemorrhoids, are anal fissures, prolapsus ani, and changes in the *mucous membrane* described by Virchow as relaxation, with the formation of puffs or folds, slightly thickened and grayish white. The submucous tissue is increased and relaxed and very vascular. The membrane is usually covered with a tough, whitish mucus.

protrude. When they are congested and protrude, they appear as dark purple, soft, vascular tumors. The amount of blood lost in internal hemorrhoids varies from a couple of drachms to a quart; in the latter case there is marked exhaustion and anæmia. Bleeding may be venous or arterial, regular, irregular, or periodical. The latter relieves renal and hepatic congestion, and may ward off gouty and apoplectic seizures. Many "reflex symptoms" accompany hemorrhoids; such as irritable bladder, urethra, and vagina. This class of patients are usually low-spirited, irritable, sallow and anæmic. They may become subjects of melancholia.

Differential Diagnosis.—Hemorrhoids may be mistaken for *proctitis, cancer* of the *rectum, stricture* of the rectum, *prolapsus ani, venereal excrescences*, or *intestinal hemorrhages.* The points in the differential diagnosis of the first three have been given. A careful examination of the everted but normal mucous membrane in prolapse readily distinguishes it from hemorrhoids. *Venereal* growths are hard, have well-defined borders, a cauliflower-like surface, are exceedingly slow in their development, and are accompanied by other evidences of syphilis. *Intestinal* hemorrhage is distinguished from bleeding hemorrhoids by an ocular examination of the rectum. Internal hemorrhoids may be distinguished from *rectal polypus* by the fact that rectal polypus occurs chiefly in the young, as a large solitary and *pale* colored tumor, having a well-marked pedicle.

Prognosis.—The prognosis in uncomplicated hemorrhoids is good; long-standing piles in the aged are rarely permanently cured. Copious bleedings from internal hemorrhoids often hasten a fatal termination in chronic diseases of the liver and lungs.

Treatment.—Those who have hemorrhoids should never allow themselves to become constipated. The diet should be nutritious, and so regulated as to induce free daily evacuations from the bowels. Violent exercise, especially lifting heavy weights, long walks, sitting on damp, warm seats, alcoholic stimulants, and highly seasoned food should be avoided. The best cathartics for this class are rhubarb, senna, sulphur, glycerine, and aloes. The careful use of mineral water is of service in those who are the subjects of hepatic disease. In external piles, a cold sitz-bath should precede the use of astringents, and these, combined with opium and chloroform, will often give marked relief. Inflamed external piles call for the application of leeches and poultices about the anus. Recently, the topical application of iodoform, and the injection *into* the hemorrhoidal tumors of carbolic acid has been recommended. A surgical procedure is the only sure and permanent relief.

INTESTINAL PARASITES.

(*Worms.*)

The history of intestinal worms dates from the earliest medical writings. In the middle ages the history of the tape-worm was closely associated with the doctrine of spontaneous generation. It is within the last fifty years that the doctrines of metamorphosis and migration have been established. There was a time when nearly every malady was attributed to worms,—while a reaction of sentiment ascribed the utmost benefit to their presence. Every year discovers some new parasite; of the fifty or sixty now known, only about ten per cent. are common in, or peculiar to man. The worms which have their habitat within the intestinal canal of the human subject are comparatively few. I shall only give a brief history of those which are of frequent occurrence.

Tænia Solium, or tape-worm, is the final development of an embryo, usually lodged in the flesh of some animal. It is from seven to thirty feet long, has a globular head, connected by a slender neck to its numerous flat segments or joints. The neck is an inch in length, and gradually widens into a joint. The head measures about 1-40th of an inch; around its convexity is arranged a double coronet of hooklets,—the "armed tape-worm,"—and it is provided with some two or four suckers. The flat, thin joints vary from one-half to one-eighth inch in length, being smallest near the neck. The lower border of each segment is larger than the upper. Each mature joint contains both male and female sexual organs (hermaphrodite). The uterus is a long tube, with seven or ten branches on either side, in which the ova develop. An ordinary-sized tape-worm contains five millions of ripe ova. These ova, 1-1700th inch in diameter, become in the pig *cysticercus cellulosæ* (measly pork). From one to forty T. solium may be present in the same intestinal tract; their hooklets and suckers are firmly embedded in the mucous membrane of the *small intestine*—they are almost exclusively confined to its upper third. The proglottides sometimes hang far down into the large intestine; the terminal ripe ones are constantly falling off, and are discharged with the fæces. T. solium is the development of the C. cellulosus from measly pork, the embryo being swallowed, and its vesicular annex removed by the stomach juices, the hooklets become fixed just below the pylorus, and in a few months the tape-worm reaches considerable size.

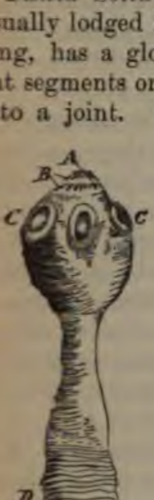

FIG. 67.

Head of Tænia Solium.

A. The Rostellum.
B. Double row of hooks.
C. Sucking discs.
D. Commencing segments below the neck.

Enlarged about 12 times.

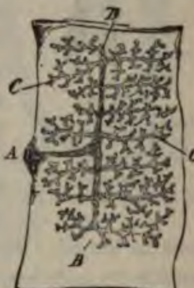

FIG 68.

Mature Segment of Tænia Solium.

A. Genital pore.
B, B. Uterus.
C, C. Lateral branches of the uterus. × 30.

Tænia saginata, or *T. medio-cannellata*, also called "unarmed tape-worm," is larger, stronger, and thicker than T. solium. The segments are broader, far more opaque, and harder than those of T. solium; the head has no hooklets, measures about 1-10th inch, and has four strong and prominent suckers. The uterus is more finely divided, and there are from fifteen to twenty dichotomous branches. The head of the medio-cannellata is more club-shaped than that of the T. solium. The neck is very short. The larval form of this worm is the cysticercus T. saginata, or cysticercus bovis, the embryo being found in *beef*. The ova of T. saginata are oval, and larger than those of T. solium. In their larval state they occur not only in beef, but in the sheep, goat and giraffe. Its mode of entrance, locality, and development are precisely the same as the T. solium. It occurs far more frequently, and more extensively than T. solium, which formerly was the variety always meant when tape-worm was mentioned.

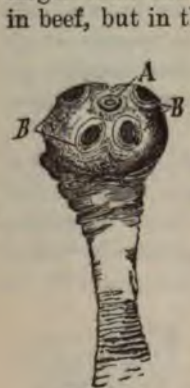

Fig. 69.
Head of Tænia Saginata, or Unarmed Tape-worm.
A. Rostellum.
B, B. Four discs. × 8.

The *Bothriocephalus latus* is the largest worm infesting man; the head of this tape-worm is club-shaped, deeply grooved on either side, and is "unarmed." The head is shaped like an egg, 1-10th of an inch long and 1-26th of an inch wide. The neck is very short and thread-like; the joints are about three times as broad as they are long, but toward the end of the worm they are square. The genital pores look like a small rosette, and are situated about the centre of the segment, and, being all placed on the same side of the worm, this cestoid can be said to have a belly and a back. The eggs are oval, from 1-350th to 1-550th inch, brown in color and at first ciliated. They possess six hooklets. This worm sometimes reaches sixty feet in length; its color, unlike the others, is a dull bluish-gray. The segments do not drop off when ripe, and have not an independent life. It is estimated that ten feet of this worm can produce twelve million ova. The development of this worm is unknown; some suppose its embryo to be found in a fish or mollusk. It is found in the small intestine singly, or with the other two varieties; several may inhabit the same individual.

Round worms, or the hematoids, are more highly organized than the cestoids; the common round worm, or *Ascaris lumbricoides,* is of a brown color, with a cylindrical body, 10 × 1–8 inch in length and breadth in the male, and 15 × 1–4 in the female. The head terminates in three thick semilunar lips, each lip having about 200 teeth. The mouth opens into the alimentary canal, which can be seen through the transparent body. The tail is curved strongly toward the abdomen in the male, this, with its small size, distinguishing it from the female. The female contains two long coils of ovary and oviduct, the length of the generative tubes being eleven times the length of the animal. At the end of the tail, in the male, two projecting spiculæ can be seen connected with the generative organs, which are coils of tubes eight times as long as the worm. The ova are oval in shape (1–340 to 1–440), are produced in immense numbers (sixty millions in a mature female), and are discharged with the fæces. The vitality of these ova is wonderful. How they obtain entrance into man is not known, but it is probable that they previously pass through an intermediate state, and that they attain their full development after entrance. The worms inhabit the small intestine, and vary in numbers from one to thousands. They wander, however, through the tract, may pass through the nose or mouth, or may enter the hepatic, gall or pancreatic duct, into the gall-bladder, or into any fistulous channel, and reach the kidneys, spleen, lung, larynx, etc. The round worm occurs in the ox and pig as well as in man.

The "thread worm," "maw-worm," *Oxyuris vermicularis,* looks like an ordinary piece of thread; the male (like the round worm) is smaller than the female, and is about one-sixth of an inch long. The female is from one-third to one-half an inch long. The body is cylindrical, the tail is much sharper than the head. The head terminates in a mouth surrounded by three lips, from which extends the alimentary tract. The end of the tail, in the male, is curved up toward the abdomen. The eggs are oval, 1-100 to 1-500 inch, each female containing about ten thousand. They are very hardy, having a stout envelope. All their stages of development take place within the intestinal canal.

The *"seat-worm,"* as its name indicates, has its habitat in the large intestine, especially about the rectum, whence it may pass into the vagina or insinuate itself into narrow folds of skin in the anal region. Mature females especially inhabit the cæcum. They vary in number: sometimes the mucous membrane is completely covered with them. The ova enter by means of the food or directly through personal contamination; they die in a few hours after they are placed in water.

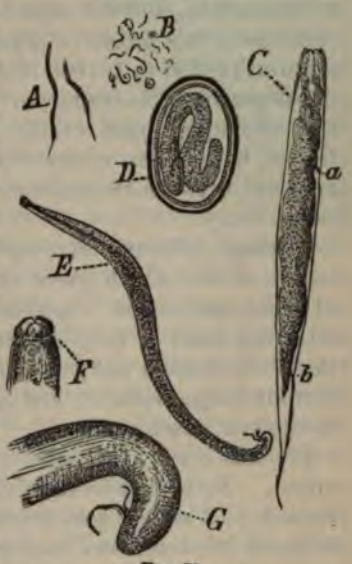

FIG. 70.

A. *Oxyuris vermicularis, or thread worm, female.* B, *Same, male—both natural size.*
C. *Female of same, magnified about ten times, containing ova.* a, *anus;* b, *vulva.*
D. *Mature egg of the thread worm.* × 250.
E. *Ascaris lumbricoides, male, natural size.* F, *head, and* G, *tail of same, magnified about four diameters.*

The *Trichocephalus dispar*, or *"whip-worm,"* is a small worm about one and one-half to two inches in length, the female being the larger. It has been called the *"hair-headed"* worm because its head, which constitutes about two-thirds of its length, is thread-like. The thick body contains the genitals and the intestinal canal; the body of the male is curved into a spiral. The male organ presents a spicula projecting from the cloaca; it is set with numerous sharp points, and is surrounded by a sheath. The uterus, when distended with eggs (60 ova), fills nearly the whole posterior part of the body. The eggs are brown and oval (1-5,000—1-12,000 inch) with a nipple-like appearance at either pole. Its mode of entrance into the body is unknown. There is probably no intermediate state of the ova of T. dispar. The embryos are probably liberated in the stomach, and, developing as they travel onwards, reach the large intestine. Their numbers vary from fifty to one thousand. The T. dispar is found in some varieties of apes; and the T. crenatus of the pig is probably the same as this worm.

Trichina spiralis belongs to general diseases, and will not be considered here.

The *Anchylostomum duodonale,* or *dochmius duodenalis,* is a small cylindrical worm, the females being seven-tenths and the males four-tenths inch in length. It is thicker than the seat-worm, almost as thick as the body of the whip-worm. The mouth is furnished with eight teeth. The armature of the mouth, and the capsule about the head are very complex. The male terminates in a lobate enlargement, but the female is pointed. The eggs are oval (1–350 to 1–1000 inch), have a very thin shell, but regarding their development little is known. It inhabits the lower part of the duodenum and the upper part of the jejunum. It probably has no intermediate bearer, but as soon as developed attaches itself to the villi and may become encysted between the mucous and muscular coats. It may cause intestinal hemorrhage.

Etiology.—Worms develop in the intestines of man, either by the entrance of ova which grow into the mother parasite; or by the entrance of what are called "intermediate parasites." Their entrance into the intestinal tract is only effected through food and drink. Butchers, and those who handle raw meat, are more subject to them than others. Filthy surroundings, squalor, and personal uncleanliness are conditions which favor their development.

Cestoids occur at all ages; tape-worm has been found in a five-day-old infant. *Bothriocephalus latus* is found chiefly in Scandinavia, Russia, and Poland. *T. solium* occurs wherever the pig is domesticated. *T. saginata* is found wherever raw beef is used for food. *The Tænia* are not found among Jews and those who eat no pork. The monks of the Carthusian order, who eat only fish, are free from worms. Iceland is the only country in which the hæmatoids are not found. The round worm occurs in warm climates oftener than in cold. Their number and extent are in direct proportion to the filth of the surroundings. They are more common in women and *children* than in men; in those who live in the country than in those who live in cities. The oxyuris vermicularis is everywhere prevalent. It occurs especially in young children, but is not uncommon in adults. The itching which these worms cause, especially at night in a warm bed, induces so much scratching, that when two or more children sleep together the worms may be carried from one to another by the hands. Those who have charge of children, nurses, etc., often become infected in this way.

T. dispar abounds in this country, Europe, Syria, and Egypt; it is present at all ages, but, strangely enough, in the first and second years of life it is *rare*. The whip-worm produces so few symptoms, and can be so readily overlooked at a *post-mortem*, that its etiology is obscure. The Anchylostomum duodenale (or the dochmius or strongylus duodenalis) was discovered by Dubini in 1838, in Northern Italy. It prevails in Brazil and Egypt. The negro is oftener subject to it than the white; but it can be shown that bad food and drink are of more importance in causing it than race. Women are affected oftener than men. The conditions known as cachexia Africana, mal d'estomac, etc., are due to the presence of this parasite.

Symptoms.—The only symptom which gives positive evidence of the existence of intestinal worms or their ova is their discovery in the stools or about the anus. Tænia produces no constant symptoms. The bowels are usually irregular. There may be colicky pains in the abdomen; the appetite is capricious, the face may be pale and the mouth drawn. Often the stomach feels weak, and there is nausea, perhaps vomiting. In some instances these stomach symptoms, with colic, occur after certain articles of food ; in others, certain kinds of food relieve them. Among the reflex symptoms are headache, dizziness, ringing in the ears, sudden sweatings, irregular attacks of palpitation, depression of spirits, lassitude, ocular spectra, sudden salivation, and itching about the nose and anus. Chorea, grinding the teeth, hysteria, anomalies in menstruation, epileptiform and maniacal actions have occurred in those in whom tape-worms were found to be present. The special senses may be temporarily involved :—deafness, blindness and loss of speech have occasionally occurred. All the senses are enfeebled to a marked degree. These symptoms are more those of hypochondria and hysteria than of tænia. The subjective sensations which one who believes he has tænia may describe are innumerable, and each patient will have his own peculiar group of special notions. Often patients with one large or several small tæniæ enjoy perfect health so long as their existence is not suspected. The diagnosis can only be made by the discovery of detached joints or segments of the worms in the fæces.

The *ascaris lumbricoides* or *round worm* may be present in large numbers, and yet give no symptoms of its presence. Usually, however, there are certain symptoms which are regarded as "signs of worms," such as itching and picking at the nose, foul breath, colicky pains, especially about the umbilicus, bloody mucous diarrhœa, perverted appetite, restlessness, disturbed sleep in which the child grinds its teeth, with nausea and vomiting which is regarded as evidence that the "worms have passed into the stomach." The vomiting, however, is reflex. The abdomen is usually tumid, distended, and doughy to the feel. The urine looks somewhat like rice-water, the lower eyelid is of a dark purple color, or there may be rings about the eyes ; the pupils are often unequal. Later there are hysterical convulsions, with choreal movements, and the child becomes emaciated. These worms, by coiling themselves into a bundle, have caused intestinal obstruction. They have entered the larynx and induced death by suffocation, have reached the ductus communis and caused jaundice and hepatic abscess, and they may take their way through any artificial opening into the peritoneal cavity or bladder, but they cannot cause intestinal perforation. It is a question if lumbrici can induce catarrh and ulceration of the intestine. At an autopsy, where large colonies of lumbrici have been found, the intestine has been intensely congested in the neighborhood, leaving no doubt as to the cause. Attacks of laryngismus stridulus are sometimes induced by lumbrici in highly nervous children. While the existence of these worms may be suspected, their diagnosis can only be made by their discharge from the stomach or intestine.

The *seat-worm*, or oxyuris vermicularis, when present in small numbers, produces few symptoms. When they are numerous in nervous and susceptible patients, there is intense itching about the anus, especially on retiring, when the increased warmth causes them to be very active, and by this sleep is more or less disturbed. They often cause a frequent desire to go to stool, and sometimes there is an abnormal amount of mucus mixed with the fæces showing that they have produced extensive local irritation. In such cases, there will be punctate redness about the anus, and in female children, where the worm wanders into the vagina, there will be irritation of the vulva, which leads to vulvitis. Sometimes with the itching there will be pain and tenesmus, and the fetid stools will be bloody and streaked. The genito-urinary disturbance may cause such abnormal excitement of the sexual organs, that it may lead to onanism, seminal loss, and nymphomania. Hysterical, epileptiform, choreal, and cataleptic symptoms have been induced by the irritation produced by these worms. On account of the local irritation which they produce and their easy detection on careful examination, their diagnosis is not difficult, for their ova or the parasites themselves will be found in the fæces, or in the folds at the margin of the anus. A careful inspection of the rectum in those who suppose they are suffering from hemorrhoids will often disclose the presence of the seat-worm as the cause of the anal irritation.

Trichocephalus dispar produces no symptoms. Either the worms or their eggs must be found in the fæces to establish a diagnosis.

Anchylostomum duodenale induces a chlorosis-like anæmia; the skin and mucous membranes are pale, and the cardiac and venous murmurs of anæmia are well marked. The loss of flesh and strength is constant and progressive. Dyspepsia and anorexia, alternating with bulimia, and an appetite for certain and peculiar articles of food, are early symptoms. In most cases there is a sense of weight or pain in the abdomen; the stools are frequently colorless, and the urine is pale and abundant. Sometimes slight intestinal hemorrhage will occur.

Differential Diagnosis.—The diagnosis of the presence of intestinal parasites is made by the discovery of the parasites or their ova in the stools or the matters vomited. *Acute hydrocephalus* may be mistaken for worms, but in hydrocephalus the projectile vomiting, the slowed pulse, the fever with irregular exacerbations and remissions, the constipation, the hydrocephalic cry, and the retracted abdomen, all stand in marked contrast to the symptoms of intestinal parasites.

Prognosis.—Intestinal parasites may cause death, first by their entrance into the larynx, the ductus communis (causing abscess of the liver), or when they collect in masses and cause fatal intestinal obstruction; secondly, when extreme anæmia and exhaustion are produced by anchylostomum duodenale. The prognosis in tænia is good, except in very young children, and in the enfeebled. When parasites have resided a long time in the intestines, some of the reflex symptoms may remain after their removal. It is thought that the ascaris lumbricoides does not remain longer than a year in the human body, unless there is an exposure to new sources of infection. Death occurs with symptoms of exhaustion, greatly increased by the intestinal hemorrhage, profuse diarrhœa, and persistent vomiting.

Treatment.—Prophylaxis demands that all raw or "underdone" meat shall be avoided. Measly pork should not be allowed to be sold in the markets, and wells and springs from which drinking water is obtained should be removed from the neighborhood of stockyards. A point in prophylaxis that has never been mentioned is the *washing* of green vegetables, such as lettuce, as the Swiss and Germans sprinkle their growing vegetables with the water drained from human excrements.

The means to be employed for the expulsion of the tape-worm have for their object the dislodgment of its head ; so long as this remains, it is useless to hope for cessation of the symptoms. Whatever anthelmintic is administered for this purpose, the bowels must be thoroughly evacuated. This can be effected by some of the saline purges, or by a copious draught of some purgative mineral water. The diet should be restricted for two or three days before its administration, and then either pomegranate, Kousso, male fern, or pumpkin-seeds may be given in full doses. As the administration of anthelmintics may induce vomiting, a cup of black coffee may be given a few minutes before they are taken. The *cortex radicis punicæ granati* is used in the form of a decoction (three ounces to a pint and a half of water). When boiled down one-half, it should be given in three divided doses. To this may be added filix mas, gamboge, or tansy ; and in case the worm is not dislodged, a dose of castor-oil should follow. Kousso, the flower of the *Brayera anthelmintica,* is given in one-half oz. doses mixed with water ; or in an infusion (one-quarter oz. to four oz. of water). The odor of the Kousso is very offensive.

The male fern (*aspidium filix mas*) is one of the oldest and best known vermifuges. The ethereal extract—oleoresin—is given in capsules ; dose, one-half drachm. It may be given as the powder of the rhizome, 60 to 100 grains. It should also be followed by a dose of castor-oil, gamboge, or calomel. Oil of turpentine, in one-half or one-ounce doses, is very effective ; it may produce headache, giddiness, or a kind of intoxication. Petroleum, in 20 to 30-drop doses, has been used in Egypt. Kameela (*Rottlera tinctoria*) is to be given in one-third drachm doses every three hours. Carbolic acid (5 grains) and salicylic acid (12 grains) have also been found efficacious. An emulsion of pumpkin-seeds frequently acts efficiently. The active principle of pomegranate-root bark, *pelletierine,* is thought by some to possess all the powers of the root. All the above-named drugs are efficacious, and when they fail it is usually because they are not properly administered.

For round worms, besides the vermicides mentioned, santonin, spigelia, calomel, and chenopodium may be used. Santonin is by far the most reliable, but it requires care in its use, on account of the severe gastric and nervous symptoms which it causes ; one-half grain for a child and three to six grains for an adult is a maximum dose. The oil of chonopodium is recommended, —dose, five to ten drops ; and the fluid extract of senna and spigelia is often effective.

The thread or seat-worm may be destroyed and washed away by enemata of quassia, oak-bark, alum, salt and water, or carbolic acid. At the same time the vermifuges should be given, and the bowels gently moved by castor-oil. Thoroughly washing the anus and the parts around it with a one per cent. solution of carbolic acid, and subsequent attention to cleanliness, suffice in the majority of instances. It is said that turpentine and calomel are the best means of getting rid of the A. duodenale. Following the expulsion, tonics should be used. For either the round, seat, or whip-worm, santonin is the best remedy; and for the last two varieties, thorough local treatment and absolute cleanliness will generally suffice.

FUNCTIONAL DISEASES OF THE INTESTINES.

The principal functional diseases of the intestines are *constipation* and *colic* or *enteralgia*.

CONSTIPATION.

Constipation is a relative term, for some perfectly healthy persons have only one movement from the bowels every second or third day, while others have two stools daily.[1] It is difficult to explain these differences, and to say what constitutes constipation in an individual unless his habit is known. No standard can be applied indiscriminately to all persons. Those who suffer from constipation are always able to make their own diagnosis. In the majority of instances constipation is due to a deficiency in the peristalsis of the large intestine.

Morbid Anatomy.—There are no lesions which are constant in functional constipation, but if it has been of long standing it may cause changes which after a time become an additional cause, such as dilatation of the intestine and hypertrophy of its walls. The colon may become so dilated as to measure from twelve to fifteen inches in circumference. If hypertrophy occurs it is usually most marked at the upper part of the rectum and at the sigmoid flexure of the colon. More or less paralysis of the muscular coat precedes and accompanies dilatation, and pouches may form along the colon containing masses of mucus and fæcal matter. These pouches occur most frequently at the sigmoid flexure, and may be arranged in rows. Ulceration and perforation of the dilated and weakened intestinal wall may cause fatal peritonitis. Sometimes the intestines rupture *without* ulceration from prolonged and severe peristalsis at the seat of the fæcal obstruction. Typhlitis and perityphlitis may be a result of fæcal impaction due to habitual constipation. Many diseases of the rectum and adjacent viscera are also among its results, such as hæmaturia, rectal abscess, fistulæ, anal fissures, prolapsus ani, and passive hyperæmia of the pelvic viscera. Hemorrhoids usually complicate long-standing constipation.

Etiology.—Constipation often results from the same habits and mode of life which cause dyspepsia, and it is a very frequent accompaniment of it. It may arise from the prolonged use of opium and the abuse of laxatives. It occurs with certain diseases of the brain and spinal cord. In those who have what is called a "costive habit" collections of pills which have been taken for its relief sometimes form a nucleus about which masses of impacted fæces collect. A change in habits of life or diet is frequently followed by temporary constipation. Those who lead a sedentary life, the feeble, infirm, the bed-ridden, and child-bearing women are predisposed to constipation. Loss in the contractile power of the abdominal muscles from any cause may induce constipation. Abnormalities in the intestinal secretion, as in chronic alcoholismus, and organic or functional disturbances of the liver lead to constipation; heart disease, bronchitis, emphysema, and asthma are included in this list. It may also result from unnatural dryness of the fæces, such as occurs in diabetes, where large quantities of fluid are carried off by the kidneys. This dryness may also occur in those whose occupations cause profuse perspiration. General anæmia and chlorosis cause it. Many conditions giving rise to reflex irritation, more especially diseases of the genito-urinary organs, induce atony of the intestine and consequent constipation.

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[1] Cases are recorded where periods of three months have elapsed between two successive movements, and yet the individual was apparently in good health.

One of its most frequent causes is anxiety and prolonged mental labor, especially in those leading a sedentary life. It is common in melancholia and insanity, and may occur with hysteria. The long-continued use of cathartics is a frequent cause of obstinate constipation. Hereditary predisposition may be classed as a cause. Old age is always a predisposing cause. Cases are on record where from boyhood until the seventieth year the bowels did not move more than once a week, and yet the individual enjoyed perfect health. In those accustomed to large doses of opium the bowels have been known to move only four times in the year. Departure from the standard natural to each individual will determine the existence or non-existence of constipation.

Symptoms.—Usually when a person whose bowels have been accustomed to move daily habitually passes two or three days without defecation he complains of a sense of fulness in the rectum, with flatulence, headache, vertigo, a foul breath, anorexia, and well-marked dyspeptic symptoms. Nervous subjects become hypochondriacal, and there is mental inactivity with insomnia, or the individual awakes unrefreshed from a broken sleep. The skin becomes parched, shrivelled, sallow and pasty. Eruptions such as psoriasis, eczema, prurigo, erythema and urticaria often appear upon the surface. There are frequent flushings of the face, and the eyes are surrounded by deep purplish rings. The tongue is flaccid, often indented by the teeth. The breath and the perspiration have an offensive odor, and frequent attacks of cardiac palpitation cause the patient to become anxious about himself. Those who are habitually constipated are subject to fits of vertigo and temporary loss of consciousness.

Besides the subjective symptoms of constipation there are those produced by the mechanical interference caused by the hard fæcal masses. If the colon is distended there is more or less pain, which is nearly always located in "the chest." A distended transverse colon may cause such pressure upon the duodenum as to interfere with its function and give rise to dyspeptic symptoms. The pressure of large fæcal collections in the descending colon and cæcum sometimes causes irritation along the genito-urinary tract, irritability of the bladder, and neuralgic pains in the groins, ovaries, testicles, loins, and lower extremities. At any time the symptoms of intestinal obstruction may occur. Diarrhœa may follow prolonged constipation, from the catarrh excited by the irritation of the mucous membrane produced by the fæcal mass, and pressure on the biliary duct may cause an obstructive jaundice.

The impacted fæcal masses may give rise to one large tumor, or to several small yet distinct masses which can easily be detected along the line of the large intestine; they may be felt often in the transverse or ascending colon as movable tumors, but generally the largest accumulations collect in the sigmoid flexure and cæcum. These tumors are often so large that they cause *tenesmus*. In the aged, torpor of the rectum is often marked by spurious diarrhœa, acute pain in the lower part of the abdomen, great tenesmus and bearing down at stool, accompanied by dysuria, and, often, retention of the urine.

Differential Diagnosis.—The method of the diagnosis of impacted fæcal masses has been given under the head of "Intestinal Obstruction." Impacted fæces in the rectum may be mistaken for *cancer;* a digital examination of the rectum will establish the diagnosis.

Prognosis.—When the constipation is functional, and not the result of malignant growths, or intestinal obstruction other than fæcal, the prognosis is good. In very old people it is almost impossible to overcome habitual constipation, on account of their constant indolence and apathy. Inflammatory complications always render the prognosis unfavorable; and after long-continued constipation the symptoms of intestinal obstruction are apt to be followed by peritonitis of a low type, which may not be suspected during life.

Treatment.—The treatment of temporary constipation consists in the administration of a dose of Epsom or Rochelle salts, or a tumbler of any one of the many efficient natural waters; or, if indicated, a mercurial purge followed by a saline. It is not often that a physician is consulted for simple constipation; care, diet, and exercise, with an occasional cold water enema, are usually all that is required to keep the bowels open.

Habitual constipation, however, frequently attains the dignity of a disease, and it requires much care and patience, both on the part of the physician and patient, to overcome it. One who suffers from habitual constipation should endeavor to establish a regular hour for the evacuation of the bowels. Straining at stool should be avoided. Regular habits in this respect are most efficient for overcoming obstinate constipation; the success of any plan of treatment will depend largely upon the perseverance of the individual. The dietetic measures consist in partaking freely of those articles of food which leave a bulky residue, such as the coarser vegetables, cracked wheat, oatmeal, etc. Fruits which have fine seeds (figs, strawberries) that will stimulate the intestinal mucous membrane, are of service if they do not cause indigestion. Prunes sweetened with molasses are sometimes very efficient. Great care should be exercised not to overload the stomach with food difficult of digestion, and each individual is a law unto himself in this matter. A goblet of cold or hot water just before retiring and on rising will often overcome a long-standing constipation, while the daily use of saline waters is to be avoided, for such use often makes the constipated habit more inveterate. Daily exercise in walking or horseback riding, is a most efficient means for overcoming constipation in those who are strong and vigorous. Water should be taken freely before and after the exercise. The tonic effects of a cold sponge or shower-bath on rising are often of great service. The mechanical means consist in friction and kneading of the abdomen. In the old and bed-ridden, bending the body backward and forward will be found to provoke and aid defecation. The galvanic current is especially beneficial in the aged and paralyzed. Included in the list of mechanical means are enemata and suppositories. Cold water, salt water, soap and water, castor-oil, etc., are at first very efficient as enemata, but the rectum very soon becomes accustomed to them and ceases to respond to their stimulus.

If mechanical means, diet and change of habits fail to overcome the constipation, recourse must be had to medicinal agents. These are very numerous ; the rule is to begin with the mildest. Cases are often met with where an individual has taken stronger and stronger cathartics without avail, and until the great object of his life seems to be to get a movement from the bowels. It will generally be found in such cases that reliance has been placed wholly on drugs ; by changing to the milder cathartics, regulating the diet, and insisting upon daily exercise, the constipation is easily overcome. It is always to be borne in mind that drugs are only aids to other measures. Tonics should always be combined with laxatives ; gentian, strychnine and quinine, combined with aloes, will often effect more than the most drastic purgatives. Favorite cathartic combinations are :—(1) aloes, myrrh, colocynth, gentian, and quinine ;—(2) aloes, rhubarb and strychnia ;—(3) strychnia and aloin ;—(4) nux vomica, aloes, belladonna, and podophyllum. In all combinations for constipation in females, belladonna and hyoscyamus are very active agents. Podophyllum produces slow and painless evacuations, and acts efficiently for a long time. In very obstinate cases, colocynth, scammony, or one-sixth of a drop of croton-oil may be required until the habit of daily evacuation is established. Rhubarb and magnesia is a favorite cathartic in children and young girls. In old age and in children, drastic cathartics are always to be avoided. If a large fæcal mass becomes impacted in the lower part of the colon, it will often have to be scooped out with the finger or rectal scoop. If the mass is exceedingly hard, it is best to throw a steady stream of moderately hot water and glycerine against it before attempting to remove it. Enemata are adjuvants to all plans of treating constipation, where there is evidence of a large fæcal accumulation in the lower bowel.

INTESTINAL COLIC.

The term intestinal colic, in its wider sense, includes all painful affections of the intestines which are not caused by structural changes in the intestinal walls. Its varieties are *flatulent, bilious, lead, copper, gouty* and *rheumatic* colic. It belongs to the class of neuroses, and is purely functional in its nature. It is attended by irregular spasmodic contraction of the muscular coat of the intestine.

Etiology.—It occurs most frequently in the young, the liability to it steadily decreasing with advancing years, and in females oftener than in males. Neurotic temperaments and a sedentary mode of life, rheumatism, chronic alcoholismus, and gout predispose to it. Its most frequent direct cause is excessive distention of a portion of the intestinal canal. It is apt to occur in the hysterical and hypochondriacal, and in those who are the subjects of malarial and syphilitic cachexia. Hepatic and biliary derangements induce it. Cold, especially cold to the feet, is often its exciting cause. Direct irritation of the bowels by undigested food, certain articles of food, as cucumbers, shell-fish, strawberries, etc., will cause colic in some persons. Gaseous collections and distention of the intestine by fæces, or by bundles of worms, sometimes excite it. Lead and the copper salts cause *colica pictonum* and *copper colic*. All metallic colics seem to result from hyperæsthesia of the terminal nerves.

Symptoms.—An attack of intestinal colic may be preceded by a sense of distention in the abdomen, slight nausea and belching, languor, numbness, irritability of temper, or apathy. The attack itself comes on suddenly.

In *flatulent* colic, there is a severe twisting, paroxysmal pain around the umbilicus, or in the region of the colon. The abdomen becomes distended with the flatus, the bowels are constipated, eructations and borborygmi are present, and there may be vomiting. The escape of flatus, change of position, and steady pressure over the abdomen relieve the pain ; rarely is the abdomen tender. There is no rise of temperature, the surface, if the pain is severe, is cold and covered with clammy perspiration. The pulse is small and feeble. At the height of the attack the patient groans and rolls about, frequently throwing himself across some hard substance, so as to cause pressure on the abdomen. In children, convulsions, projectile vomiting, syncope, strangury, priapism, and cardiac palpitation are not infrequent. A large quantity of limpid urine is usually secreted, and there is a frequent desire to urinate. After several hours, during which many spasms of the colic have occurred, large discharges of flatus, rumbling of the bowels and milder paroxysms of pain, mark the termination of the attack. In the weak and nervous, the expression of the countenance, the condition of the pulse, and the signs of collapse may cause one to suspect intestinal perforation. On palpation during a spasm, the intestine at points may be felt rigid and hard ; the symptoms disappear as the paroxysm subsides.

Flatulent colic is often called *crapulous*, when it follows a too hearty meal or the ingestion of indigestible articles of food. In crapulous colic, the tongue is either covered with a white fur, is enlarged, showing the red papillæ through it, or it is bright red at the tip and edge. Crapulous colic is accompanied by pains in the head and dimness of sight ; and sometimes urticaria and roseola, strophulus, and other lichenous eruptions appear on the skin. Flatulent colic is most frequently met with in infants, and the picture presented by a child with wind colic is too familiar to need further description. In adults, flatulent colic may be due to malarial influence, and then the attacks will be periodical.

Bilious colic is accompanied by nausea and vomiting, the vomited matters being greenish and yellow. It is preceded by nausea, anorexia and a coated tongue. It sometimes begins with a chill. The bowels are obstinately constipated, there is slight fever, the abdomen is tender and *slightly* distended, or it may be retracted. When prolonged, bilious colic may be accompanied by jaundice. Bilious colic occurs in summer and autumn, chiefly in malarial districts. A form of colic which is often a distinct "cramp," is obviously due to a gouty or rheumatic diathesis; beyond its etiology, it does not differ from flatulent colic. It may be metastatic.

Of the metallic colics, *lead colic*, "colica pictonum," is far the most frequent; it is a true colic, no lesions being found in the intestines of those who have died of it. The metallic colics are produced by the primary action of the metal on the nervous system, and are preceded by the general symptoms of the poisoning. Lead colic comes on with moderately severe paroxysms of pain, which gradually increase in severity until a series of intense paroxysms rapidly follow each other. The pain is located about the umbilicus, and is twisting or grinding in character. With the colic there may be cramps and pains in the extremities. The abdomen is contracted and hard; knots of rigid intestine can sometimes be felt. The abdomen is not tender, and forcible pressure markedly relieves the pain. The bowels are obstinately constipated, but as the attack passes off diarrhœa often occurs. After the subsidence of the pain another attack may be excited by taking food, or one may return without any apparent cause. The pulse is slow during an attack, and there is no rise of temperature. An individual suffering from lead poison is sallow, anæmic, and more or less enfeebled. The extensors of the fore-arm are often paralyzed (drop-wrist), and there may be amaurosis (due to optic neuritis) and epileptiform convulsions. Along the edge of the gums is a deep blue dotted line composed of lead, formed by the sulphuretted hydrogen produced by decomposing food lodged between the teeth reacting on the lead which circulates in the capillaries. This is the distinctive sign of lead poisoning. The pain in lead colic radiates in all directions, and its point of maximum intensity is located at different times in different regions of the abdomen.

Copper colic may be distinguished from lead colic by the fact that the pain is increased by pressure, the abdomen is distended instead of retracted, and in place of obstinate constipation there is diarrhœa with greenish stools; there is a *purplish* line about the gums, and there may be attacks of dyspnœa from laryngeal and bronchial spasm.

Differential Diagnosis.—Intestinal colic may be confounded with *peritonitis*, *intestinal obstruction*, *gall-stone colic*, *intestinal perforation*, *spinal disease*, *aneurism*, *labor-pains*, *hernia* and *muscular rheumatism*.

In *peritonitis* there is usually a distinct febrile movement, the pulse is accelerated and is tense and wiry in character. In colic there is no fever or increase in pulse-rate, the rule being rather a *slowed* pulse. In peritonitis the patient avoids the slightest motion of the body, and firm pressure over the abdomen increases the pain, while in colic the patient tosses from one side to the other, and firm pressure over the abdomen relieves the pain. The pain of peritonitis is constant, that of colic is paroxysmal.

Perforation of the intestine is to be distinguished by the intensity and rapidly increasing severity of the pain, rapidity of the pulse, rapidly developing tympanitis and collapse.

In *spinal disease* the pain is along the course of the nerves and all the intestinal symptoms of colic are absent.

Aneurism of the abdominal aorta is distinguished by the physical signs of aneurismal tumor, by the change in the femoral pulse, and by constant localized pain in the back.

Hernia has an external tumor, there is stercoraceous vomiting, and only great carelessness in the examination will allow of error in the diagnosis.

Labor-pains may simulate colic, and there are cases on record wherein concealed pregnancy—the true state of affairs was not recognized until labor was completed.

Muscular rheumatism is attended by intense and constant pain, aggravated by motion and pressure, having its maximum intensity at the origin and insertion of the muscles. There will be a history of exposure, and also of frequent rheumatic attacks in other parts of the body.

Prognosis.—The prognosis is always favorable. Death has occurred from rupture of the intestine from excessive gaseous distention, and from convulsions in very young children.

Treatment.—The indications for treatment are to be found in the etiology of each attack. In flatulent colic, and in that from impacted fæces and undigested food, evacuants are indicated. The internal administration of castor-oil and an aperient enema generally give relief. If the colic is due to exposure of the feet or abdomen to cold, hot aromatic teas and diaphoretics are indicated. In all forms, opium, chloroform, hydrate of chloral, or ether may be given to relieve the pain and spasm. In hysterical and nervous subjects, at the onset of the attack, Hoffman's anodyne, musk, asafœtida, valerian, and the essential oils often quickly relieve the pain and remove the flatus. In children, bromide of potassium in carminative waters, often affords speedy relief. In some cases it will be necessary to relieve an overloaded stomach by administering an emetic. Malarial colics demand for their relief quinine combined with calomel. Gouty colic is best treated with oil of cajeput, Warner's cordial, and carminatives. The feet are to be placed in a mustard bath, and a mustard plaster is to be placed on the abdomen. Anti-gout remedies are to be given as soon as the severity of the colic is relieved.

In lead colic, opium is the most efficient remedy. It will often relieve the constipation. My rule is to combine it with belladonna and croton oil (1 grain of opium, 1-6th grain of the extract of belladonna, and 1 drop of croton oil), every two hours, until relief is obtained. Sulphate of magnesia is preferred by some to the croton oil; a warm bath will often give immediate relief and hasten the action of the croton oil. As soon as the bowels are acted upon the pain disappears. Faradic electricity and pilocarpin are advocated by some German authorities. A milk diet acts as a prophylactic and curative agent, and workmen in lead factories should drink large quantities of it. It is claimed by some, that sulphuric-acid lemonade is a good preventive, as it forms insoluble lead sulphate. A long time elapses before all the lead is removed from the system.

In *copper colic* sulphur baths, turpentine stupes or sinapisms, ether and opium, and a milk diet, with the casual indications, are all that is necessary for its successful management. In all forms there are two prominent indications for treatment, viz.: to relieve pain with opium, and to evacuate the bowels. Warm fomentations and sinapisms to the abdomen are always of service. Cold applications are contra-indicated. Always seek for, and, if possible, find the cause before cathartics are given.

PERITONITIS.

Peritonitis is an inflammation of the whole or a part of the serous membrane which lines the abdominal wall and covers the viscera contained in the abdominal cavity. It may be *acute* or *chronic; local* (circumscribed) or *general*. The acute form usually begins at one point and rapidly spreads over the entire membrane. The chronic may result from the acute, or it may be interstitial, hemorrhagic, tubercular or cancerous.

Morbid Anatomy.—*Acute, general* (or diffused) peritonitis begins with an intense injection of the capillaries of some portion of the visceral or parietal layer of the peritoneum. Sometimes the injection is so intense that the capillaries rupture at points and cause small blood extravasations. The inflamed portion at first presents a mottled appearance ; the redness is most intense at the starting point of the inflammation. With the capillary hyperæmia there is desquamation of the endothelial cells, and the peritoneum loses its natural glistening appearance, becomes dry and lustreless, and there is swelling and an increase in the number of its fixed connective-tissue cells. Following this, a more or less abundant exudation takes place upon its free surface, into its substance and underneath it. This exudation may be fibrinous, sero-fibrinous, or purulent. The changes in general peritonitis are usually most marked in the parietal portion of the peritoneum, in layers of the omentum, and in the meso-colon.

With the advance of the disease, the fibrinous exudation increases in amount, and assumes a distinctly yellowish tinge ; as it increases in thickness it presents the appearance of a roughened false membrane, which may vary in thickness from a mere film to a quarter of an inch or more ; its consistency varies from a pulpy mass to a coherent, elastic membrane. It may form a continuous layer over the visceral or parietal portion of the peritoneum, and agglutinate its opposing surfaces more or less firmly to each other. If a serous effusion occurs at the same time, it tends to gravitate to the most dependent portion of the peritoneal cavity ; it is usually small in quantity. After a time cells appear in the layer of fibrinous exudation, probably derived from the connective-tissue of the peritoneum, which cause the development of a layer of new connective-tissue, which may give rise to permanent thickenings and adhesions between the surfaces. Papillary connective-tissue growths may also form and cause adhesions ; these adhesions are usually in patches. The new connective-tissue is most extensive and thickest over the solid viscera, as the uterus, liver and spleen.

The bands of adhesion may bind down a portion of the intestine and cause fatal obstruction, or they may form tense cords underneath which a loop of intestine may be suddenly incarcerated. On the surface of the solid viscera the new tissue gradually becomes indurated, resembling cicatricial tissue. Sometimes the new connective-tissue bands are so slight that the peristaltic motion of the intestines causes them to disappear. Firmer adhesions may cause displacement of the viscera or twisting of the intestines, or the whole contents of the abdominal cavity may be matted into one globular mass. Acute peritonitis may cause a general thickening of the peritoneum without adhesions, its tissue becoming dense, white and fibrous. In these cases the fibro-cellular developments are chiefly in the substance of the peritoneum—not on its surface.

The changes in the intestines vary: at first, vascular lines are seen running along their long axis, and in very acute cases their whole surface is reddened. Interstitial cell-growth of the sub-peritoneal coat, accompanied by inflammatory œdema, causes their peritoneal surface to present an opaque appearance. The muscular coat loses its contractile power, and they become distended with gas, so that when the abdomen is opened they protrude through the incision. The abdominal muscles and the surface of the viscera, especially the liver and spleen, are paler than normal. The intestinal mucous membrane is sometimes paler than normal, sometimes intensely hyperæmic.

In non-adhesive or sero-fibrinous peritonitis, with the plastic exudation there is a more abundant effusion of fluid, containing flocculi of lymph and cells which are most abundant in the most dependent portions of the abdominal cavity. Its color varies from a delicate straw color to a grayish red. Underneath the fluid on the surface of the peritoneum there is a layer of exudation which in its anatomical arrangement is the same as that in adhesive peritonitis, and it undergoes similar changes. Displacements of the abdominal and thoracic organs often occur from the pressure of a large fluid effusion. The fluid effusion may undergo absorption, and, the two plastic layers coming in contact, adhesions will form as in the adhesive variety.

Acute suppurative peritonitis may have for its product a fibro- or sero-purulent exudation. In this variety, the parietal and visceral layers of the peritoneum are partially or completely covered and infiltrated with a gray, opaque, soft, fibrous exudation, which is infiltrated with pus cells. The effusion varies in color and consistency; it may be thick, creamy, and viscid, or turbid, thin and watery. It collects in the dependent portions of the pelvic cavity. The purulent exudation may be spread out over the entire surface of the membrane, or it may be associated with adhesions when it occurs in distinct collections; it is bounded by organized septa, and appears as if there were numerous separate abscesses. An ulcerative process may be established, and the purulent accumulation may be discharged through the abdominal walls into the intestinal canal, bladder, vagina, or even into the thoracic cavity. The purulent accumulation may find exit, in rare instances, along the plane of the psoas muscle. In "puerperal peritonitis," the uterus and its appendages are thickly covered and infiltrated with pus. Sometimes the ovaries and the Fallopian tubes contain pus. In nearly every instance of acute suppurative peritonitis the surfaces of the viscera present evidences of lymphangitis, phlebitis, or superficial abscesses. If recovery takes place without a discharge of the purulent accumulation, a part of it is absorbed, and the remainder becomes cheesy and encysted.

If acute local peritonitis is secondary to visceral inflammation, the inflammatory process in the viscus reaches its surface and involves the peritoneum covering it. These inflammations have received various names, as perihepatitis, perisplenitis, perimetritis, etc.; the inflamed peritoneum in this variety is usually sharply defined. Adhesions are quickly formed, and encysted purulent effusions frequently result. By the establishment of local peritonitis, ulcers of the stomach or intestine and abscesses of the liver are prevented from penetrating the abdominal cavity and causing a rapidly fatal general peritonitis.

General Chronic Peritonitis.—An acute general peritonitis may run a protracted course, become chronic, and cause sero-purulent collections, or it may be chronic from the onset. An extensive adhesive or sero-fibrinous peritonitis may become chronic, causing numerous adhesions and thickenings of the peritoneum, and a more or less abundant collection of fluid contained in the spaces formed by the adhesions. The fluid after a time usually becomes sero-purulent or purulent, and in the latter case may be converted into a cheesy mass. Coils of intestine are matted together, or very firm adhesions with organs or with the abdominal parietes occur. In all cases of chronic peritonitis there are extensive peritoneal adhesions and thickenings. When a considerable quantity of pus is circumscribed by fibrous septa, either an external opening takes place or it becomes encapsulated. In some cases of chronic general peritonitis, there is a gradual ascitic accumulation. In most cases, pigmented and hemorrhagic spots stud the thickened peritoneum. Local or circumscribed chronic peritonitis may be developed over an enlarged spleen, or a cirrhotic liver, or in connection with chronic intestinal diseases. Its anatomical changes are similar to those of general chronic peritonitis.

In *hemorrhagic* sub-acute peritonitis, the new tissue formations are exceedingly vascular, and the thin walls of the vessels may rupture. The new membrane may consist of one thin layer, or of several strata with effused blood between them. Sometimes the new tissue is infiltrated, and the entire surface of the peritoneum may assume a dark brown color, the fluid in its cavity having a distinct chocolate hue. This form of peritonitis is especially liable to occur with hypertrophic cirrhosis.

In *tubercular* peritonitis there may be only a few tubercular nodules on the surface of the peritoneum, or there may be a granular infiltration of the entire membrane. In its milder form only a few gray, semi-transparent tubercles will be found in that portion of the peritoneum which overlies intestinal ulcerations. In severe or extensive tubercular peritonitis, the surface of the peritoneum is studded with tubercular granulations, which are also disseminated through the new tissue formation, and in the subjacent peritoneal and subperitoneal tissue. The mesentery and omentum are also studded with granules. The adhesions formed in tubercular peritonitis divide the cavity into compartments, which contain the effusion. The effusion may be sero-fibrinous or purulent; in some instances it is hemorrhagic, and varies in color from a light pink to a deep chocolate. In very severe cases tubercular peritonitis is always hemorrhagic. Ecchymotic spots and petechiæ are frequently present in the new membrane. No form of diffuse peritonitis, except cancerous, causes such extensive thickenings, adhesions, and distortions as tubercular.

Cancerous Peritonitis.—Cancer of the peritoneum is rarely primary, but is propagated to the peritoneum from adjacent organs. When peritonitis is the result of cancer in the peritoneum, it commences with the primary cancerous developments, or is established when cancer of the abdominal or pelvic viscera reaches their surface and involves the peritoneum covering them. The cancerous developments may begin in the omentum and gradually involve the entire peritoneum. Cancerous peritonitis may begin as a diffused suppurative peritonitis, in connection with rapid cancerous developments in some of the abdominal viscera, especially in the uterus. Sometimes, in cancerous peritonitis, the peritoneum may be distended with a serous, lemon-colored or whey-like fluid, accompanied by a more or less abundant plastic exudation with hemorrhages into the exudation. The hemorrhage into the effusion colors it as well as the cancerous nodules on the surface of the peritoneum. Adhesions are formed as in the other varieties of peritonitis, and collections of fluid may thus become encapsulated. This variety of peritonitis is not only attended by the development of tough, leathery membranes, but entire organs may be enveloped by the new tissue formations; in these cases the mucous membrane of the intestinal tract is usually the seat of chronic enteritis.

Etiology.—Peritonitis may occur at any age, in the strong and robust as well as in the weak and feeble. It is met with more frequently in females than in males; certain localities predispose to it, and the tendency to it is greater in those suffering from chronic diseases. Rarely, if ever, is acute peritonitis of spontaneous origin. But the discovery of its cause during life is often very difficult, yet very important, for on the cause depends the prognosis and to some extent the treatment.

The exciting causes of acute peritonitis are: *first,* intestinal obstructions and perforations. Under this head may be included typhlitis and perityphlitis, with ulceration; rupture of hepatic and other abscesses; ulceration and rupture of the stomach, the gall or urinary bladder; rupture of hydatid and ovarian cysts; ulceration and perforation of the intestines in typhoid fever, syphilitic or tubercular intestinal ulcers; and the rupture of an abdominal aneurism. In rare instances, hydatids of the lung or purulent pleural accumulations open into the cavity of the abdomen and set up a diffuse peritonitis. Injections into the uterus may pass through the Fallopian tubes into the peritoneal cavity and cause peritonitis. Rupture of an organ from a blow or fall, and penetrating wounds of the abdomen, are causes of traumatic peritonitis. Abscesses of the abdominal parietes, of the vesiculæ seminales, or psoas and lumbar abscesses from caries and necrosis of the spine, ribs or pelvic bones, may open into the peritoneal cavity and cause general peritonitis.

Secondly, the extension to the peritoneum of inflammation of organs covered by peritoneum is a common cause of local peritonitis. In this class of cases the peritonitis is first local, and then it may become general. Inflammation of the stomach or intestines may, by extension, involve the peritoneum covering them. Peritonitis may result from extension of inflammation from the uterus and its appendages, liver, spleen and kidneys. In typhlitis, perityphlitis, proctitis, periproctitis and chronic ulcer of the rectum, peritonitis may occur by extension of inflammation without perforation. Venous thrombi, especially lymphangitis and phlebitis of the uterus, or severe contusions of the abdomen may cause peritonitis by extension. Intestinal intussusceptions, volvuli, herniæ, etc., quickly induce peritonitis even when no rupture has occurred. Gangrenous and inflammatory processes in the umbilical vessels often give rise to peritonitis in the newborn. In the very young, incomplete descent of the testicle may cause it. Diverticula from hernia of the mucous membrane of the lower bowel through the muscular coat, may become filled with fæces and excite peritonitis.[1]

Thirdly, in many instances acute general peritonitis is the immediate result of infection; pyæmia, septicæmia, and puerperal fever are the conditions in which infectious peritonitis is most likely to occur. Puerperal peritonitis may occur with or without pyæmia. Intra-uterine peritonitis can often be traced to a syphilitic taint, and to puerperal sepsis in the mother. Exposure to cold and wet rarely, if ever, directly causes peritonitis. Serous inflammations of a rheumatic character are very interesting in their combinations; we may find peritonitis with pericarditis and pleurisy, or with pneumonia and dysentery. Erysipelas has been complicated by peritonitis. Chronic general peritonitis may result from acute diffuse, or from acute local peritonitis, or from tubercle and cancer. It may be caused by long-standing ascites, in connection with cirrhosis of the liver and chronic splenitis. Chronic local peritonitis follows inflammatory conditions in organs which have a serous covering, by simple contiguity of tissue, as in hepatitis, hobnailed liver, enlarged spleen, chronic dysenteric ulcers, chronic typhlitis, etc., etc. Tumors may excite local chronic peritonitis when they are in contact with the peritoneum, as ovarian tumors. Chronic peritonitis has occurred, according to Virchow, in intra-uterine life. Extra-uterine pregnancy without rupture, when the fœtus undergoes degeneration, may lead to chronic peritonitis. Hemorrhagic peritonitis occurs most frequently with hypertrophic cirrhosis of the liver, Bright's disease, general tuberculosis, and acute articular rheumatism. Tubercular peritonitis is met with most frequently in early life, and cancerous peritonitis between the ages of forty and sixty-five.

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[1] American Clinical Lectures, page 281.

Symptoms.—The symptoms of acute peritonitis vary with its extent, severity, and the causes which produce it. If it is the result of intestinal perforation, its onset will be marked by excessive pain over the whole abdomen. In infectious peritonitis, the first symptom will be a severe chill. Peritonitis resulting from the extension of an already existing visceral inflammation begins with local and gradually increasing pain. All varieties of acute peritonitis from whatever cause are ushered in by *pain* as one of the earliest symptoms. The pain may be local or diffuse. In severe cases, if local at first, it becomes diffuse in a few hours. It is described as a cutting, burning pain, aggravated by pressure and by movements of the abdomen. The more sudden the onset, the more intense the pain. In some cases, the weight of the bedclothes cannot be borne. The pain causes the patient to remain motionless, he lies on his back, with the knees drawn up, the breathing is wholly thoracic, the respirations are rapid and superficial, and the face, by its pallid, drawn and anxious look, is almost diagnostic of the disease. In most cases, the pain is at first paroxysmal.

If the peritonitis is general the abdomen soon becomes distended and tympanitic, the tympanitis increasing as the disease advances. At the onset of acute peritonitis, the abdominal muscles are rigid and contracted; after this tonic rigidity they relax and allow of abdominal distention. Sometimes the distention is so great that the diaphragm is pushed up as far as the third or fourth rib, the lungs are compressed, and the heart, liver and spleen are displaced. In local acute peritonitis, the tympanitis is usually slight; in diffused it is excessive and increases the pain and causes dyspnœa, the respirations often being increased to forty or sixty per minute. As the intestines become distended with gas, percussion elicits a tympanitic note over the whole abdominal cavity. If there is a rapid effusion of serum, it will gravitate to the most dependent portion of the peritoneal cavity and an abnormal area of dulness will mark its position, the line of which will change with a change in the position of the patient. If a large amount of coagulable lymph is poured out over that portion of the peritoneum which covers the liver or spleen, a distinct fremitus may be communicated to the hand as it passes over the hepatic and splenic regions, accompanied by distinctly audible friction sounds.

The *temperature* in acute peritonitis has no typical range; it may not rise above the normal. In most cases it ranges from 102 to 103° F.; it is of the remittent type, being lowest in the morning. If recovery takes place, it gradually falls to normal. In fatal cases it may fall below the normal during the period of collapse. The pulse is accelerated, often reaching 140 per minute. For hours before a fatal issue it may beat 200 per minute. It is small, hard and wiry in character, and when very rapid is hardly perceptible at the wrist. In exceptional cases it is tolerably full and strong, and does not rise to more than 90 beats per minute.

Vomiting is a prominent symptom; if that portion of the peritoneum covering the stomach is first involved, it precedes all other symptoms. It usually comes on about the second day; the vomited matters at first consist of the contents of the stomach, later they are a mucus mingled with a spinach-green material, which by some is regarded as characteristic. Whenever stercoraceous vomiting occurs in peritonitis, it is evidence of intestinal obstruction, such an obstruction being the cause or the result of the peritonitis. Total paresis of the lower bowel in rare instances may cause stercoraceous vomiting when the muscular wall of the intestine above is still active. Sometimes there is constant nausea without vomiting; hiccough and gaseous eructations indicate that the diaphragmatic portion of the peritoneum is involved. The tongue is covered with a thick coating, and anorexia is present from the onset. Constipation due to paralysis of the muscular coat of the intestine is the rule, especially in the early stage of peritonitis. Yet diarrhœa may not only occur during the later stages of the disease, but it may exist throughout its entire course. In puerperal peritonitis there is usually watery diarrhœa, and diarrhœa is often present in the peritonitis of children.

The *urine* is scanty and deposits urates; "scalding" frequently occurs, and if the peritoneal covering of the bladder is involved there may be retention of urine or painful micturition. The tendency to heart failure and to collapse is one of the most striking characteristics of acute peritonitis. In all varieties it must be remembered that the disease rarely runs a typical course; even pain may be absent. A sudden collapse attended by a soft, feeble pulse and brown tongue, quickly terminating in death, may be followed by an autopsy which shows the intestines matted together by recent inflammatory products. When peritonitis follows intestinal perforation, all the symptoms from the onset are severe. The face quickly becomes haggard, drawn, and dejected; the eyes are sunken and surrounded by dark purple rings; the nose and cheeks are pinched, the lips are blue, the upper one being lifted and tightly drawn across the teeth, the voice becomes feeble, or the patient speaks in a husky whisper, the extremities are cold and covered with a clammy perspiration, the radial pulse is hardly perceptible, the respirations assume the type known as "Cheyne-Stokes" respiration, general cyanosis supervenes and death is reached within forty-eight hours. Sometimes death occurs within three or four hours from the shock of the perforation. The mind is usually clear throughout the entire course of the disease; in infectious peritonitis loss of consciousness, apathy, or delirium may precede death by a few hours. The pulse and the amount of cyanosis are measures of the heart failure. In cases where there is a large amount of fluid effusion the pain subsides with the occurrence of the effusion, and this sometimes leads to a mistake in prognosis on account of the supposed subsidence of the peritonitis. In suppurative peritonitis the pain is not infrequently absent, but typhoid symptoms are present from the onset, delirium is the rule rather than the exception, recurring rigors are common, the fever increases toward evening, and the pulse becomes very rapid. Occasionally in typhlitis, gastric ulcer, and intestinal perforation, the shock of the perforation, or the feeling as if something had suddenly burst, or been torn within the abdomen, is distinctly appreciated by the patient.

Local or circumscribed peritonitis usually pursues a sub-acute rather than an acute course. Chronic peritonitis (non-tubercular and non-cancerous) is usually the sequela of an acute attack. If convalescence is not established during the first week of an acute general peritonitis, the character of the inflammation changes and it becomes chronic. Rigors alternate with irregular sweats, and a steady increase in the size of the abdomen marks the passage from an acute to a chronic peritonitis. There is rapid loss of flesh and strength, and a marked diminution in the general vital powers. The face assumes the haggard, drawn look so often found with chronic abdominal disease. The intense pain of the acute attack subsides, and a "dull ache" with more or less tenderness remains. The pain assumes a colicky character and not infrequently is increased by taking food. The abdominal muscles remain rigid and tense. The temperature ranges from 99° to 104° F. The pulse continues rapid and feeble. There is anorexia and progressive exhaustion; diarrhœa alternates with constipation. Fluid accumulates in the peritoneal cavity, sufficient in some cases to cause dyspnœa. The thickenings and adhesions which develop may so interfere with the venous return that œdema, thrombosis and albuminuria may result. In latent general chronic peritonitis there may be large ascitic accumulations accompanied by abdominal tenderness, loss of appetite and progressive anæmia: The pulse is small and rapid, the vomiting is persistent, and with the accompanying diarrhœa exhausts the patient. Recurring attacks of acute local peritonitis hasten the fatal issue.

In tubercular peritonitis the pain is paroxysmal in character. Its onset is often sudden, attended by fever and well-marked constitutional disturbance, the pulse is rapid and feeble, there is nausea, vomiting and diarrhœa. The tongue is heavily coated, thirst is intense, and there is rapid loss of flesh and strength. The skin becomes harsh and dry. Typhoid symptoms appear early, fluids gradually accumulate in the peritoneal cavity and the patient dies of asthenia. Redness and œdema about the umbilicus are regarded as characteristic of tubercular peritonitis. In some cases the pain is so slight as to amount only to a sense of tension and fulness in the abdomen; and yet there may be a large effusion into the peritoneal cavity. The tongue becomes red and shining, the stomach is irritable; hectic fever is accompanied by profuse sweats during sleep, and the abdomen has a doughy feel. Some cases are unattended by ascites, and knots of intestine embedded in firm hard masses are felt in the region of the umbilicus. Friction sounds may be heard over these masses. Tubercular peritonitis may have for its chief and only symptoms, ascites, anæmia, and the evidences of general tuberculosis; its progress is interrupted, now there is marked improvement and cessation of all the abdominal symptoms, and then there follows a period when death seems imminent. As a rule, there is moderate fever and slight pain, with considerable ascites. The mesenteric glands are usually enlarged.

Cancerous peritonitis is attended by the same local symptoms as tubercular. Sometimes a tumor may be felt, especially in the region of the omentum and mesentery. There is always ascites ; the fluid collects gradually, and often in very large quantities ; constipation is more frequent than diarrhœa, and death is often the result of intestinal obstruction. In some cases the abdomen is very sensitive, and paroxysms of colicky pains are not infrequent. The temperature rarely reaches 100° F. If the peritonitis has extended from the stomach, liver or intestine, the symptoms of the primary disease will have been well defined before the development of the peritonitis. At any period in the course of cancerous peritonitis all the symptoms of acute general peritonitis are liable to be developed. The diagnosis rests on the presence of a gradually increasing tumor and the cancerous cachexia.

Differential Diagnosis.—Peritonitis may be mistaken for *colic, intestinal obstruction* (without peritonitis), *enteritis, abdominal neuralgia, hysteria, rheumatism* of the abdominal muscles, *renal* and *biliary colics,* and *suppurative cellulitis* of the abdominal walls. The ascites of chronic peritonitis may be mistaken for that of the last stages of *cirrhosis of the liver.* The differential diagnosis of colic, intestinal obstruction, and enteritis has already been given.

The pain in abdominal *neuralgia* simulates that produced by a tightly drawn cord about the abdomen, and follows the course of the genito-crural nerve. There is tenderness on pressure only at the point of exit of the nerve from the spine. There is no tympanitis, no ascites, no rise of temperature, or acceleration of the pulse, and no signs of collapse. The muscular rigidity of commencing peritonitis is absent.

In *hysteria,* the patient is ready to complain of increased pain before the hand touches the abdomen, yet the firmest pressure does not increase the pain if the attention of the patient is engaged. The pulse, temperature, and signs of collapse of peritonitis are absent, the countenance is not that of peritonitis, and there is present the globus hystericus, and the attack is followed by the passage of a large quantity of watery, straw-colored urine.

In *rheumatism* of the abdominal muscles, the pain and tenderness are most intense at the origin and insertion of the muscles. There is no rise of temperature, no vomiting, and no signs of collapse ; the pulse is normal, and there will be a history of acute or sub-acute articular rheumatism.

In the passage of a *gall-stone,* and in *renal colic,* the patient throws himself about in excruciating agony, and the pain is referred to the region of the common bile-duct, or to the course of the ureter. In the passage of a gall-stone, it is paroxysmal in character, and will shoot back from the margin of the ribs over the gall-bladder to the spinal column. If it continues twenty-four hours, the patient becomes jaundiced. In renal colic the pain radiates from the kidney along the ureter to the testicle, which is retracted. Both are accompanied by characteristic changes in the urine or fæces, neither is attended by rise of temperature or great acceleration of pulse, and there is no tympanitis or tenderness on firm pressure in either.

Suppuration of the abdominal parietes is at first difficult to distinguish from peritonitis, but after the first two days the superficial swelling and the absence of the constitutional symptoms of peritonitis establish the diagnosis.

Prognosis.—Acute general peritonitis is a very fatal disease. Its average duration is from four to eight days; death may occur in a few hours, or be delayed two or three weeks. The prognosis in any case is to a great extent determined by its cause; it is most unfavorable when it results from perforation, intestinal obstruction, or sepsis. General puerperal peritonitis is almost always fatal. The presence of typhoid symptoms, a very rapid and feeble pulse, cold extremities, with the other symptoms of impending collapse, indicate an unfavorable termination. Peritonitis from rupture of an organ is always fatal. The prognosis is favorable when the peritonitis is due to extension of inflammation from a viscus. When the pain and vomiting cease, the tympanitis subsides, the pulse diminishes in frequency, the temperature reaches the normal, and the patient is able to turn in bed, a favorable termination is to be expected. Chronic diffuse peritonitis in children, unless purulent, usually terminates in recovery. Tubercular peritonitis, after weeks and months of anæmia and exhaustion, terminates in death. The same is true of carcinomatous peritonitis. Death in acute peritonitis may result from shock, from asthenia with typhoid symptoms, and from exhaustion. Among its sequelæ are collections of pus, stenosis or complete obstruction of the intestine, pyæmia, and septicæmia. Permanent jaundice may result from narrowing of the bile duct by the contraction of new tissue formations in the transverse fissure.

Treatment.—Acute peritonitis is a severe, rapidly progressive, and dangerous inflammation, and on this account has always been treated heroically. Formerly patients with acute peritonitis were subjected to excessive bleedings, tartar emetic was administered in nauseating doses, and to prolong the effects of the bleeding, and as an adjunct to these calomel was given for its specific effect. At the same time many physicians of recognized authority were eager to obtain the purgative effects of cathartics, and for this purpose recommended and administered large doses of drastic purgatives. Local bleeding by leeches is often of great service in local peritonitis, but it should be resorted to only at the very onset of the attack in the strong and robust. Tartar emetic and calomel, so highly regarded as antiplastics, have fallen into disuse. While acute peritonitis is progressing the bowels cannot be moved, and no benefit would result if they were; so that under no circumstance should there be an attempt at purgation.

The plan of treatment which I have followed for years—a plan which gains in favor with me with every new experience—is the *opium plan.* Prof. Alonzo Clark first developed this plan and brought it to the notice of the profession. The details of it are as follows:—as soon as the unmistakable symptoms of peritonitis are developed, administer at one dose from two to five grains of opium or one-half to one grain of morphine. The exact quantity in each case is to be determined by the condition of the patient; the rule is to bring the patient as soon as possible fully under the influence of the drug. In the treatment of this disease, it will be observed how greatly pain and inflammation modify the effects of this powerful drug. I have administered to patients with peritonitis four grains of opium every two hours for twenty-four hours, and then have obtained only a moderate effect of the drug. The point which must be reached in its administration is moderate narcotism, in which state the patient must be kept, not only until all pain and tenderness have subsided, but until the pulse has reached its normal standard and the tympanitis has entirely subsided. The question arises: what are the indications which are to govern the administration of each dose of opium? One must be prepared at the commencement of the treatment of a case of peritonitis, according to this plan, to be present and decide upon the quantity of opium to be given at each dose, until the patient has fully convalesced. It cannot be trusted to attendants, however intelligent they may be. As the patient is brought fully under the influence of the opiate, it will be noticed that the entire surface of the body becomes bathed in a profuse perspiration. In twenty-four hours a rash, due to the opium, will make its appearance on the surface and neck; this is accompanied by an itching of the surface and a constant disposition to rub the nose. The pupils become contracted, the eyes suffused, the countenance assumes a dull expression, and there is a constant irresistible disposition to sleep. The pulse becomes lessened in frequency and force, and the respirations, which, before the administration of the opium, may have ranged from 40 to 60 in a minute, become less and less frequent as the patient comes fully under its influence, until they are only twelve in a minute. Now the greatest care is to be exercised in the administration of the opium; the patient is in the condition in which it is desirable to keep him. By holding him in this state of semi-narcotism, all will be accomplished that can be by the opium plan of treatment, and with the respiration at twelve per minute the patient is perfectly safe. The amount of sleep is not to be taken into account, but the profoundness of the slumber is of great importance. If it is found difficult to arouse the patient, the administration of the opium must be stopped until he can be easily aroused. If by mistake or negligence the patient becomes fully narcotized, the respirations will sometimes diminish in frequency to seven or even five in a minute. In this extremity, if the administration of opium be stopped, the patient will usually rally from its effects after a few hours; but avoid extremes, endeavor to keep the patient in a quiet sleep, not profound, but one from which he can be easily aroused. When the pulse begins to diminish in frequency and becomes fuller, one may be certain that he is controlling the peritonitis, and as it is controlled the patient will become more and more susceptible to the influence of the opium. Slowness of respiration and absence of pain cannot be relied on as sure indications that the opium is controlling the inflammatory action; but a diminution in the frequency of the pulse, and a subsidence of the tympanitis are sure indications that the peritonitis is arrested, and that ultimate recovery is probable. In most cases, if an acute peritonitis does not depend for its exciting cause upon the escape of intestinal gases into the peritoneal cavity, or upon complete intestinal obstruction, the inflammatory action can be controlled within forty-eight hours from the commencement of the attack by adopting, within twelve hours, this plan of treatment. It must, however, be continued four or five days longer, for there is still danger of a renewal of the inflammation. As the condition of the patient demands less opium, the dose may be diminished, or the interval between the doses lengthened. A safe rule by which to be guided is that, so long as any tympanitis exists, the opium should be continued.

When convalescence is fully established, one should not be too anxious to overcome the constipation which usually exists, for a free, spontaneous movement of the bowels generally follows a complete subsidence of the peritonitis. Wait at least a week for this result before administering a cathartic, and then, if necessary, employ one mild in its action, such as castor-oil. Warm poultices over the abdomen are usually the only local application which I have employed. It is claimed by some that cold compresses have a much more beneficial effect than warm applications. My experience leads me to doubt the utility of the former, while the latter are far safer, and I believe equally efficacious. It has been stated that when the peritonitis becomes general, excessive gaseous distention of the intestines occurs, and this distention greatly increases the danger to the patient; under such circumstances I have recently resorted to minute puncturing of the distended intestine with a hypodermic or a very small aspirating needle, and have thus relieved the intestinal distention by allowing the gas to escape. By so doing, not only is the tension of the peritoneum (which becomes an exciting cause of the peritonitis) relieved, but the principal obstruction to the respiration is removed, and thus cyanosis is diminished. Immediate and marked relief is afforded by such a procedure, and as thus far I have had no bad results follow, I am disposed to resort to it in all cases where the abdomen becomes excessively distended and tympanitic. I remember one case in which the gaseous distention was excessive, and the peritonitis was supposed to be due to strangulation of a portion of intestine from old peritoneal adhesions, where the relief of the distended intestine by puncture was soon followed by a removal of the intestinal obstruction and the rapid recovery of the patient. From this circumstance I can readily understand how a portion of intestine that was partially constricted by a band of adhesion might become completely obstructed at the point of stricture by a rapid gaseous distention of the intestine above the point of constriction, and the relief of the intestinal distention by puncture would very likely liberate the constricted portion and thus overcome the strangulation, and so, perhaps, save the life of the patient.

The necessity of absolute quiet, and of the frequent administration of nourishment and sometimes of stimulants, in small quantities, to this class of patients, is apparent. Preceding and during the stage of plastic exudation, large doses of quinine are beneficial; but little nutriment should be administered, and that only in a fluid and a highly condensed form. Cracked ice may be given to relieve the thirst, and, if there are signs of asthenia, iced champagne or brandy should be given in small doses. If hiccough is distressing, it should be relieved temporarily by the inhalation of chloroform. Vomiting is sometimes allayed by carbonated water, cracked ice and champagne, or hydrocyanic acid. Turpentine, as an injection and employed locally as an embrocation, will sometimes relieve the tympanitis. With the asthenic form of peritonitis, a stimulating plan of treatment should be em ployed with the opium. In puerperal peritonitis, great attention should be paid to the condition of the uterus and its appendages. Chronic peritonitis is treated by local applications of iodine and mercury, and by the internal use of iodide of potassium. Its products may be removed by tapping. The nutrition of the patient must be carried to the highest point. Tubercular peritonitis demands small doses of opium, warm anodyne applications, and the administration of tonics, cod-liver oil especially. The treatment of cancerous peritonitis is purely symptomatic; nausea and attacks of diarrhœa and constipation must be promptly relieved. Narcotics may be given for the sleeplessness. Concerning the prophylactic and sanitary treatment of puerperal peritonitis, the reader is referred to obstetrical works.

ASCITES.

(*Abdominal Dropsy.*)

Ascites is a local dropsy,—an accumulation of serum in the peritoneal cavity. It has also been called *peritoneal dropsy, dropsy of the abdomen,* and *hydro-peritoneum.* The circumstances under which it occurs are similar to those which allow of general dropsy—viz.: obstruction to the capillary or lymphatic circulation of the peritoneum, a diminished amount of albumen in the blood, and degenerations of the peritoneum. Those hydræmic conditions which accompany exhausting chronic diseases, especially diseases of the kidneys, will induce it. One or several of these conditions may be present in the same case.

Morbid Anatomy.—The amount of fluid present in ascites may vary from a few ounces to four or five gallons. In consistency it may be viscid or watery. It is usually of a light straw color, having a faint greenish opalescent tint. It may be opaque and dark, from admixture of blood. With disease of the lymphatics it is milky and opalescent. Sometimes it does not differ in appearance from pure water. It is alkaline in reaction, and may contain albumen, blood, fibrin, fibrinogen, bile-pigments, kreatin, kreatinin, lymph flocculi, and bile acids. Albuminate of soda, leucocytes and pus cells are its occasional ingredients. The endothelia of the peritoneum are turbid, thick, and in various stages of fatty degeneration. The sub-serous tissue is thickened, and the whole membrane has the look and feel of being water-logged. The blood changes that cause it consist chiefly in a diminution of albumen and an increase of water. Compression, dislocation, and diminished function of the abdominal viscera are the results of the ascitic accumulation.

Etiology.—Ascites may be a late symptom of general dropsy. In most other instances it results from damming back of the blood in the portal tributaries, from pressure on the portal vein—either from hepatic and abdominal tumors, or from a diseased condition of the liver substance—as in cirrhosis, waxy degeneration, abscess, hepatic atrophy, portal thrombosis, enlarged lymphatics in the transverse fissure, and the constrictions due to perihepatitis. These all mechanically impede the blood current in the portal vessels. Diseases of the heart or lungs which interfere with the normal flow of the blood from the cavæ will induce it in connection with general dropsy ;—under this head are included tricuspid obstruction and insufficiency, chronic bronchitis and emphysema, fibrous phthisis, and certain forms of mediastinal tumors. Anæmia, hydræmia, chlorosis, malarial cachexia, purpura, chronic arsenical poisoning, scurvy and chronic Bright's disease, producing hydræmia—and old age or great exhaustion without structural disease—lead to what is often called *asthenic ascites* or *cachectic dropsy*. Peritoneal dropsy not infrequently accompanies extensive degeneration of the peritoneum, such as tubercle and carcinoma. Finally, ascites may occur from unknown causes—from taking cold, after suppression of the menses, after the sudden disappearance of acute and chronic cutaneous eruptions and ulcers, and perhaps from atmospheric causes.[1] It has been suggested that malignant disease of the ovaries and other pelvic organs, and of the mesenteric and retro-peritoneal glands, obstructs the capillaries and the lymphatic orifices, increasing the functional activity of the endothelia, and thus induces ascites.[2]

Symptoms.—The first sign of ascites is a gradual increase in the size of the abdomen. The enlargement in simple ascites takes place without pain, tenderness, or local subjective symptoms. There is a feeling of fulness, and the patient is rendered uncomfortable by pressure of the fluid. The respiratory movements are interfered with, and dyspnœa soon results. The functions of the stomach may be disturbed, and there may be vomiting, anorexia, and perhaps hæmatemesis. Flatulence and diarrhœa are frequently present, but when the accumulation of fluid is large it produces colicky pains, and often obstinate constipation. All these symptoms are relieved as soon as the fluid is removed. Gradually the dyspnœa increases, the patient walks with difficulty, with the legs spread widely apart ; the urinary secretion is diminished from the pressure on the kidneys and renal vessels. The recumbent posture greatly aggravates the dyspnœa. The skin and mucous membranes become dry ; the liver and pelvic viscera are displaced ; the heart and lungs are pushed upward, and the skin over the abdomen becomes tense and shining. The umbilicus is bulged out in the form of a globular tumor. The superficial veins are enlarged and tortuous. If the inguinal canal is open, fluid may pass into the scrotum ; and excessive ascites, by pressure on the vena cava, causes œdema of the feet and legs. In hepatic diseases the fluid is chiefly confined to the abdomen, but in cardiac and pulmonary dropsies the fluid accumulates first about the feet and extends upward, and the abdominal dropsy is then a part of a general anasarca. In hepatic dropsies the extremities emaciate while the abdomen enlarges ; the skin has a muddy jaundiced hue, and the patient becomes exhausted and apathetic. Jaundice, uræmia, delirium, convulsions, coma, and cholæmia are prominent symptoms as death approaches.

Physical Signs.—The physical examination of the abdomen is most important in the diagnosis of ascites.

Inspection.—The abdomen, if distended with fluid, presents the appearance of a globular or dome-like tumor, the false ribs are elevated and pressed out, and the superficial veins are visible and prominent. The circumferential measurement of the abdomen will often be three times as great as normal. If the effusion is moderate, the shape of the abdomen changes with a change of the position of the patient: it broadens when he lies on his back, and when he stands the enlargement will be confined to the lower portion. The fluid always gravitates to the most dependent portion.

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[1] Wagner, Gen. Path., pp. 234-5.

[2] Oppolzer.

Palpation.—Fluctuation is obtained when the level of the fluid is above the pelvic brim. To obtain the wave most distinctly place the patient on his back, place the flat of the hand on one side of the broadened abdomen, and with the other hand give one smart tap at a point opposite; the impulse of the blow will be felt by the palm of the hand.

Percussion.—There will be flatness below the level of the fluid, and tympanitic resonance above. The line of dulness changes with the change of position, and accurately measures the amount of fluid. When only a small amount of fluid is present the physical signs of its presence are commonly obtained by placing the patient in the "knee-elbow" position.

Differential Diagnosis.—Ascites may be mistaken for *ovarian dropsy, distended bladder, pregnancy, hydatid cysts of the liver*, and *enlargement of the spleen*. It is important in making a differential diagnosis between ascites and ovarian dropsy to have a perfect history of the case. The abdominal enlargement in ascites is uniform, in ovarian dropsy it is irregular. Ascites, however slight, begins at the most dependent portion of the abdomen, while ovarian dropsy begins in one of the iliac fossæ and gradually extends upwards toward the umbilicus. With every change of position, in ascites, the line of dulness changes; a large ovarian cyst is to be recognized by its fixed position and non-gravitation of its fluid. In ascites there is fluctuation on palpation; in ovarian dropsy, fluctuation is absent or localized. The abdomen is usually tympanitic above the level of the fluid and flat below in ascites, while in ovarian tumor there is often a tympanitic percussion sound at the most dependent portion of the abdominal cavity. In ovarian dropsy the outline of the cyst is generally appreciable, except in very large tumors where the peculiar form of the cyst may be lost, but a rectal or vaginal exploration will generally at once remove all doubts. In ascites there will generally be a history of liver, heart, or kidney disease, and the uterine organs and functions will be normal. On tapping the abdomen a serous fluid will be withdrawn in ascites; in ovarian tumors, it will be a dark, highly albuminous fluid and contain granular non-nucleated cells, supposed to be characteristic.

A distended and *sacculated bladder* may be mistaken for dropsy, but the introduction of the catheter will decide the question.

Pregnancy will afford ballottement, placental bruit, the sounds of the fœtal heart, and will be accompanied by distinct mammary changes. The uterine tumor can be distinctly mapped out, and a vaginal examination combined with external palpation will rarely fail to make a differential diagnosis between it and ascites.

An hydatid cyst of the liver produces flatness undeviating in area, which gradually extends from above downwards, and seldom reaches the pelvic brim. Hydatids produce hydatid fremitus on percussion, which is characteristic. Again, on withdrawal of the fluid, a miscroscopical examination will often discover the hooklets of the echinococci.

Enlargement of the spleen is unsymmetrical; the tumor is fixed, there is no tympanitis, no fluctuation, and the boundaries of the enlarged organ can be mapped out on palpation and percussion. Usually the notch at the anterior border of the spleen is so distinct that it at once indicates the gland.

Prognosis.—The prognosis depends upon the conditions under which the ascites occurs; if it is dependent upon organic disease of the liver, heart, or kidney, the prognosis is unfavorable, but when it is not dependent upon structural visceral lesions, *e. g.*, idiopathic and anæmic ascites, the prognosis is good. The ascitic accumulation may take place rapidly, or weeks or months may elapse before the cavity of the abdomen is distended. The average duration of hepatic ascites is about six months. So long as the cause remains, the fluid will accumulate. Ascites may terminate in recovery by the spontaneous or mechanical removal of the fluid, or by the removal of its cause, or it may terminate in death from complications, as peritonitis, albuminuria or heart-failure, or from pure slow asthenia.

Treatment.—The first and most important thing in the treatment of ascites is to discover the cause, and either to remove or palliate it. In most cases the treatment merges into the treatment of the diseased conditions which produce it. In all cases the diet should be highly nutritious and concentrated; as little fluid as possible should be taken. The continued use of powerful diuretics and hydragogue cathartics usually does harm. They weaken the patient and often favor rather than retard the ascitic accumulation. Elaterium is the most efficient drastic cathartic, the potash salts, nitre, squills and juniper are the most efficient diuretics. Jaborandi has recently been much employed for the removal of dropsical accumulations. In most cases these accumulations can be rapidly removed by this drug, but my own experience leads me to the conclusion that it hastens rather than retards the fatal issue. Hot-air baths should never be employed for the removal of ascitic accumulations.

Paracentesis abdominis will have to be resorted to sooner or later in these cases, but the rule is to postpone it as long as possible. I am, however, in favor of tapping before the accumulated fluid has caused pressure upon the viscera. I am convinced that whenever fluid accumulation takes place in the peritoneal cavity, tapping should be promptly resorted to, unless the cause can be removed by mild cathartics or non-stimulating diuretics; and the number of recoveries and the prolongations of life which have followed this course in my experience cause me unequivocally to recommend it in preference to the prolonged use of those remedial measures which increase the discharges from the skin, kidneys and bowels. In a large number of cases, improvement of the patient's general health by tonics, of which quinine, iron and cod-liver oil are the best, is followed by subsidence of the dropsy, and its return is also prevented for a long time after its removal by tapping.

DISEASES OF THE LIVER.

Diseases of the liver may be classified under the following heads:

- I. *Hyperæmia:—*
  - *a. Active or Fluxion.*
  - *b. Passive or Congestion.*
- II. *Inflammations:—*
  - *a. Interstitial Hepatitis* **or** *Cirrhosis.*
  - *b. Circumscribed Hepatitis or Abscess.*
  - *c. Diffused Hepatitis or Acute Yellow Atrophy.*
  - *Perihepatitis, Local or General.*
  - *Pylephlebitis, Adhesive and Suppurative.*
- III. *Degenerations:—*
  - *Amyloid or* ***Lardaceous.***
  - *Fatty.*
  - *Pigmentary.*
  - *Atrophy.*
- IV. *New Growths:—*
  - *Cancer.*
  - *Gummata.*
  - *Hydatids.*
  - *Tubercle.*
- V. *Jaundice, Hepatogenous and Hematogenous.*
- VI. *Diseases of the Gall Ducts and Gall Bladder.*
- VII. *Functional Derangements.*

ACTIVE HYPERÆMIA OF THE LIVER.

Active hyperæmia of the liver is an abnormal determination of blood to the organ. It may be acute or chronic.

Morbid Anatomy.—A liver that is the seat of active hyperæmia is more or less enlarged in all directions. Its color varies from a light to a dark red. It has a firmer feel than normal, although its consistency is really diminished. The organ is heavier and smoother than normal, its surface presenting a peculiar shining appearance.

On section, its substance shows a uniform red color, blood flows freely over its cut surface, from the arteries and capillaries which are dilated and sometimes tortuous. When the hyperæmia is intense, the glandular substance of the organ is compressed and there may be evidences of sub-peritoneal effusion. So intense may be the hyperæmia that hemorrhagic softening and apoplectic extravasation result, and isolated clots or an unbroken layer of coagulated blood may be found under its serous covering.

In *chronic hyperæmia* the liver is often found in a state of partial fatty degeneration, somewhat softened, and of a light red or yellow color. In rare instances, chronic hyperæmia may lead to induration and incipient cirrhosis. In the severer types abscesses may be found, and the infiltration of a substance resembling albumen has in some cases advanced so far as to give distinct *colloid degeneration.* In syphilitic new-born children, active hepatic hyperæmia is sometimes found associated with a peculiar plastic exudation. It is important to remember that the normal hepatic hyperæmia temporarily developed after hearty meals or the free use of stimulants may be mistaken for active hyperæmia. Both acute and chronic hyperæmia of the liver may be associated with catarrh of the bile ducts.

Etiology.—There is a normal functional hyperæmia of the liver induced by an unusually large meal, or one very rich in hydrocarbons, or by the free use of wines: this hyperæmia is due to increased blood pressure in the vena portæ; it becomes abnormal in those who daily indulge in eating to excess, especially if they lead sedentary lives. If the liver-tissue, which supports the walls of the capillary vessels, becomes relaxed, there will be an abnormal afflux of blood to the organ. This is the case in traumatic hyperæmia, where a blow over the viscus causes a localized fluxion. Any inflammation or growth causing softening of the parenchyma will induce it. The action of drugs, spices and alcohol is best explained on this basis. Intense hepatic hyperæmia may be caused by miasmatic influences, malaria, and other blood-poisons. Under the latter head is included a peculiar active hyperæmia which occurs in the livers of syphilitic children, and in secondary syphilis of adults. High temperature undoubtedly gives rise to active hepatic hyperæmia, especially when it is associated either with acute or chronic malarial infection. Vaso-motor disturbances may undoubtedly lead to active hepatic hyperæmia. It sometimes occurs during and after pregnancy from some unknown cause; also before the establishment of the menses, and during the menopause. Capillary embolism may cause localized hepatic hyperæmia.

Symptoms.—Active hyperæmia of the liver is usually attended by a sense of weight and constriction in the right hypochondrium, with some tenderness on pressure under the free border of the ribs. In active malarial hyperæmia, there is also gastro-intestinal catarrh, nausea, vomiting, diarrhœa, and slight jaundice. There is a bitter taste in the mouth, loss of appetite, coated tongue, drowsiness and apathy. Headache is frequent, and the patient complains of pain shooting up the right side to the right shoulder. This pain is due to pressure on the phrenic nerve, and is more intense after meals and when lying on the left side. A sense of dizziness comes on when the patient assumes any other position than on the back or right side. It is more or less increased by pressure upward against the liver. In severe cases of malarial hyperæmia, or when it is associated with extensive blood changes, such as scurvy, the symptoms are often masked by those of the condition with which it occurs.

Physical Signs.—*Inspection* in severe cases may show bulging of the right hypochondrium, and loss of motion of the lower ribs on the right side.

On palpation the liver is found enlarged and smooth, and its free border is felt below the ribs; firm pressure against its under surface causes pain.

Percussion.—The area of hepatic dulness is increased in every direction, but more vertically than laterally.

Differential Diagnosis.—A severe active hyperæmia may be mistaken for *circumscribed hepatitis* with *abscess*. In circumscribed hepatitis there is acceleration of the pulse, rigors followed by a slight rise of temperature, and localized pain. Recurring chills and sweats indicate the formation of pus. In abscess the hepatic enlargement is irregular, while in active hyperæmia it is uniform. If the case is seen early, and the enlargement is carefully followed, in hyperæmia it will be seen to take place rapidly, while in abscess it will be slow. The hepatic enlargement from active hyperæmia may be distinguished from *displacement* of the *liver* downwards, by the fact that, although its free border may extend far below the free border of the ribs, the normal area of hepatic dulness is not increased.

Prognosis.—Active hyperæmia generally subsides as rapidly as it occurs. The only danger is that the causes which produce it may be continued, and lead to some form of hepatic degeneration.

Treatment.—The main indication in the treatment of this condition is to remove its cause. When high living and alcoholic stimulants cause it, restrict the diet and stop the alcohol. When it occurs from prolonged high temperature, or from malarial influences, a change of residence is the only remedy. An excess of blood in the liver may be temporarily removed by saline or mercurial purges, by taraxacum or podophyllum ; their action will be increased by the application of one or two leeches about the anus. In active malarial hyperæmia, the mercurial purges and leeches may be followed by full doses of quinine. Turpentine stupes may be applied over a very tender liver. When there is gastro-intestinal catarrh with diarrhœa, chloride of ammonium and ipecacuanha will be found of service. In those who have a predisposition to active hepatic hyperæmia, the daily use of mineral waters will be found of service.

PASSIVE HYPERÆMIA OF THE LIVER.

Passive or mechanical hepatic hyperæmia ("congestion of the liver") consists in an excess of blood, chiefly in the portal veins, with a slowed current.

Morbid Anatomy.—A congested liver, in its early stage, is larger, heavier and darker in color than the normal liver, the extent of the increase in size corresponding to the degree of the congestion. The capsule may be stretched tightly over the enlarged organ, and present a shining appearance. The consistency of the organ is increased, frequently amounting to a stony hardness.

On section, the cut surface appears mottled, rarely uniformly red in color; the small dark spots seen upon its cut surface are the enlarged and thickened veins in the centre of the liver lobules, and as the return of blood by these veins is impeded, the surrounding cells undergo atrophy, and a granular pigment is deposited about the *venæ centrales.* This change in color is made more apparent by a deposit of fat globules in the periphery of the lobules, which causes a dirty white ring around the dark central spot. Occasionally there are yellowish spots about the central vein due either to a catarrh in, or obstruction of the bile ducts, or to distention of the minute ramifications of the portal vein. In the *advanced stage* of hepatic congestion, the liver is diminished in size and has a peculiar hard feel. On section, it presents the characteristic "nutmeg" appearance, which has been called the red granular liver. The processes which were established in the earlier stage of the congestion, and the new connective-tissue which has been developed in the interlobular spaces, diminish the parenchyma of the organ.[1] The rootlets of the central vein become dilated and hypertrophied and crowd upon the liver cells; thus the central spot spreads nearly to the periphery of the lobules. Atrophy of the cells grouped around the central vein occurs, and a soft pultaceous mass, in which appear new vessels, takes their place. The cells at the periphery are in a state of fatty infiltration, and connective-tissue passing inward from the interlobular spaces produces the contracted, stony hard liver. In connection with these changes in the liver, the mucous membrane of the stomach is usually the seat of chronic catarrh, and the spleen is enlarged.

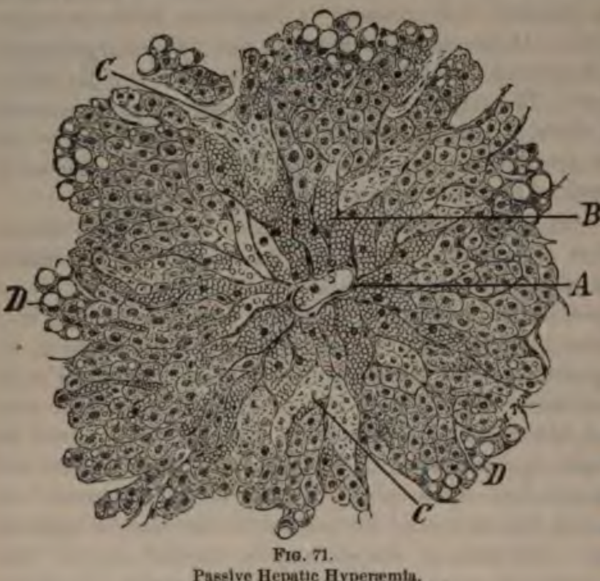

FIG. 71.
Passive Hepatic Hyperæmia.
Section of liver showing a single lobule.

A. Central vein of lobule.
B. Area of congestion.—Vessels filled with blood, crowding the hepatic cells.
C. Atrophied liver cells.
D. Commencing fatty infiltration of cells in peripheral zone. × 350.

Etiology.—The causes of hepatic congestion are mainly included under the head of impeded venous return. Heart disease is the most frequent cause of such obstruction. The damming back of the blood in the hepatic veins is the necessary result of tricuspid insufficiency, and of right heart failure. When right ventricular hypertrophy fails to compensate for valvular lesions in the left heart, or when disease of the lungs, as emphysema and chronic pleurisy, obstructs the blood current in the pulmonary artery so that the right ventricle is unable to empty itself, engorgement of the hepatic veins necessarily follows. The absence of valves in these veins, and the fact that they cannot collapse, favor this result. In the same way enfeebled heart power, occurring in the course of exhausting diseases, causes congestion of the liver. Habitual constipation and a sedentary mode of life, either singly or combined, may produce it. The sudden suppression of long-continued hemorrhages, as menorrhagia, or bleeding hemorrhoids, may lead to passive hyperæmia of the liver. A large mediastinal tumor, such as a thoracic aneurism, may also produce it by pressure on the cava.

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[1] Atrophy is *chronic congestion* with dilatation of the central vessels and their radicles.

Symptoms.—As there is usually some derangement in the circulation of the thoracic organs, the early symptoms are very apt to be confounded with those of cardiac or pulmonary disease. But soon slight jaundice follows the headache, drowsiness and apathy, and it lacks the peculiar greenish hue of that which sometimes accompanies the cyanosis of long-standing heart disease. Gastric catarrh will usually attend these symptoms, marked by loss of appetite, nausea, and vomiting. In the "India Liver" there is anæmia, and soon a cachexia is developed. The skin is dry and harsh. Later, hemorrhoids appear, and after a paroxysm of dyspnœa and cyanosis the hepatic dulness is markedly increased. In severe cases hæmatemesis may occur. If congestion has reached the stage of induration, the gastric symptoms become greatly aggravated, and tympanitis, gastric hemorrhage, and general dropsy occur. The bowels become constipated, and the fæces clay-colored. The urine is scanty, high-colored, and usually presents traces of albumen ; it is loaded with lithates. These patients finally become irritable, and are subject to fits of palpitation and irregularities of the pulse.

Physical Signs.—*Inspection* may show slight bulging of the right hypochondrium and some restriction in the movements of the lower portion of the right thoracic walls.

Palpation.—In its early stage, the free border of the liver is readily felt below the margin of the ribs ; it is smooth and tender. Later the organ is diminished in size, but its free margin can still be felt, and is hard and uneven.

Percussion.—At its commencement the normal hepatic dulness is increased in every direction, and firm percussion elicits pain. In advanced cases the area of hepatic dulness is uniformly diminished. It is always important to subject the chest to a thorough physical examination to determine the presence or absence of pulmonary or cardiac disease.

Differential Diagnosis.—Congestion of the liver may be so masked by the primary disease which produces it, that it will be overlooked, but it will rarely be confounded with any other form of hepatic disease.

Prognosis.—The prognosis depends upon the condition which causes it. If constipation and a sedentary life cause it, the prognosis is favorable. In chronic pleurisy and emphysema it can only partially be relieved. When it is associated with extreme cardiac disease, recovery is impossible and relief is only temporary.

Treatment.—When the symptoms which attend congestion of the liver are urgent, a mercurial purge or a brisk saline cathartic with the application of a few leeches about the anus will give relief. If the symptoms are not urgent, a mild laxative followed by a course of mineral waters will be beneficial. If the gastric symptoms are severe leeches may be applied over the stomach. The diet must be as free from carbo-hydrates as is compatible with nutrition. Nitro-muriatic acid internally and externally is recommended by English and East Indian physicians. Chloride of ammonium and iodide of potassium are often advantageous, reducing the enlarged organs. In some cases of extensive cardiac disease, mineral waters are *not well borne;* and although digitalis will relieve the congestion, it is apt to interfere with digestion. Each case is peculiar and requires its special treatment, which at best is only palliative.

INTERSTITIAL HEPATITIS.

(*Cirrhosis.*)

Interstitial hepatitis is an inflammation of the connective-tissue of the liver. It has been variously named sclerosis of the liver, *cirrhosis*, the "gin-drinker's" liver, the "hob-nailed" liver, granular, and gouty liver. Cirrhosis of the liver, the name most commonly used, was first applied by Laënnec. It means yellow, referring to the color, and not to the consistence of the organ.

Morbid Anatomy.—The anatomical changes in interstitial hepatitis begin in the connective-tissue covering the smaller twigs of the vena portæ, and gradually extend to its larger branches. The hyperplastic process consists in the formation of a soft, red, pulpy or gelatinous mass, which makes its appearance first in the portal canals. This mass consists of an immense number of small round cells, which soon undergo fibrilization and form new connective-tissue. The new tissue contracts; this contraction may be limited, or it may involve the whole organ. In the latter case, both stages—namely, the stage of enlargement due to the hyperplasia, and the stage of diminution in size, the result of connective-tissue contraction—may exist in the same liver at the same time. The new tissue, contracting, presses on the portal capillaries and liver-cells, and gradually encroaches on the intra-lobular structures, causing atrophy and disappearance of the cells at the periphery of the lobules, while those at the centre undergo fatty change, the result of defective nutrition. Sometimes the cells at the periphery undergo fatty change before they atrophy. The bile-ducts and hepatic capillaries suffer from compression, and the cells around the central vein are bile-stained.

In the first stage of cirrhosis, the liver is slightly enlarged, noticeably in the vertical direction; it is resistant and hard to the feel, the edges are rounded and smooth, and the capsule becomes opaque and thickened. Upon the capsule are numerous small flattened projections, which are, however, not sufficiently prominent to destroy the smoothness. In the first stage the liver is uniformly enlarged and hyperæmic. In the second stage it is smaller than normal, the left lobe usually being but a caudal-like appendage to the right, which is nearly globular in shape. The whole organ presents a hob-nailed appearance, and is hard, rough, leathery and granular. The serous covering assumes a dull gray color, and fibrous bands bind the organ to the adjacent parts, especially to the diaphragm. In syphilitic cirrhosis these changes are in patches, which are large and well defined.

On section the liver-tissue, during the first stage, is extremely hyperæmic. The new cell growth around the branches of the portal vein and between the lobules has a pulpy, fleshy look. In the second stage, the liver gives a cartilaginous feel to the knife, and the capsule is thickened and resistant. The cut surface presents a yellow, mottled appearance, the mottling being due to three changes: first, the non-vascular fibrinous bands, which are of a slate color; second, the obstruction of the bile ducts interfering with the outflow of bile, and the centres of the atrophied cells becoming pigmented; third, the cells at the periphery of the lobes becoming the seat of fatty degeneration. The new-formed connective-tissue is filled with an abundance of round cells, formerly called nuclei; they are now known as lymphatic corpuscles, and are probably emigrated leucocytes. The smaller branches of the portal vein are shrunken and twisted, and in extreme cases new capillaries are developed, which communicate with both hepatic and portal vessels. Again, a whole lobule may have disappeared and its place be occupied by connective-tissue. The bile-ducts have their commencing rootlets destroyed and their mucous membrane tumefied. In cases of long-standing cirrhosis, the gall-bladder will be found bound to the adjacent parts by adhesions, and its walls are thick and tough; while the spleen is enlarged, softened and congested. The gross appearance of the liver in cirrhosis may vary in different cases, but the anatomical lesions are the same in all.

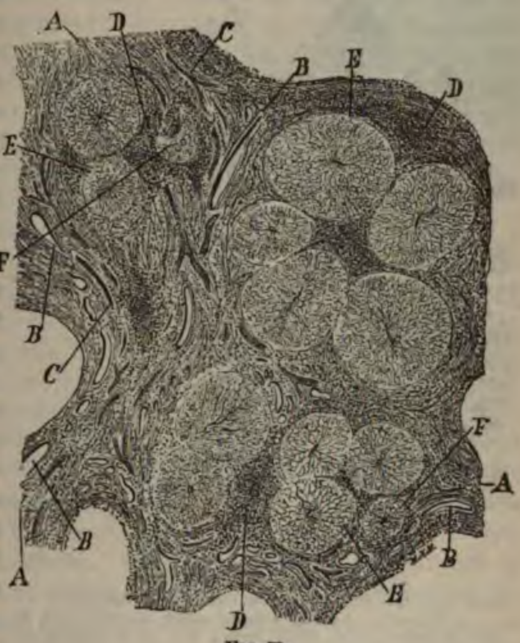

FIG. 72.

Interstitial Hepatitis.

Section of liver in advanced cirrhosis, as shown by low magnifying power.

A, A, A. *Bands of connective-tissue.*
B, B, B. *Branches of portal vein.*
C, C, C. *Hepatic ducts.*
D, D. *Newly formed connective-tissue.*
E, E, E. *Lobules, separated by the advancing cirrhosis.*
F, F. *Lobules nearly obliterated.* × 40.

There is a form of this disease which has been called *hypertrophic cirrhosis*, because the liver is markedly increased in size, sometimes reaching six or seven pounds in weight; but the fibroid change, the yellow staining, the atrophy of liver-cells, and the fatty change at the periphery of the lobule, are the same as in the ordinary variety, the only difference being a marked increase in the size of the liver.

As a result of the compression and obliteration of the branches of the portal and hepatic vein from the new connective-tissue in the advanced stage of cirrhosis, a chronic venous congestion occurs in those viscera which empty their blood into the portal vein. The spleen, stomach and intestines consequently become the seat of chronic venous congestion. The result of this is, that with cirrhosis of the liver we find an enlarged spleen, and a chronic gastric and intestinal catarrh. If the portal obstruction is very great, there will be transudation of serum from the vessels of the peritoneum into the peritoneal cavity, causing *ascites.*

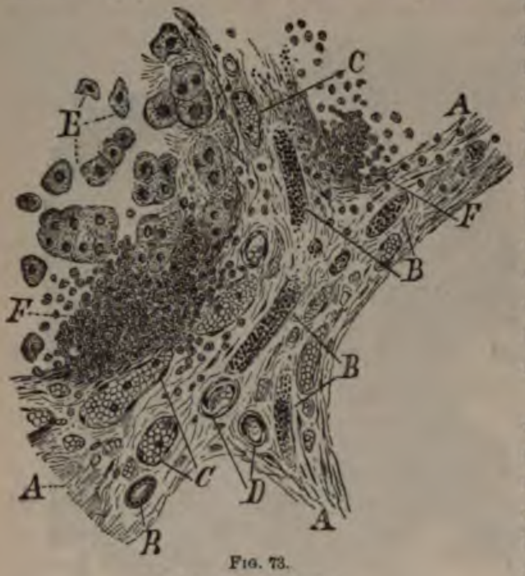

FIG. 73.

Interstitial Hepatitis.

Same tissue as in last illustration, more highly magnified.

A, A, A. Connective-tissue of a portal canal, containing, B, B, Hepatic ducts; C, C, Portal veins; and D, D, Hepatic arteries.

E. Atrophied hepatic cells in periphery of a lobule.

F. Infiltration of round cells—the commencement of the new connective-tissue growth. × 300.

Etiology.—The chief cause of cirrhosis is the intemperate use of alcohol. Alcohol is most rapidly absorbed when the stomach is empty. When it is taken in a concentrated form without food it acts as a direct irritant to the hepatic circulation. If this irritation is long continued interstitial hepatitis is the result. Those who take alcohol before breakfast as well as through the day are almost certain to develop cirrhosis of the liver. Those who partake freely and daily of highly seasoned food, even though they may not use alcohol, are also liable to develop this disease, especially if they reside in hot climates. Syphilis, gout and rheumatism cause it. Gout, especially starting from faulty liver digestion, is liable to develop cirrhosis. Malarial hyperæmia, if long continued, may lead to it. Extension of inflammation from the capsule of Glisson may develop cirrhosis. It may also occur without any assignable cause.

Symptoms.—The early symptoms of interstitial hepatitis are those of hepatic congestion. Following the dull pain and tenderness in the hepatic region, the dyspnœa, apathy, headache, nausea and furred tongue, there is loss of appetite, especially for meats. The individual has a desire for food, but after a few mouthfuls the sense of hunger gives place to loathing; this occurs most frequently at breakfast. Diarrhœa alternates with constipation, and distressing attacks of retching, especially on waking in the morning, are followed by intestinal pain and flatulence. As the dyspeptic symptoms increase, slight jaundice appears, but it is never very marked, for although the bile secretion is diminished, there is no obstruction to its passage into the intestine. Hemorrhoids are early signs of obstructed portal circulation and are a very constant accompaniment of cirrhosis, and, with the gastric symptoms, are the earliest indications of obstruction to the portal circulation. If cirrhosis has been induced by the excessive use of alcohol, a craving for alcohol persists. Attacks of vertigo and occasional slight elevations of temperature with emaciation and cachexia mark the end of its first stage. The dingy hue of the surface, which was early present, disappears, and the skin gradually assumes an earthy pallor, rarely tinged with yellow. The increase in the hepatic symptoms and the severe pain and tenderness over the hepatic region, which are sometimes present, are due to *intercurrent* attacks of perihepatitis.

At the commencement of the *second* stage gastritis is established as a result of the mechanical obstruction to the capillary circulation of the mucous membrane of the stomach, and is marked by acidity, nausea, and often by vomiting after taking food, in consequence of which emaciation, weakness, and depression of spirits occur; venous stigmata may now appear on the cheeks. The obstruction of the vessels of the gastric mucous membrane is often so great that hæmatemesis occurs, and although the patient may experience temporary relief after the first bleeding, the hemorrhage will recur, and may be the direct cause of death. Intestinal hemorrhage occasionally occurs from the same cause. If there is persistent diarrhœa during this stage, it indicates that there is gastro-intestinal catarrh. Tympanitis, as well as intestinal catarrh, usually precedes the occurrence of ascites, which is slow and insidious in its advent, and so masked by the preceding tympanitic distention that it is often difficult to determine the exact time of its occurrence. It will usually be noticed that, before the appearance of the ascites, the abdominal veins, especially of the right side, are distended, sometimes enormously.

Ascites is sometimes absent in advanced stages of cirrhosis. When this happens, any or all of the following conditions may exist to account for its absence: *first*, during the contraction of the new connective-tissue, the branches of the vena portæ sometimes remain patent. *Second*, the hemorrhoidal branches of the inferior mesenteric may inosculate with the internal iliac. *Third*, the veins of the colon and duodenum may anastomose with the left renal vein. *Fourth*, the phrenic vein may communicate with some of the more superficial branches of the vena portæ. *Fifth*, new vessels may be formed in the adhesions which bind the liver to the adjacent parts, and thus relieve the obstructed portal circulation. *Sixth*, the portal and hepatic vessels may anastomose sufficiently within the liver to relieve the portal obstruction. *Seventh*, branches of the vena portæ, which are distributed on the under surface of the diaphragm, and on the inner surface of the abdominal parietes, may anastomose with the internal mammary and epigastric veins, and thus assist in returning the blood to the right auricle. When the internal mammary in its turn becomes engorged, there is a dark bluish mass surrounding the umbilicus, due to distention of the cutaneous veins, and called the "*caput Medusæ*"

When ascites is once developed it progressively increases. By its pressure dyspnœa and often pulmonary œdema are developed, and the gastric derangements are so increased that rapid emaciation quickly follows its accumulation. Œdema, beginning in the feet, gradually extends upward. Fluid drawn from the abdominal cavity is of a pale amber color, highly albuminous, and of a specific gravity varying from 1.010 to 1.020 ; it is not turbid, and contains no inflammatory products. Slight jaundice may appear with the ascites, but if *excessive* is due to pressure on the ducts, either from connective-tissue indurations or from enlarged lymphatics in the transverse fissure, which obstruct the outflow from the bile ducts. The mind is usually clear to the last, but sometimes the patient will pass into a state of complete stupor, which is preceded by delirium and active cerebral symptoms. At first, it seems plausible to ascribe the cerebral symptoms to cholæmia, but I have found the jaundice in inverse proportion to the cerebral symptoms. Cholæmia may occur in cirrhosis, and then, of course, exhibits its peculiar train of symptoms ; but I think the more reasonable view is the one that ascribes the delirium, coma, and other cerebral symptoms which come on late in this disease, to alcohol. The stools in cirrhosis are characteristic. They are clay-colored in the centre ; surrounding this there is a dull pinkish ring, and around this a slaty gray ring tinged with mucus. The urine is scanty and very dark colored ; in one-third of the cases it contains albumen. Bile pigment is present in the urine when jaundice exists. The urine is rich in urates, and a pinkish sediment of lithates is very common.

Physical Signs.—*Inspection,* in the *early stage* of cirrhosis, may show a slight loss of motion over the lower portion of the right side. In a few instances the faint outline of the liver margin is seen below the free border of the ribs.

Palpation.—The surface of the liver is smooth, or finely granular; the edges are round ; on *firm* pressure there is a marked tenderness, and more or less resistance.

Percussion.—The area of hepatic dulness is somewhat increased in the early stage (especially over the right lobe) in a vertical direction, and so, too, is the area of splenic dulness.

Inspection, in the *advanced* stage, shows enlargement of the superficial veins of the abdomen, chiefly on the right side, and the *caput Medusæ* is often visible. There is usually more or less ascites, and the face and surface of the body are clay-colored, often tinged with yellow.

Palpation is best performed when the patient is lying on the left side, and if the surface of the liver can be felt, it will be uneven and hob-nailed, with sharp, firm edges.

Percussion gives a diminished area of hepatic dulness, and the left lobe of the liver may be so small that the line of hepatic dulness will not extend to the left of the median line. Persistent tympanitic percussion is present *above* the line of the fluid, and *flatness* below. The spleen is markedly enlarged, and the splenic dulness extends below the free border of the ribs.

(as squills and digitalis) are more efficient in this than in any other form of dropsy. If the kidneys and renal vessels are compressed by the fluid, diuretics will have little effect. Its removal must not be delayed too long, for the strength of the patient may be so diminished that after the removal of a large quantity of fluid, fatal collapse may occur. When, however, remedial measures fail and dyspnœa becomes troublesome, paracentesis abdominis should always be resorted to. The mechanical removal of the fluid may be effected either by the aspirator or trochar. After its removal, the hob-nailed surface of the liver may cause peritonitis by the constant irritation produced by the respiratory movements. There are few cases where tapping has been frequently performed, in which after death a moderate amount of chronic peritonitis is not found. The ascites will return sooner or later after paracentesis; but when tapping is only required at sufficiently long intervals for the patient to recuperate between the tappings, and the amount of fluid gradually diminishes or becomes stationary, the case will continue for years.

HYPERTROPHIC CIRRHOSIS OF THE LIVER.

Hepatic cirrhosis with hypertrophy of the organ appears in two forms. One is known as simple or biliary cirrhosis; the other as fatty hypertrophic cirrhosis.

Morbid Anatomy.—In biliary cirrhosis the bile ducts and radicles become distended, and pigment is deposited in the hepatic cells and connective-tissue. Degenerative changes affect the parenchymatous elements coincidently with an abundant development of connective-tissue. The liver thus becomes enlarged, hard, and deeply pigmented brown or black. As the degenerative changes cause in many parts complete destruction of the liver cells, the increase of size in the organ depends largely upon the new connective-tissue. Although the organ becomes harder, the fibrous formations do not contract as firmly as in simple cirrhosis, and the organ thus retains its smooth surface.

In fatty hypertrophic cirrhosis, called by the French "*hypertrophic cirrhosis with icterus*," the fibrous formations are equally abundant, but show even a less tendency to extreme degrees of contraction, while the degenerative changes within the parenchyma are distinctly fatty. These fatty products are not absorbed, and tend to still further increase the size of the organ. Such a liver differs from a simple fatty liver in the presence of abundant new connective-tissue between and within the lobules. The new growth originates in the portal spaces, and the portal vessels and bile ducts become surrounded by compact fibrous tissue from which bands radiate along the central and sublobular veins, in such amounts at times as to cause their entire obliteration. The biliary passages are affected secondarily and in less degree, the vascular apparatus being the parts particularly involved. This forms one of the prominent pathological points of difference from biliary cirrhosis.

Prognosis.—The prognosis will be determined by the stage of the cirrhosis. In its early stage its progress may be arrested, but when the stage of contraction is reached, the disease is progressive, and the prognosis is exceedingly unfavorable. A fatal result occurs in all cases. Its course is a chronic one, and though death has occurred in three months from the time the liver began to be diminished in size, I have usually found a year and a half to be its average duration. Complicating diseases influence the prognosis. Hemorrhage from the intestine and from the hemorrhoidal veins may be so great as to exhaust the patient, and render him too feeble to resist the inroads of the disease. This class of patients are especially liable to develop the cirrhotic form of Bright's disease. Delirium tremens, pleurisy, and pneumonia sometimes complicate it. Death may result from exhaustion due to faulty nutrition, from the large fluid accumulation, from repeated and profuse hemorrhages, and from wasting diarrhœa. Intercurrent pulmonary or cardiac disease, peritonitis, or delirium tremens may be the direct cause of death. Those cirrhotic patients live the longest who have large dropsical accumulations, the dropsy disappearing and recurring.

Treatment.—Cirrhosis, in its early stages, should be treated in the same way as active hepatic hyperæmia. In alcohol drinkers, all spirituous liquors must be abstained from, and the patients must be placed on a nutritious, though restricted diet, from which all irritating ingesta are excluded, and alkaline waters should be freely taken. If the hepatic congestion is intense, leeches to the anus, mercurial purges, and nitro-muriatic acid will be found of temporary service. The importance of a restricted diet, and the free use of saline waters in this stage of the disease cannot be overestimated. Cod-liver oil is indicated in this stage. After the stage of contraction is reached, the treatment can only be palliative. The most important thing to be accomplished now, is to improve nutrition, and to relieve urgent and troublesome symptoms. Mineral acids combined with vegetable tonics, such as dilute nitric acid and calumbo assist stomach digestion ; creosote and sulphite of sodium are of service when acid fermentation is a distressing symptom. Mineral waters should be discontinued during this stage. If constipation exists, the bowels may be regulated with rhubarb combined with small doses of ipecacuanha. Cod-liver oil should be constantly taken by this class of patients. Care must be taken not to suddenly check diarrhœa, or hemorrhages, but if they become exhausting opium may be cautiously given.

Ascites and general dropsy are the most troublesome symptoms in this stage of cirrhosis. When it becomes imperative to remove the dropsy, it may be attempted through the skin, kidneys and intestines, or it may be removed by tapping. If the patient is too feeble to employ drastic purges and hydragogue cathartics, diuretics and diaphoretics may be resorted to. The condition of the intestinal tract, as well as the strength of the patient, will determine whether elaterium, or any of the other drastic cathartics can be employed. It must be remembered that they may excite acute gastric and intestinal catarrh. Diuretics (as squills and digitalis) are more efficient in this than in any other form of dropsy. If the kidneys and renal vessels are compressed by the fluid, diuretics will have little effect. Its removal must not be delayed too long, for the strength of the patient may be so diminished that after the removal of a large quantity of fluid, fatal collapse may occur. When, however, remedial measures fail and dyspnœa becomes troublesome, paracentesis abdominis should always be resorted to. The mechanical removal of the fluid may be effected either by the aspirator or trochar. After its removal, the hob-nailed surface of the liver may cause peritonitis by the constant irritation produced by the respiratory movements. There are few cases where tapping has been frequently performed, in which after death a moderate amount of chronic peritonitis is not found. The ascites will return sooner or later after paracentesis; but when tapping is only required at sufficiently long intervals for the patient to recuperate between the tappings, and the amount of fluid gradually diminishes or becomes stationary, the case will continue for years.

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Hepatic cirrhosis with hypertrophy of the organ appears in two forms. One is known as simple or biliary cirrhosis; the other as fatty hypertrophic cirrhosis.

Morbid Anatomy.—In biliary cirrhosis the bile ducts and radicles become distended, and pigment is deposited in the hepatic cells and connective-tissue. Degenerative changes affect the parenchymatous elements coincidently with an abundant development of connective-tissue. The liver thus becomes enlarged, hard, and deeply pigmented brown or black. As the degenerative changes cause in many parts complete destruction of the liver cells, the increase of size in the organ depends largely upon the new connective-tissue. Although the organ becomes harder, the fibrous formations do not contract as firmly as in simple cirrhosis, and the organ thus retains its smooth surface.

In fatty hypertrophic cirrhosis, called by the French "*hypertrophic cirrhosis with icterus*," the fibrous formations are equally abundant, but show even a less tendency to extreme degrees of contraction, while the degenerative changes within the parenchyma are distinctly fatty. These fatty products are not absorbed, and tend to still further increase the size of the organ. Such a liver differs from a simple fatty liver in the presence of abundant new connective-tissue between and within the lobules. The new growth originates in the portal spaces, and the portal vessels and bile ducts become surrounded by compact fibrous tissue from which bands radiate along the central and sublobular veins, in such amounts at times as to cause their entire obliteration. The biliary passages are affected secondarily and in less degree, the vascular apparatus being the parts particularly involved. This forms one of the prominent pathological points of difference from biliary cirrhosis.

The hypertrophy, which is often extensive, affects particularly the edges of the organ, so that it is like a cube. Sabourin dwells particularly upon this. There are no granulations upon the surface, and Glisson's capsule, sometimes thickened from perihepatitis, is smooth and so transparent that through it can be seen opaline-looking new connective-tissue enclosing yellow fatty parenchyma. Upon section the hepatic parenchyma seems made up of fatty nodules, usually circular, smaller or larger than the normal hepatic lobules, and almost completely surrounded by new tissue.

The latter has sometimes induced absorption of the proper glandular elements. With a low power under the microscope there is an appearance like subcutaneous adipose tissue, with cirrhotic bands enclosing collections of fat cells. These groups of fat cells, which are often simply large oil globules, represent the hepatic lobules. The granulo-fatty degeneration peculiar to typhoid jaundice is absent. The kidneys are usually the seat of simple or similar sclerotic changes. The spleen is enlarged, often more than in simple cirrhosis.

Etiology.—Biliary sclerosis is probably dependent in almost every instance upon some form of obstruction in the larger hepatic ducts, from gall stone, cicatricial contractions, pressure from tumors, etc. The causes of fatty hypertrophic cirrhosis are obscure. It occurs in alcoholic subjects, but is by no means confined to them. It seems possible that it may follow biliary cirrhosis, or even start as a simple cirrhosis.

Symptoms.—The clinical distinctions between biliary and fatty hypertrophic cirrhosis are not clear. In the earlier stages the symptoms are all obscure and often overlooked. Fullness and oppression in the hypochondrium usually are the first to attract attention. Jaundice appears early and is persistent, being more marked, as a rule, in the biliary form. The enlargement of the liver may perhaps be recognized if attention is called to it. When the second stage sets in, there will be abdominal pain, nausea, vomiting, anorexia, attacks of vertigo, nocturnal delirium or hallucinations, hyperæsthesia of the limbs, and then fever. A diagnosis is possible in most cases with these symptoms. They are followed by œdema of the face and limbs, profuse sweats and often signs of subacute peritonitis. With the increasing persistent jaundice there may be tendency to hemorrhages. The symptoms are very apt to increase and then recede. Usually little flesh is lost, and patients may even grow fat. When the abdominal fat is not too abundant, physical examination shows a large, smooth, rounded liver, seldom very tender on pressure. Splenic enlargement can also be recognized. Ascites is seldom present; when developed, it is slight. The whole course of the disease is from one to two years, and the second stage may last four or five weeks, or be prolonged several months by deceptive intervals of apparent recovery.

Differential Diagnosis.—This must be made from other hepatic diseases causing jaundice, by the presence of signs of extensive portal obstruction, associated with enlargement of the liver. An alcoholic history, or the presence of tubercular complications, are important diagnostic points, when associated with enlarged liver, in favor of hypertrophic cirrhosis.

Prognosis.—This is invariably bad.

Treatment.—The treatment is purely palliative and expectant. None of the many theories advanced as to its cause have been accepted. Those measures employed in simple cirrhosis may relieve the symptoms, but are even more uncertain in their action in this condition.

CIRCUMSCRIBED SUPPURATIVE HEPATITIS.

Abscess of the liver is an acute circumscribed hepatitis which results in irregular areas of suppuration, the liver-tissue surrounding the points of suppuration remaining normal.

Morbid Anatomy.—In a certain proportion of cases circumscribed hepatitis has its origin in an infarction. The emboli which produce these infarctions are, in most instances, stamped with pyæmic infection or are necrotic; they may vary in number from two or three to a dozen. Immediately around the inflamed spots the liver substance is normal or in a state of intense congestion, and corresponding to them on the surface of the liver are found brownish-red elevated patches from an inch to an inch and a half in diameter, and of firm consistence. Their most frequent seat is the posterior portion of the right lobe. They may be single or multiple. Soon after the development of the infarction purulent inflammation is established. In the centre of the mass the liver cells undergo albuminoid infiltration, become larger, degenerate, and pus is formed. As the process advances small cavities containing pus are developed. These may enlarge into one large abscess or remain separate. If abscesses result from other causes than pyæmia, the process does *not* follow the lobular course, but begins by small exudations of lymph and pus, which soon coalesce and become surrounded by a membraneous wall. The cavity of the abscess varies in size from that of a hazel-nut to one capable of containing two or three quarts. Their contents are usually pale yellow pus; but when the suppurating process has broken through the wall of some vessel, then the contents are rust colored and have a granular appearance. If the process involves the bile-ducts the pus has a greenish or ochre-color.

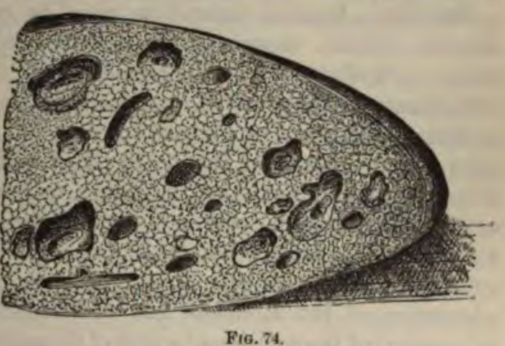

FIG. 74.
Circumscribed Suppurative Hepatitis.

Sketch showing the cut surface of a portion of the left lobe of the liver, the seat of multiple abscesses. The open mouths of the divided hepatic veins are also shown.

A variety of changes take place in these purulent collections; as they grow older, their walls may become smooth, and the encysted pus gradually become absorbed, or undergo cheesy or calcareous degeneration. If absorption takes place, a cicatrix deeply indented on the exterior of the liver marks the place where the abscess approached the surface, and within its substance cicatricial tissue indicates its exact seat. In other cases, no lining membrane is produced around the purulent collection. As the inflammation extends, a necrotic process is established which extends to the surface of the liver, and the abscess opens externally. This process may pierce the peritoneum, causing a fatal peritonitis; but this is of rare occurrence, for adhesions are formed which bind the diaphragm and adjacent organs to the liver; or the abscesses may open into the pleural cavity, the right lung, or the stomach. In rare instances the two large venous trunks, the vena portæ and inferior cava may be pierced. Frequently the abscess ruptures externally, through the adhesions formed between the surface of the liver and the abdominal walls. The intestines, the gall-bladder, or the pericardium may be perforated by the abscess. Again, the pus may burrow in the cellular tissue, and discharge itself at some point at the lower part of the trunk. At the autopsy the liver may be found irregularly enlarged, often attaining an immense size. The capsule is opaque and thickened, and on its surface are elevated flat spots varying in color from dark red to yellow. Adhesions generally exist between it and the adjacent parts, and a layer of lymph frequently envelops the entire organ. The whole liver is hyperæmic. Evidences of perforation, in any of the directions which have been mentioned, may be present.

On section, a dark fluid oozes from the congested surface, and the interior of the abscess may exhibit any of the changes above described. Near an abscess the coats of the veins, especially the portal, are thickened, and their interior is often filled with a shaggy, fibrinous deposit.

Etiology.—Pyæmic infarction must be regarded as a frequent cause of hepatic abscess, especially when associated with injuries to the cranial bones. Phlebitis, with the formation of thrombi leading to suppurative inflammation, the result of operations on the intestines (as for prolapsus ani, hemorrhoids, and strangulated hernia) and any intestinal traumatism, may give rise to abscess of the liver. Hot climates and miasmatic influences favor, if they do not cause, suppurative hepatitis. In hot climates dysentery, particularly the epidemic form, is frequently accompanied or followed by hepatic abscess, so that dysentery has come to be regarded as one of the causes of abscess of the liver. Hepatic abscess sometimes accompanies ulceration of the stomach and of the intestine, especially of the colon. Ulceration of the gall-bladder and of the appendix vermiformis, perityphlitis, pyelitis, ulcerative endocarditis, and cancer of the stomach or of the parts near the liver are often associated with hepatic abscess. Worms, calculi, or other obstructions of the ductus communis, causing inflammation of the hepatic ducts, sometimes lead to ulceration, and this ulcerative process is often followed by abscess. The prevailing tendency of modern pathology is to ascribe hepatic abscess to an infective embolus, from a preceding phlebitis, and the attempt has been made to trace back all the causes above named to such a primary cause, though, in many, direct proof is impossible. Abscess of the liver may also be the result of inflammation of the bile-ducts and of the veins of the liver; and, finally, it may be developed without any recognizable cause.

Symptoms.—An abscess of the liver of considerable size may exist without there being local or constitutional symptoms to point to its existence. A patient may have fever, gradually become emaciated, and finally die from exhaustion, without a single objective symptom of abscess, and yet a post-mortem examination will reveal a large central hepatic abscess. Again, symptoms of intermittent fever, associated with gastric and intestinal catarrh, may be all that, with the greatest care, can be elicited, when, in fact, a large abscess is developing in the liver. When abscess is associated with dysentery the difficulty is often increased; for chills and rigors, enlargement of the liver, and pain may all be attendants of dysentery without abscess. Again, in pyæmia, when metastatic abscesses in the liver are especially liable to develop, the recurring chills, the sweats, the pyrexia, and the jaundice, are all part of the history of the pyæmia, so that in many cases we are compelled to rely almost exclusively on the physical signs for a diagnosis of abscess of the liver. Such cases of hepatic abscess are frequently overlooked.

Usually the development of hepatic abscess is indicated by well-defined symptoms. A slight feeling of chilliness, sometimes a distinct chill, is followed by dull pain and weight in the right hypochondrium, the pain often radiating to the tip of the right shoulder; the chilly sensations recur, and resemble those of a slight attack of ague. The pain increases, and is aggravated by position and pressure. The tongue is brown and furred, there is loss of appetite, slight nausea, and often vomiting, which is bilious in character. The bowels are at one time constipated, at another there is a bilious diarrhœa.

The respirations are hurried and shortened, either because of slight localized pleurisy, which so often accompanies hepatic abscess, or because a long inspiration increases the pressure on the liver, and thus causes pain. With the dyspnœa there is a short dry cough resembling that of pleurisy; the skin rarely changes color. With the formation of the abscess there is a distinct exacerbation of symptoms: hectic, rigors, and recurring night sweats occur; the gastric symptoms become urgent, and there is persistent and profuse vomiting. The pain becomes sharp, lancinating and localized, and indicates the direction of the future perforation. The temperature rises to 103° or 104° F., sometimes reaching 106° F. The pulse is accelerated, generally keeping pace with the temperature. Exhaustion and emaciation are rapidly developed, and as the disease advances, typhoid symptoms may supervene. If the situation of the abscess is such that portal obstruction results, hemorrhoids, ascites and œdema of the extremities occur, though peritonitis may be suspected in those cases where there is ascites. As the abscess advances toward the surface of the liver, attacks of perihepatitis are apt to cause severe exacerbations of pain, and the hepatic tenderness becomes excessive. Toward the end delirium, jactitation, somnolence, and coma may develop. *The urine* is scanty, high-colored, and contains an abnormal amount of urates, often causing a considerable pinkish deposit.

Hepatic abscesses may be discharged (as has been stated) in a variety of ways. When an external opening is to occur, the skin becomes tense, red, shining, and œdematous at some point over the hepatic region; fluctuation can be detected, and becomes more and more marked as the abscess advances toward the surface. In some cases the heart impulse is transmitted to the abdominal walls by the intervening tumor. When the cavity of the peritoneum is perforated, signs of local or general peritonitis are developed, marked by rapidly developed tympanitis, intense and sudden collapse. When an opening occurs into the stomach, severe gastric symptoms are developed, accompanied by purulent vomiting and purulent stools. A sudden diminution in the size of the hepatic tumor, the subsidence of the pain and of the urgent symptoms, indicate that the intestine or biliary passage has suffered perforation. Symptoms of localized pneumonic inflammation in the right lower lobe precede the opening of an hepatic abscess into a bronchus. The expectoration suddenly becomes purulent and mixed with blood, serum, and shreds of disintegrated lung-tissue; the breath becomes offensive, there is cessation of pain, and subsidence of the hepatic enlargement. Recovery in such cases is rare unless the abscess is a small one, the patient usually dying of exhaustion from long-continued suppuration. When the pleural cavity communicates with the abscess, the symptoms of pleuritic effusion and empyema are well defined, and when, in this case, an external opening is to occur, the evidences of it are the same as those of empyema. Almost immediate death follows perforation of the pericardial sac by an hepatic abscess. Absorption of the fluid contents of an hepatic abscess, and the development of cicatricial tissue, followed by gradual diminution in the size of the liver, are of rare occurrence.

Physical Signs.—*Inspection.* If the abscess is large, inspection will show a bulging of the right hypochondriac region, reaching nearly to the umbilicus. The respiratory movements on the right side are restricted, and the respirations are accelerated. If the abscess is to open externally, there is a flattened, defined bulging near the free border of the ribs, between the intercostal spaces.

Palpation.—The liver is enlarged and has an uneven feel, especially when the abscesses are multiple and superficial. The pain, localized in the case of a single abscess, is increased and diffused in multiple abscesses by pressure. Fluctuation is a valuable sign, but cannot always be detected. When it can be, a ring of abnormal hardness surrounds the spot. Palpation should be made from before backward; if a single large abscess exists, its outline may be well defined.

Percussion.—The area of hepatic dulness is always more or less increased. If the abscesses are multiple, it may be increased in all directions; but if there is only one large abscess, the area of dulness will correspond to the direction of enlargement, which may be upward or downward; by its direction we are able to determine the probable mode of the termination of the abscess.

Differential Diagnosis.—The readiness with which the diagnosis of abscess of the liver may be made will depend upon its size and situation; small abscesses can only be suspected. Abscess of the liver may be mistaken for *hydatids* of the liver, *cancer*, localized *pleurisy*, *intercostal neuralgia*, *abscess* of the *abdominal walls*, *enlarged gall-bladder*, *perihepatitis*, *suppurative pylephlebitis*, and *active hyperæmia* of the *liver*.

Hydatids occur most frequently in those living in northern climates, and abscesses most in those who live, or have lived, in hot climates. Hydatid tumors run a chronic course, and are slow in growth; while abscess is usually a rapid and acute disease. Accompanying hydatids there is no pain, rigors, hectic, or sweats; but these are important symptoms in abscess. Gastric disturbances and a rapidly developing cachexia are prominent in abscess, and absent in hydatids. In some cases of hydatid tumor the hydatid "thrill" or fremitus can be detected; it is never present in abscess. With the exploring trochar the liquid in the one will be found to be pus, in the other a clear saline fluid containing hooklets of the echinococci.

Cancer of the liver is generally associated with cancer of the stomach, breast, or some other organ, primary cancer of the liver being very rare. In cancer, the hepatic enlargement is slower than in abscess, and there is usually a more or less marked cancerous cachexia. Suppurative fever, chills, hectic, and sweating are present in abscess, and absent in cancer. The temperature in cancer is normal or sub-normal, and jaundice, if present, is persistent. Ascites is common in cancer, and rarely present in abscess. In cancer, palpation discovers scattered nodular masses, which rarely fluctuate; while in abscess a large fluctuating tumor can usually be made out. The exploring needle withdraws pus from an abscess, while blood follows the puncture of a cancer nodule.

Pleurisy on the *right side* can usually be readily distinguished from abscess of the liver by the physical signs alone. The grazing friction sound accompanies loss of vocal fremitus; the dulness on percussion, the feeble respiratory murmur, and the crepitant friction sound decide the question.

Intercostal neuralgia occurs most frequently in women with a neuralgic history. The pain is located in the region of the sixth, seventh, and eighth intercostal spaces, and the three points of tenderness are almost diagnostic. When the pain of abscess becomes as excruciating as that of neuralgia, inspection, palpation, and percussion will all reveal well-marked enlargement of the liver. Gastric disturbances, chills and profuse sweats are prominent signs of abscess, and are absent in intercostal neuralgia.

In *abscess* of the *abdominal* walls, there is no history of pyæmia, dysentery, or internal ulceration, which so often precede an hepatic abscess. In hepatic abscess the line of dulness is well marked, and corresponds in outline to the hepatic area; while in abscess of the abdominal walls the line of dulness is ill defined, and does not follow the hepatic outline. A tense, shining, œdematous skin, and superficial tenderness and hardness appear early in abscess of the abdominal walls. The signs of pus formation are early in abscess of the abdominal wall, and very late in abscess of the liver, if they appear at all. The respiratory movements cause an upward and downward motion in the tumor of an hepatic abscess; while an abscess of the abdominal wall will remain stationary during the respiratory acts.

An *enlarged gall-bladder* will usually be accompanied by a history of biliary colic. The presence of a pear-shaped, movable, fluctuating tumor, occupying the normal position of the gall-bladder, a history of jaundice, and the absence of constitutional symptoms indicate enlarged gall-bladder; while the tumor in abscess of the liver is broader, less movable, less globular in shape, and is attended by chills and sweats.

Prognosis.—The majority of abscesses of the liver terminate fatally. Pyæmic abscesses are generally multiple; their average duration is three months; I have known death to occur within three weeks after the commencement, and I have known them to be prolonged over a period of two years. In abscess from other causes than pyæmia, the prognosis is favorable whenever there are no indications of an opening into the pericardium, peritoneum, or pleural cavity. When an hepatic abscess complicates a severe attack of dysentery, the prognosis is unfavorable. Their duration is shorter, and the prognosis is better when they open externally; their next most favorable termination is when they open into a bronchus, or into the intestinal canal. Pyæmia and dysentery often cause death when the accompanying abscess is too recent to have induced it. Exhaustion from suppuration may cause death, especially when accompanied by intestinal catarrh. Peritonitis, pericarditis, pneumonia, and empyema sometimes cause the fatal result.

Treatment.—When multiple abscesses occur, antiseptics have been proposed, but there is no evidence that they arrest the progress, or diminish the severity of the suppurative process. When suppurative hepatitis can be recognized early, it should be treated according to the rules which have been given for the management of acute hepatic hyperæmia. Local blood-letting by leeches may be employed when the symptoms are localized and well defined; and mercurial purges may be given at the onset, in combination with large doses of quinine, but they should be discontinued when suppuration is established. We rarely have an opportunity to carry out the preventive treatment, for the abscess is formed before the patient seeks medical advice. When pus has formed, and the locality of the abscess can be determined, aspiration should be performed. If the withdrawal of the pus is followed by decided signs of improvement, the aspiration may be repeated at intervals indicated by the amount and effects of the purulent accumulation. Few cases, however, will be permanently benefited by aspiration. I question very much if those cases reported cured by one or two aspirations were true hepatic abscesses. The dysentery and the gastro-intestinal catarrh, which are so often attendants of hepatic abscess, are best treated with large doses of ipecacuanha; a fuller description of this method of treatment will be found under the head of dysentery.

The question of operative interference is one which it is often difficult to decide. Strong opinions have been given for and against it. On the one hand, it is claimed that if a free opening is not made, death may result from exhaustion produced by large purulent accumulations, or the abscess may open into the peritoneal cavity, pericardial sac, or pleura, and thus cause death. The process is a progressive one, and each day more and more of hepatic tissue will be involved, and thus diminish the chances of recovery. On the other hand, those who oppose opening the abscess say that peritonitis and the entrance of air may result from it, that the ribs are more liable to become eroded, and the surrounding tissue to become gangrenous, when an opening is made. Some regard it as highly dangerous to pass an instrument into the liver, claiming that it may excite a suppurative process in healthy liver-tissue. All these objections are removed if antiseptic methods are employed. If no adhesions have formed between the liver and the abdominal walls, they should be established by caustics, and then the sac may be opened; if it is very large, all of the pus should not be allowed to escape at once. It is always safest to open the sac by means of caustics, using the knife to divide the superficial tissues. The abscess should be opened as soon as possible. When hepatic abscesses open into the bronchi, colon, or gall-bladder, absolute rest must be insisted upon. In all cases, during convalescence, absolute rest and a careful regimen must be maintained for months. The diet throughout the whole course of the disease should be the most nutritious, and stimulants should be freely given. The importance of sustaining the patient in every possible way is apparent.

DIFFUSE PARENCHYMATOUS HEPATITIS.

This disease, also called *acute yellow atrophy* and *malignant jaundice*, has been regarded as a "passive degeneration," the metamorphosis being more rapid than in any other gland structure in the body. Some think it due to bilious liquefication or *polycholia*, and that it is a general disease—like typhoid fever or cerebro-spinal meningitis—with a local lesion. The more recent views are that acute yellow atrophy is a diffuse inflammation of the whole hepatic structure, where the inflammatory changes are so rapid as to lead to disintegration and complete destruction of the liver cells and subsequent atrophy. Whether it is an exudative process, or one in which there first occurs albuminoid infiltration of the hepatic cells, and then molecular change, is still a disputed question, but the more reasonable view is that it comes from albuminoid infiltration, irregular cloudy swelling of the cells, and subsequent softening of the hepatic tissue.

Morbid Anatomy.—It is seldom that one sees a liver that is the seat of diffuse hepatitis until after the process is completed, but the few that have been studied present the evidences of having been the seat of an intense congestive and exudative process. The liver lobules have a dark gray muddy ring at the periphery, due to granular degeneration or albuminoid swelling of the peripheral cells, while the liver structure immediately surrounding the central vein is normal. The latter is, however, soon involved, and in place of liver cells there are fat and pigment granules, with traces of leucin and tyrosin. All outline of lobular structure disappears, the capillaries are intensely engorged, and the bile-ducts become more or less completely closed, owing to the compression which they suffer from peripheral exudation. Thus the bile formed between the central vein and the exterior of the lobule has no mode of escape, except through the central vein. The liver is diminished in size, sometimes to two-thirds of its normal size; in the early stage the organ is supposed to be very slightly enlarged. The diminution is most marked in the right lobe. It is so soft that it folds upon itself, and takes any shape and position from the pressure of the adjacent organs. At the *post-mortem*, the body will be emaciated, the skin very much discolored, and ecchymotic spots will often be found scattered over the surface.

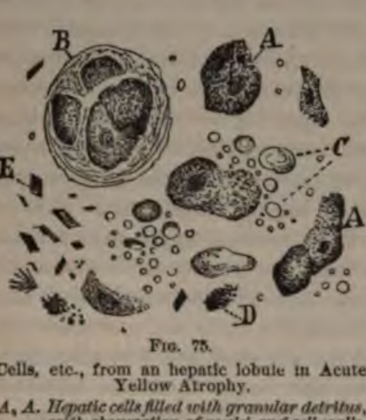

FIG. 75.

Cells, etc., from an hepatic lobule in Acute Yellow Atrophy.

A, A. Hepatic cells filled with granular detritus, with obscuration of nuclei and cell walls.
B. A group of atrophied cells.
C. Cells with fatty infiltration.
D, E. Pigment granules, with blood and tyrosin crystals. × 300.

The capsule is loose, freely movable, very much wrinkled, and opaque or yellowish in appearance. The parenchyma is soft, flabby, and brittle, and varies in color from a bright yellow to a yellow-red.

On section, when the disease is far advanced, the color of the cut surface is of a rhubarb red, the outlines of the lobules are lost, and only a detritus of granular matter is left. The blood is darker and thicker than normal and coagulates imperfectly. It may contain leucin and traces of urea. If the organ be set aside for a while it becomes covered with crystals of leucin and tyrosin.

The *heart* is jaundiced, fatty and pultaceous.

The *spleen* is enlarged and softened, and leucin is found in it. The gall-bladder is empty, or contains a small amount of pale bile or mucus.

The *kidneys* are slightly enlarged, and in most cases are in a state of acute fatty degeneration. Hemorrhages from the surface of the mucous membrane of the *stomach* and *intestines* are common. Occasionally there is softening of the central portion of the cerebral substance, and staining of the meninges. The *serous cavities* contain fluid, often bloody, and in rare instances nearly every organ in the body is blood stained and has leucin and tyrosin in its tissues.

Etiology.—Acute yellow atrophy is a rare form of disease. Its causes are regarded by some as constitutional, by others as due to a peculiar miasm. Two-thirds of the cases occur in pregnant females, between the ages of twenty and thirty. The supposed *predisposing* causes are sex, pregnancy, chronic alcoholism, syphilis, malaria, sexual excess, and a prolonged course of mercurials. But with our present knowledge, it is difficult to say whether these are really predisposing causes, or that the acute yellow atrophy is an intercurrent accident. Among the exciting causes may be named mental emotion, great grief, or fear. It is doubtful if obstruction of the bile-ducts alone can excite acute yellow atrophy; some are inclined to regard malaria as an *exciting* cause, rather than a predisposing one. While its etiology is still so obscure, the history of its development leads to the conclusion that a malarial poisoning is present in a large proportion of cases.

Symptoms.—The symptoms of the early stage of acute yellow atrophy usually pass unnoticed, for they are not in themselves distinctive. When the disease is slow in its advent, loss of appetite, occasional vomiting, a furred tongue, slight headache, and a sense of fulness in the right hypochondrium may be the only symptoms for the first week. Jaundice may precede it for a week or two. In cases where its advent is sudden, it will be ushered in by constant vomiting and great prostration. In either case during its early stage the temperature will be raised only a degree or two, and the pulse but slightly accelerated. A condition of despondency is often present, there are wandering pains simulating rheumatism, and a sense of great depression. Delirium and convulsions may be the only ushering-in symptoms. After from three to five days, the characteristic symptoms of the disease are developed; of these jaundice is the earliest and most constant, it is progressive and never very intense, first affecting the upper half of the body. The rise in temperature and increased pulse-rate which marked its premonitory stage disappear, and now even a retarded pulse and sub-normal temperature may exist. In a few instances, after the first twenty-four hours, the temperature ranges from 100° to 101° F. during the whole course of the disease. The vomited material consists of mucus tinged with bile; later it assumes the nature of black vomit, similar to that in yellow fever, the color being due to gastric capillary hemorrhage. There is intense pain in the epigastric and right hypochondriac regions, which is increased by firm pressure over the liver. In the stage of coma, the hepatic tenderness is so great that pressing the liver up against the diaphragm may rouse the patient. At first the cerebral symptoms are those of mental depression and slight headache, which rapidly increases in severity; this, later, gives place to wild delirium, jactitation, and convulsions. Twitchings of the voluntary muscles of the head and neck (trismus) mark the convulsive stage of the disease. These spasms usually follow the vomiting, but in cases where the disease runs a rapid course, typhoid symptoms make their appearance, sordes collect on the teeth, while low muttering delirium, subsultus, muscular tremors, and partial stupor precede the convulsions. The convulsions are epileptiform, and are sometimes ushered in by a peculiar shrill cry.

During the period of nervous excitement, the pulse undergoes remarkable changes. It may rapidly rise to 120, 130, or even 140 beats in a minute, falling in moments of calm to 80 or 90, the temperature remaining unchanged. The breathing during the convulsions is interrupted or stertorous, and a peculiar groaning noise is heard with each inspiration; the expirations are prolonged and puffing. Whether the convulsions have been preceded by typhoid symptoms or not, the patient gradually becomes more and more tranquil, passes into stupor, and finally into deep coma, from which he cannot be aroused. The discharges from the bowels and bladder are either passed involuntarily or retained. The pupils are normal or slightly dilated, and respond to light slowly. The breathing becomes sighing, the pulse reaches 140 to 150, and grows shorter and shorter until death occurs. The skin during the progress of the disease has become more or less deeply jaundiced, ecchymotic and petechial spots sometimes appear on the surface, and there may be hemorrhages from the stomach, nose, intestine, uterus, and kidneys. In pregnant females abortion is likely to occur before death. The *fæces* are firm, clay-colored, and often blood-stained. The *urine* is acid and dark in color, is not quite up to the normal amount, and often contains albumen and blood; urea and uric acid have *totally* disappeared, the sulphates and phosphates are diminished in quantity, and leucin and tyrosin are found in their place. The duration of the disease varies from one to three weeks.

Physical Signs.—*Palpation* elicits extreme tenderness over the epigastrium and right hypochondrium.

Percussion.—The area of hepatic dulness rapidly diminishes from day to day, and as the liver decreases in size it is displaced backward, so that there is no well-defined area of hepatic dulness in front. As the liver diminishes in size the spleen enlarges.

Differential Diagnosis.—Diffuse parenchymatous hepatitis may be mistaken for *yellow fever*, *pyæmia*, *typhoid fever*, and the *bilious remittent* variety of pernicious fever.

In acute yellow atrophy, the liver is diminishing in size from day to day, while in *yellow fever* it is steadily increasing. The spleen is increased in size in acute yellow atrophy, and is unchanged in yellow fever. The urine in yellow atrophy is acid throughout, and contains leucin and tyrosin; while as soon as jaundice appears in yellow fever the urine becomes alkaline. Yellow fever is ushered in by a distinct chill, while yellow atrophy of the liver rarely begins with a chill. The pulse, in severe forms of yellow fever, is gaseous in character and is rarely over 110, while in acute atrophy the pulse may reach 140 or 150 per minute. The stools are dark and fluid in yellow fever, and firm and clay-colored in acute atrophy.

Pyæmia is ushered in by distinct chills. The chills in pyæmia are followed by irregular rigors and exhausting sweats, which do not occur in acute atrophy. In pyæmia there is diarrhœa, and in acute yellow atrophy the stools are firm and clay-colored. In pyæmia there is a peculiar sweet sickish breath, which is absent in acute atrophy. Evidences of multiple abscesses, especially in the lungs, soon follow the sweats of pyæmia; these do not occur in acute atrophy. The presence of leucin and tyrosin and the absence of urea, with the other urinary symptoms of acute atrophy, are in marked contrast with the normal urine of pyæmia. Physical (hepatic) signs are negative in pyæmia, while a daily diminishing area of hepatic dulness is usually present in acute yellow atrophy.

Typhoid fever has nearly the same premonitory symptoms as acute yellow atrophy, but the steady rise in temperature with the typical morning and evening exacerbations and remissions during the first week, are in marked contrast with the continual low temperature of acute atrophy. The delirium is wandering in typhoid, and wild in acute yellow atrophy. The characteristic "rose rash" appears about the seventh day of typhoid fever. Diarrhœa is the rule in typhoid fever, while constipation and clay-colored fæces are the rule in acute atrophy. In typhoid, the urine is simply diminished in amount, and the urea is increased, while in acute yellow atrophy the urea is greatly diminished in quantity, and often completely absent, and the other (mentioned) urinary changes are present. In typhoid fever the liver is slightly enlarged, while in acute yellow atrophy it is markedly diminished in size.

The "*bilious remittent*" form of pernicious fever very closely resembles in its symptoms acute atrophy of the liver. The severe sudden chill, rapid rise in temperature to 105° or 107°, the sweating, and the remission in pernicious fever are all, however, absent in acute atrophy. Free pigment exists in the blood in bilious fever, and is absent in atrophy. Jaundice is a late symptom of pernicious fever, but occurs early in acute atrophy. The liver is markedly enlarged in pernicious fever, and as markedly diminished in size in acute yellow atrophy. Poisoning from phosphorus can only be diagnosticated from acute yellow atrophy when we know the drug has been taken.

Prognosis.—This is exceedingly unfavorable, and those cases where a cure has been reported are in the doubtful list. The average duration is one week, the extreme limits being twelve hours and four weeks. Cholæmia and uræmia, by inducing the cerebral symptoms which have been referred to, may be the direct cause of death. Peritonitis and hemorrhages from the stomach and bowels are also frequent causes of death.

Treatment.—All plans of treatment have thus far failed either to arrest the progress, or to diminish the fatal tendency of this disease. It has been preferred, in the early stages, to administer drastic purges, and apply leeches over the region of the liver and about the anus, and in the robust and plethoric to practice venesection; there is, however, no evidence that these measures have any controlling influence over the disease. Pregnant females should be placed in pleasant apartments, with cheerful surroundings. When the pain over the liver is intense, leeches and hot fomentations over the hepatic region, with morphine hypodermically, will afford relief. When the cerebral symptoms develop, chloric ether in drachm doses every hour will often quiet the wildest delirium. Hemorrhages from the mucous surfaces can usually be checked by astringents and cold. Bismuth or strychnia will sometimes relieve the vomiting. Bi-carbonate of soda in ten-grain doses every hour has been given with apparent benefit.

PERIHEPATITIS.

Perihepatitis is an inflammation of the capsule of the liver. It is important to remember that the liver has two envelopes, the outer, the *serous* covering which is part of the peritoneum, and an inner, its true *fibrous* covering, the capsule of Glisson.

Morbid Anatomy.—A liver which has been the seat of perihepatitis is diminished in size, except when it is complicated by those diseases of the organ which give rise to enlargement. The capsule is thickened, the thickening varying from a few lines to half an inch; it is more or less firmly adherent to the colon, stomach, diaphragm, and abdominal walls. In syphilitic perihepatitis, Glisson's capsule is hard and leathery and has a granular appearance. Sometimes the capsule is so thickened and contracted in the transverse fissure of the liver as to obstruct the portal vein and hepatic duct. The liver substance usually remains normal, being only slightly compressed on the surface of the organ, corresponding to the furrows between the larger lobules. In "perihepatitis syphilitica" prolongations of new connective-tissue will penetrate the parenchyma, and the outlines of the lobules will be indistinct. This condition is called induration. Slight atrophy of the parenchyma may occur at points corresponding to the circumscribed capsular thickening. The coats of the hepatic veins may be thickened and the bile-ducts dilated. The gall-bladder is sometimes displaced by the contraction of the new tissue, and the ductus communis may be partially occluded by fibrous bands. Perihepatitis is usually accompanied by pleurisy in the lower part of the right pleural cavity.

Etiology.—Exposure to cold, when the liver is in a state of active hyperæmia, is the most frequent cause of perihepatitis. Blows over the hepatic region often excite it, and it may come from an extension of inflammation from the peritoneum or from the right pleura. In all inflammatory forms of hepatic disease and during the development of new growths, perihepatitis is of frequent occurrence. Syphilis is a very common cause.

Symptoms.—It is often ushered in by a chill, followed by a slight rise in temperature and a corresponding increase in the pulse rate. The pulse is tense and wiry in character; pain in the hepatic region is its most constant symptom, and is increased by pressure, by a full inspiration, by coughing, and by lying on the right side. Jaundice is rare, but when the new tissue compresses the common duct it may be developed. A dry, hacking cough is rarely absent. New tissue developments in the transverse fissure may cause sufficient obstruction to the portal vein to produce ascites. From obstructions of the common duct, under similar circumstances, gall-stones may form and be found in the fæces.

Physical Signs.—On *palpation* the liver will be found intensely tender, slight pressure causing severe pain. It may be diminished in size, its edges lobulated, rounded, smooth and harsh.

Percussion.—The area of hepatic dulness is somewhat smaller than normal.

Auscultation.—In the early stage there is sometimes heard over the liver a rubbing sound, like the friction sound in pleurisy.

Differential Diagnosis.—Perihepatitis may be confounded with *intercostal neuralgia* of the right side, with *pleurisy,* and with *abscess* of the *liver.*

In *intercostal neuralgia* there is usually a neuralgic history and three diagnostic points of tenderness:—*first,* at the exit of the nerve from the spinal canal; *second,* midway between the sternum and the spine; *third,* just at the edge of the sternum. The pain is usually confined to the sixth, seventh and eighth intercostal spaces. In perihepatitis there is generally *equal* tenderness over the whole hepatic region, and pressure up under the ribs increases the pain. Elevation of temperature, increase in the pulse-rate, and the history of a chill are all absent in intercostal neuralgia.

In *pleurisy,* the pain is located under the right nipple. The pain is lower down in perihepatitis, and pressure up under the ribs will cause a marked increase in its severity. The dyspnœa is more urgent in pleurisy and the cough has a teasing, hacking character. With the advent of plastic exudation in pleurisy, there is diminished vocal fremitus, dulness on percussion, feeble respiratory murmur, and a "sticky" crepitating friction-sound.

Perihepatitis often accompanies *abscess of the liver,* and then the differential diagnosis is difficult. In abscess there are hectic, rigors, and recurring sweats; while in perihepatitis there is but one chill, and that at the commencement. The temperature in abscess is 103° and 105°, while it is lower, rarely above 101° F., in perihepatitis. Urgent gastric symptoms, profuse and persistent bilious vomiting, are marked in abscess of the liver, and absent in perihepatitis. In abscess there is a rapidly developing cachexia, which does not exist in perihepatitis. In abscess, distinct fluctuation on palpation is often present, while it never occurs in perihepatitis. Percussion in abscess shows an area of hepatic dulness either uniformly increased, or increased in one direction, while the area of hepatic dulness is never increased in perihepatitis.

Prognosis.—The prognosis in perihepatitis is good; it is influenced, however, by the disease which it accompanies. The chief danger is that repeated attacks will lead to "induration" or compression of the portal vein, and subsequent atrophy of the liver. In the latter case, all the symptoms of cirrhosis will follow. When obstruction to the ductus communis is sufficient to cause jaundice, the prognosis is unfavorable.

Treatment.—Rest in the recumbent posture is essential to the successful treatment of this disease. The severe pain which usually attends it can be relieved by hypodermic injections of morphia and the application of leeches over the hepatic region. Warm anodyne poultices should be applied after the leeches. In those cases where there is active hepatic hyperæmia, a mercurial or saline purge is indicated, unless general peritonitis exist. In all cases the diet should be non-stimulating and nutritious, and an individual who has once had perihepatitis should abstain from all forms of alcoholic stimulants.

PYLEPHLEBITIS.

Pylephlebitis is an inflammation of the portal vein, accompanied by coagulation of its contents. Under this term are now included all cases of "portal thrombosis," whether the thrombosis is preceded, followed, or unattended by an inflammatory process. It is of two varieties, *adhesive* and *suppurative*. In *adhesive* pylephlebitis there is more or less extensive obliteration of the veins; in *suppurative*, the thrombus which forms in the vein becomes a centre of purulent accumulation. When the unqualified term pylephlebitis is used, the adhesive variety is always indicated.

Morbid Anatomy.—In adhesive pylephlebitis the coats of the portal veins become thickened and their calibre is diminished, fibrin collects upon the constricted portion, and thus thrombi are formed. Sometimes the coagulum forms *before any recognizable change* in the coats of the vein has occurred. When this happens, the process may commence in a small branch and extend to the main trunk, or a single spot in a large branch may be the point where blood first coagulates. In either case, obliteration of the vein is the result. The wall of the vein is the seat of hyperplasia, adhesion of its two surfaces occurs, and as a result the vein is obliterated and a fibro-cellular cord alone remains. As a rule the liver is smaller in size than normal, and may exhibit on its surface cicatricial contraction, showing the lines of the obliterated vein.

On section, coagula may be found in all stages of formation.

The spleen is usually found much enlarged. The *abdominal cavity* is often filled with fluid, and the superficial abdominal veins on the right side are enlarged and tortuous. The *gall-bladder* is usually found full of greenish bile.

Etiology.—Certain blood conditions predispose to adhesive pylephlebitis, and chief among these are acute septic and malarial poisons. The most common and direct cause is *narrowing* of the trunk of the portal vein, from contraction of cicatricial tissue in the transverse fissure of the liver, or from pressure of enlarged lymphatic glands, tumors of the pancreas, omentum, or stomach :—hence cirrhosis plays the most important part. Blows, injuries to the walls of the vein, and inflammation of the tissue immediately about it, act as direct causes. The *secondary* causes are an extension of inflammation from inflamed hemorrhoidal tumors, from the umbilical phlebitis of the new-born, from severe local inflammation of the intestine, from extension of inflammation from the mesentery to the mesenteric vein, from a peculiar form of phlebitis called "gouty," and from a chronic inflammation excited by pressure of gall-stones.

Symptoms.—When the main trunk of the portal vein or its larger branches are not involved, the disease cannot be recognized. But when they are extensively involved, fluid rapidly accumulates in the peritoneal cavity, and after withdrawal it quickly reaccumulates. This is an important point in the diagnosis. The veins of the abdomen, and often those of the thorax, become enlarged, tortuous and prominent; at the same time hemorrhoids, which often attain immense size and become very painful, are developed. The spleen enlarges so rapidly in some cases that the extent of the enlargement can be determined each day. Profuse and exhausting vomiting, with hæmatemesis, is common, and diarrhœa, with frequent discharges of fluid blood from the bowels, marks the advanced stage. Gastro-intestinal hemorrhages and epistaxis may lead to fatal syncope. In the majority of cases its course is rapid;—if it is slow in its development, it gives rise to precisely the same symptoms as those of the latter stage of cirrhosis of the liver. Jaundice is never a prominent symptom. If it does occur, it is usually due to a complicating catarrh of the bile-ducts.

Physical Signs.—*Inspection* and *palpation* will give the evidences of fluid in the abdominal cavity, and the superficial veins will be markedly enlarged and cord-like.

Percussion.—The normal area of the hepatic dulness is diminished, unless waxy degeneration or some other disease of the liver precedes its development. The *spleen* is enlarged in all cases.

Differential Diagnosis.—*Cirrhosis* is the only disease which would be liable to be confounded with pylephlebitis. In the advanced stage, it is impossible to make a differential diagnosis. The previous history of the patient is important:—in *cirrhosis* it is one of chronic alcoholismus, gout, rheumatism, or syphilis, none of which can be regarded as causes of pylephlebitis. Cirrhosis is much slower in its development than pylephlebitis. The abdominal dropsy accumulates rapidly in pylephlebitis, while in cirrhosis it accumulates slowly, and does not return quickly after paracentesis. The stools in cirrhosis are firm and clay-colored. The urine contains abundant urates in cirrhosis; these are absent in pylephlebitis. Persistent tympanitis precedes the ascites of cirrhosis, and is absent in pylephlebitis.

Prognosis.—The prognosis is unfavorable. Death may result from asphyxia, from gastric and intestinal hemorrhage, and from exhausting diarrhœa.

Treatment.—Medication avails little in this disease; the treatment is altogether palliative. The diarrhœa and hemorrhage should be checked with vegetable astringents. If dyspnœa becomes urgent, on account of the large accumulation of the fluid in the abdominal cavity, paracentesis should be performed. The food should be highly nutritious, and taken in small quantities, at short intervals.

SUPPURATIVE PYLEPHLEBITIS.

Suppurative inflammation of the portal vein is always a secondary disease, and leads to the formation of small hepatic abscesses.

Morbid Anatomy.—The *wall* of the vein is the seat of the inflammatory process; it becomes thickened, and its cavity is filled with a puriform fluid, coagulated blood, or a stratified thrombus. The primary seat of the process may be the trunk of the vein before it enters the liver. It may extend to the smaller branches, and from them to the liver substance. If coagula occupy the venous twigs as well as the trunk of the vein, it is common for the puriform infiltration to take place only in them, while a firm clot obstructs the main channel. When the veins near the surface of the liver are the seat of suppurative pylephlebitis, extension of the process from the sheath of the vessels to the adjacent parenchyma gives rise to small abscesses. The liver becomes enlarged and softened, and circumscribed collections of pus are visible underneath its capsule.

On section, the calibre of the vena portæ is seen enlarged and gaping; the wall is thickened. Its contents vary: sometimes it only contains pus; at others, fibrinous matter and small coagula of blood are mixed in the purulent fluid. Abscesses are found along the course of the larger portal veins, and the smaller branches often terminate in larger collections of pus. If pieces of thrombi have been swept into the blood current, infarctions are found in all stages, from reddish-brown clots to purulent masses. The spleen is usually found enlarged, and of a dark purplish color.

Etiology.—The chief causes are ulceration and inflammatory processes in the abdominal cavity. Typhlitis, perityphlitis and ulceration of the vermiform appendix sometimes induce it. Diseases of the rectum, as recto-urethral fistulæ and suppurating hemorrhoidal tumors, chronic peritonitis, abscess of the spleen, suppurating mesenteric glands, diseases of the mesentery which have pus as their product, and diseases of the bile ducts, such as inflammation, ulceration and perforation, especially when caused by impacted gall-stones, often excite suppuration in the portal vein. Severe blows over the region of the liver have been followed by pylephlebitis. Suppurative gastritis may be followed by it.

Symptoms.—The symptoms of this disease are usually well marked. Pain is the first and most constant symptom. The location of the pain varies in different cases; it is generally most intense about the umbilicus and right hypochondriac region, just to the right of and below the xiphoid cartilage. Frequently it is felt below the spleen, and again it seems to come from, or extend to, the region about the cæcum. The pain is burning in character, and accompanied by slight tympanitis and tenderness. With the pain the temperature is elevated, the pulse-rate increased, and soon a more or less prolonged rigor occurs, during which the temperature will rise to 101°, 102° F., or even higher. After this comes a profuse and exhausting sweat. The rigors and sweats continue for two or three days, and may occur so regularly in the morning or evening as to suggest the presence of some form of malarial fever; usually, however, the chills are *irregular*. Slight jaundice, gradually deepening, but never very intense, is soon present, and sometimes assumes a greenish tint. The pulse is gradually increased in frequency, reaching in some cases 130 per minute. The spleen increases in size daily, and is quite tender to pressure. The appearance of the patient is that of one suffering from some grave form of disease. He becomes greatly emaciated, and there is more or less profuse diarrhœa, often containing blood. Hæmatemesis and bilious vomiting are frequently present, and as the disease advances the fever assumes a hectic type, with signs of general peritonitis, accompanied by painful tympanitis and obstinate vomiting. Ascites, if present, is slight. Petechiæ appear upon the surface, and aphthæ develop in the mouth. Typhoid symptoms usually come on toward the close, with low, muttering delirium, subsultus, somnolence, and fatal coma. The mind may be clear to the last, the patient dying in an extremely emaciated condition. In this disease there sometimes occur distinct remissions at the end of the first week, but this must not mislead one, for exhausting rigors and sweats will soon follow and lead to a fatal result. The *urine* is scanty, non-albuminous, and usually contains bile pigment.

Physical Signs.—By *palpation* and *percussion* both liver and spleen are found uniformly enlarged, and very tender, but the spleen is relatively much more enlarged than the liver.

Differential Diagnosis.—Suppurative inflammation of the portal vein may be mistaken for *adhesive* pylephlebitis, for *malarial fever*, *abscess* of the liver, and *catarrh* of the bile-ducts.

In suppurative pylephlebitis severe pain, rigors and sweats usher in the disease, and recur irregularly throughout its course; these never mark the advent of the *adhesive* variety. A large amount of fluid accumulates rapidly in the abdominal cavity in adhesive pylephlebitis, and it rarely, if ever, occurs in suppurative. Jaundice is the rule in suppurative pylephlebitis, and the exception in adhesive. The liver is smaller than normal in adhesive, and larger than normal, and tender, in suppurative. The spleen is enlarged in both diseases, but it is excessively tender in the suppurative form.

In *malarial fever* the rigors and sweats follow a definite order, while in suppurative pylephlebitis they occur irregularly. There is no pain in malaria, while in suppurative pylephlebitis it is diffused over the hepatic, umbilical and splenic regions.

Diarrhœa rarely occurs in *abscess* of the liver, and if present it is of short duration, often alternating with constipation, when the stools are firm and clay-colored; while profuse diarrhœa exists from the commencement in suppurative pylephlebitis. Jaundice is rare in hepatic abscess, and of common occurrence in suppurative pylephlebitis. Fluctuation is often present in abscess of the liver, and never in suppurative pylephlebitis.

In *catarrh of the bile-ducts* slight fever soon gives place to a normal temperature and a slow pulse, while there is a high temperature and rapid pulse throughout the course of suppurative pylephlebitis.

Prognosis.—Nearly all the cases of suppurative pylephlebitis are fatal. Its duration varies from one or two weeks to one or two months, the average being about one month. Death may occur from diarrhœa, from hemorrhage, from exhaustion, and from the intense gastric catarrh which may complicate the disease.

Treatment.—We are powerless to arrest this disease; and its treatment is altogether palliative. Morphia hypodermatically is the only reliable means of relieving the pain which is so distressing. Diarrhœa is a part of its natural history, and all the resisting power of the patient is required to withstand the exhaustion and cachexia which it produces. Although quinine has no controlling power over the disease, it may be used as an antipyretic and stimulant, and should be freely administered in connection with stimulants and a most nutritious diet.

AMYLOID DEGENERATION.

The most common degenerations of the liver are the amyloid and the fatty.

Amyloid, *waxy* or lardaceous degeneration of the liver, is never a primary disease. It is one of the painless enlargements of the liver.

Morbid Anatomy.—The degenerative process begins in the walls of the capillaries and small arteries, very rarely in the veins. Various theories have been advanced concerning the nature of this degeneration; some claim that it depends upon blood changes, and refer to the connection between waxy change and syphilis in support of their views. Others maintain that, the alkalinity of the blood being diminished, the normal relationship between its other constituents is disturbed, and as a consequence amyloid material or "*dealkalized fibrin*" is deposited; that the process is not one of simple infiltration. In detail the changes are as follows:—the capillaries are stretched and consequently have their diameter increased; their walls then become thickened by infiltration or deposit, so that their channel is narrowed or wholly occluded. The material deposited is a substance resembling albumen in its reaction; it is nitrogenous, homogeneous, and translucent, with a dull, shining surface. Its reaction is characteristic, a watery solution of iodine changing it to a deep red-brown color, which gradually passes off; if before it entirely disappears a drop of concentrated sulphuric acid is poured over it, a violet or deep blue-black color results. The change in the capillary walls is rapidly followed by a similar one in the walls of the arterioles; all the coats of the smaller arteries are involved simultaneously, the most marked change, however, being in their *muscular* coat. The amyloid change in the liver always begins in the radicles, midway between the centre and the periphery of the hepatic lobules. An extension of the infiltration to the adjacent liver-cells causes them to enlarge, become irregular in outline, and coalesce in masses; finally a whole lobule becomes involved. This enlargement, the increased lateral pressure, and the diminution of the lumen of the vessels, cause a decrease in the blood supply, and this leads to atrophy of the liver-cells. The liver is uniformly enlarged, sometimes to such an extent as to nearly fill the abdominal cavity. It is stony hard, non-elastic, heavier than normal, its specific gravity is increased, and its edges are sharp and well defined. The *capsule* is tense, shining, and has a gray "waxy" look. In some rare cases enlarged lymphatics are found in its transverse fissure, and then jaundice may be present.

On section, the liver cuts with a "creaking" sound, like bacon (hence its name lardaceous), and the cut surface has a "cheese yellow," or dull gray, glistening appearance. The whole or a part of the liver may be involved. If the whole liver has undergone amyloid degeneration, the cut surface presents a homogeneous appearance, and either the outline of the lobules is lost or they are seen to be enlarged and irregular; sometimes a "yellow rim" can be traced at their periphery, due to *fatty* change. The *microscope* shows the lobules to be increased in size; the liver cells at the periphery of the lobules are infiltrated with small spherules of fat; midway between the surface and centre of the lobule there is a zone of amyloid matter, and in some instances there is a pigment deposit in the zone just about the vena centralis. We have, then; *first*, the fatty zone at the periphery of the lobules; *secondly*, the waxy intermediate zone; and, *thirdly*, the pigment zone around the central vein. The liver cells lose their polygonal outline,[1] and become irregularly oval or circular in shape. Their cell-walls

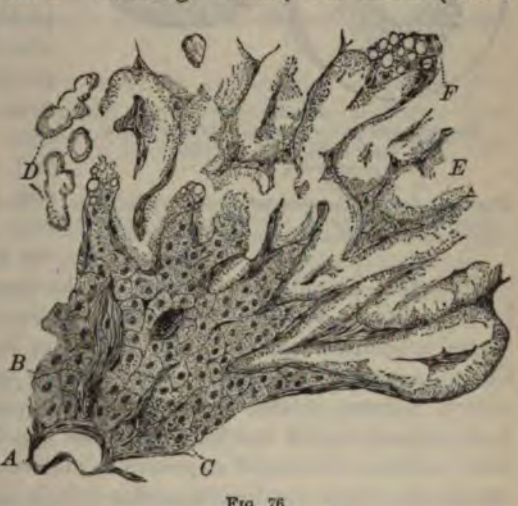

FIG. 76.

Amyloid Degeneration.

Section of a Lobule of the Liver in amyloid degeneration.

A. Central vein of the lobule.
B. Normal hepatic cells.
C. Pigmented cells.
D. Commencement of the amyloid change.
E. Waxy zone—the hepatic cells completely changed. At F, cells are shown containing fat. × 350.

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[1] Quite recently Cornil, in examining many specimens, found no change in the hepatic cells.

cannot be traced, but merge into the neighboring mass of amyloid material. The contents of the cell are atrophied, nuclei are not visible, though occasionally a nucleus of one cell stands out enlarged and shining. A semi-transparent homogeneous mass fills the cell, causing it to present the appearance of "waxy scales." Fatty degeneration frequently coexists with amyloid change. The liver will then partake of the characters of both waxy and fatty change. Cirrhosis or simple atrophy may precede or be associated with waxy degeneration, and syphilitic nodules and cicatrices from "perihepatitis syphilitica" may exist in a liver which had subsequently undergone waxy degeneration. The *kidneys* undergo amyloid degeneration. The *spleen* is enlarged, firm and waxy; the *lymphatics* generally, and the *gastro-intestinal mucous membrane* may also become the seat of the amyloid change.

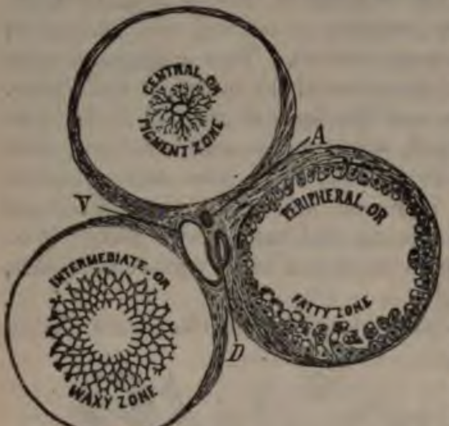

FIG. 77.
Diagram showing the three Intralobular Zones.
V. Small branch of Portal Vein.
A. Hepatic Artery.
D. Bile Duct.
The three vessels are surrounded by fibrous connective-tissue, a prolongation of Glisson's capsule, and altogether constitute the elements of the Portal Canal.

Etiology.—Seventy-five per cent. of the cases of waxy liver occur in males between twenty and fifty years of age. Syphilis is its chief cause. Prolonged suppuration and chronic diseases of bone are also prominent causes. Caries, necrosis, especially when the larger joints are involved, rickets, dysentery, chronic intestinal ulceration, and sometimes chronic pyelitis are reckoned among the morbid conditions which predispose to it. A scrofulous diathesis, prolonged exposure to malaria, and a cancerous cachexia are among the rarer conditions under which amyloid degeneration is developed.

Symptoms.—Its advent is never well defined, occurring as it does with diseases which are prone to cause anæmia and wasting of the body; its *subjective* symptoms are at first very obscure. There is no pyrexia accompanying it. The patient has a sense of weight, fulness, and constriction in the right hypochondrium, never amounting to pain, the sensation being rather one of discomfort. Jaundice and ascites are not part of the natural history of amyloid liver. When jaundice is present, it is due either to an intercurrent catarrh of the bile-ducts, or to pressure from the enlarged lymphatics in the transverse fissure. Ascitic accumulations result from complicating peritonitis or from the pressure of enlarged glands in the transverse fissure. Late in the disease, diarrhœa and vomiting are induced by the slightest irregularities in diet, on account of the implication of the gastro-intestinal tract in the amyloid change. On an examination of the blood of one who has suffered from waxy degeneration of the liver, the proportion of white blood globules will be found increased. The skin has a pale, "waxy" look, and oftentimes exhales a peculiar odor. Early in the disease the fæces are firm, and pale in color, because of absence of bile; later, when the so-called "waxy diarrhœa" sets in, there are pale mucous stools, sometimes having a dysenteric odor. The *urine* is increased in amount, is of a pale lemon-yellow color, low specific gravity, averaging about 1.010, and contains albumen. The amount of the albumen increases as the disease progresses; epithelial and large hyaline casts are present. Anasarca may occur in the advanced stage of the disease, with general dropsy.

Physical Signs.—*Inspection* in the advanced stage of the disease shows bulging of the hepatic and splenic regions. The sharp edge of the liver will be found projecting below the free border of the ribs, with a firm, hard, resistant feel and a smooth surface. The *spleen* is increased sometimes to three times its normal size, and is resistant.

Percussion.—The areas of hepatic and splenic dulness are increased equally in all directions.

Differential Diagnosis.—Waxy liver may be confounded with the *first stage* of *cirrhosis*, which has already been referred to, and also with *fatty liver*, the diagnosis of which is considered in the history of that disease.

Prognosis.—The prognosis is unfavorable; the disease is progressive and fatal, and we can only hope to arrest its progress when it occurs with syphilis. Its exact duration cannot be estimated, since its beginning is so obscure. It is usually slow in its development, and extends over a period of many months and sometimes years. Among its most frequent *complications* are diarrhœa, purulent peritonitis, perihepatitis, fatty and waxy kidney, dysentery, pulmonary œdema, pneumonia, and pulmonary gangrene. Death may result from exhaustion due to faulty nutrition or diarrhœa, from general dropsy, and from uræmia or other complicating diseases.

Treatment.—The first indication for treatment is to be found in its causation. If it is developed in connection with disease of the bones, the diseased bones should be removed, and prolonged suppuration arrested. If syphilis exist, antisyphilitic measures are indicated. In phthisis, empyema, and other similar diseases, attention must be directed to the primary disease. Alkalies have been administered, on the ground that the amyloid material is "dealkalized fibrin," and that with the suppurative process a large quantity of alkalies pass rapidly out of the system. When once the amyloid process is well established, the diet should consist largely of meat; sugars and starch should be avoided. Alcoholic stimulants may be taken in moderation. The climate, clothing, and general hygienic surroundings of the patient are important. Tonics, and iron combined with some preparation of iodine are indicated in all cases. But when a history of syphilis is clearly elicited, then iodide of potassium may be given in large doses, with the hope of arresting the progress of the disease. Alkalies, chiefly potassic salts, are in great repute among the advocates of the "alkaline treatment," and they can be given without fear of injury in nearly every case. It is claimed by some that ammonium chloride produces the most beneficial effects, but my own experience does not sustain the strong statements that have been made regarding it. The mineral waters are too exhausting for this class of patients, and, although they may give temporary relief, should not be used in its treatment. External applications, such as iodine ointments, and nitro-muriatic acid baths, have been used, but without any markedly favorable results. If uræmic symptoms develop, measures for their relief should be promptly instituted. Drastic purges, however, must not be employed, for the condition of the gastro-intestinal tract contra-indicates their use.

CHRONIC ATROPHY OF THE LIVER.

The term "atrophy" includes all those forms of hepatic disease in which there is a diminution in the size of the liver, due to decrease in either the *number* or the *size* of the hepatic cells. Strictly speaking there are six *varieties* of hepatic atrophy, viz.:—*acute yellow* atrophy, *induration* atrophy, from repeated attacks of perihepatitis, *cirrhosis*, atrophy from long continued *hyperæmia*, atrophy from *adhesive pylephlebitis*, and *chronic atrophy*. All these varieties have already been considered under their proper head, except the one termed *chronic atrophy*. The liver in chronic atrophy may have a brown or red color; hence the term chronic brown or *red atrophy*. The pathological processes which lead to it are similar to those which take place in atrophy of any gland tissue.

Morbid Anatomy.—Chronic atrophy may be partial or general. The liver is smaller than normal, and its diminution in size is uniform. Sometimes its weight is decreased to twenty-four ounces. It is flabby and tenacious, its edges are thickened, its *capsule* is smooth, of normal thickness, and free from adhesions. Sometimes it is shrivelled, but never "hob-nailed" or lobulated. In partial atrophy, there are often large depressions on the surface, the result of the pressure of neighboring organs, or of tight lacing, or the wearing of belts tightly about the waist. A large quantity of thin blood flows from its *cut surface*, which has a uniform brown-red or mottled appearance. The sections of the larger portal vessels gape. The outline of the lobules is obliterated. The portal vein and its branches are enlarged, the walls assume a yellow-red color, the fibrous sheath, derived from Glisson's capsule, is thickened, and its finest ramifications end in blind pouches or club-shaped extremities near the periphery of the lobule. The capillaries are usually filled with pigment granules. Sometimes the hepatic vein is involved, but never to the same degree as the portal. The bile ducts are either empty or contain a small amount of pale, turbid fluid, having traces of albumen. By the *microscope* the granular contents and nuclei of the hepatic cells will be found to have disappeared. The cell walls will be indented and shrivelled, and often pigment granules, traces of bile coloring matter, or little fatty spherules will be seen occupying their place. When the atrophy is partial, these morbid changes will be found to exist underneath the depressions on the surface, where pressure has been long continued. The *spleen* is usually enlarged, but only slightly. The *gastro-intestinal* mucous membrane is the seat of catarrh, and sometimes there are punctate hemorrhages beneath its mucous surface.

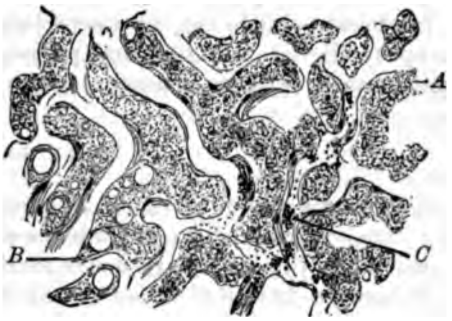

FIG. 78.

Chronic Atrophy.

Section of portion of a Lobule.

A. Hepatic cells. shrivelled and pigmented, with disappearance of nuclei.
B. Cells containing fat spherules.
C. Pigmented capillaries. × 800.

Etiology.—The causes of *partial* chronic red atrophy are tight lacing and pressure from peritoneal effusions and from abdominal tumors. It may also be caused by extensive adhesions to adjacent organs. General atrophy may be due to the contraction of the new connective-tissue developed in the substance and on the surface of the organ, and to chronic malarial infection.

Symptoms.—The symptoms closely resemble those of cirrhosis of the liver. There is loss of appetite, furred tongue, a sense of weight in the right hypochondrium, accompanied by the train of symptoms which attend chronic gastritis. There is profuse and exhausting diarrhœa alternating with constipation, hemorrhoidal tumors, hæmatemesis, intestinal hemorrhages, tympanitis, ascites and emaciation,—all which may be present in interstitial hepatitis.

Physical Signs.—*Palpation.* If the surface of the liver can be reached, it will be found smooth and resistant.

Percussion.—The area of hepatic dulness will be diminished in every direction.

Differential Diagnosis.—The differential diagnosis between chronic red atrophy and *cirrhosis of the liver* is always difficult. In *cirrhosis* there will be the history of spirit drinking, of gout or rheumatism; none of which will form a part of the history of chronic atrophy. In cirrhosis, slight jaundice is common toward the end of the disease; it never exists in uncomplicated red atrophy. Venous stigmata, which are so often met with on the cheeks in cirrhosis of the liver, are absent in chronic atrophy. Diarrhœa is not so common or persistent in cirrhosis as in atrophy. The urine in cirrhosis is high colored and contains albumen, bile pigment, and lithates; while in atrophy it is pale, and bile pigment is rarely present. In cirrhosis the liver is hob-nailed and rough on palpation, while in atrophy it is smooth on its surface.

Prognosis.—Recovery from chronic red atrophy never occurs. Death may result from exhaustion due to the diarrhœa, from hæmatemesis or intestinal hemorrhages, and from general dropsy.

Treatment.—Little can be accomplished in the treatment of this disease except to alleviate suffering and prolong life; it is incurable. When the ascites causes dyspnœa it must be removed by mechanical means.

FATTY LIVER.

Fatty degeneration of the liver occurs either as a fatty *infiltration* or as a *metamorphosis* of the albuminous elements of liver-tissue into fat. It is one of the painless enlargements of the liver.

Temporary fatty infiltration of the liver is a physiological state which occurs after the ingestion of food rich in hydrocarbons.

Morbid Anatomy.—In fatty infiltration, the liver is increased in size and has a peculiar flattened appearance. Its surface is smooth and presents a pale brown or light yellow color, according to the degree of infiltration; its borders are smooth and rounded, and it has a doughy, flabby feel, and pits on pressure. Its *capsule* is tense, shining, and transparent; enlarged tortuous vessels are frequently seen traversing it.

On section the organ cuts readily, and the warmed knife blade is coated with oil globules; little blood flows from the cut surface. In the early stage it presents a reticulated, mottled appearance, of a dull yellow color. This appearance is due to the rim of fat globules around the periphery of the acini, while the parts immediately about the central vein are intensely congested and pigmented. In the latter stage, the whole surface presents a homogeneous bright "butter yellow" color, and fat cells are found occupying the centre of the lobule. Amyloid degeneration and fatty infiltration may be found in the same organ. With the *microscope* the lobules will be found enlarged, and the cells at their periphery are rounded, larger than normal, and filled with fat globules. These fat globules vary in size, sometimes a single oil drop occupies the entire cell space, the clouded nucleus and glandular contents being pressed up against the cell wall. At first the capillaries near the central vein are distended, and the cells about the vein are infiltrated with fat to a slight extent; later on

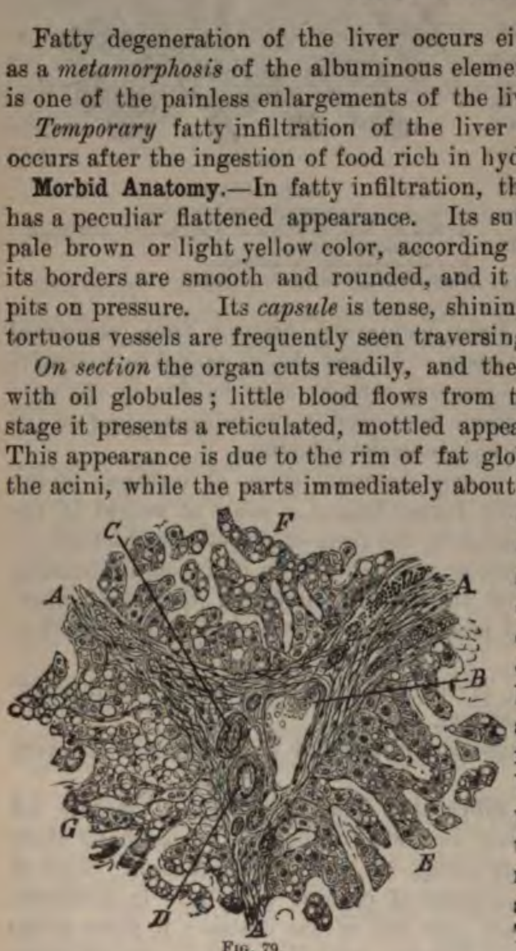

FIG. 79.

Fatty Infiltration.

Section of a Portal Canal and portion of three Lobules.

A, A, A. *Connective-tissue of Portal Canal.*
B. *Branch of Portal Vein.*
C. *Hepatic Artery.*
D. *Hepatic Duct.*
E. *Periphery of a Lobule, in which small fat globules are seen in the liver-cells.*
F. *Same as E, with increased amount of infiltration.*
G. *Periphery of a third Lobule, in which the lesion is still further advanced.* × 280.

appeared. The cell walls will be indented and shrivelled, and often pigment granules, traces of bile coloring matter, or little fatty spherules will be seen occupying their place. When the atrophy is partial, these morbid changes will be found to exist underneath the depressions on the surface, where pressure has been long continued. The *spleen* is usually enlarged, but only slightly. The *gastro-intestinal* mucous membrane is the seat of catarrh, and sometimes there are punctate hemorrhages beneath its mucous surface.

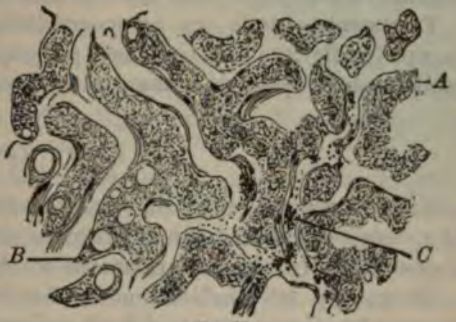

FIG. 78.

Chronic Atrophy.

Section of portion of a Lobule.

A. Hepatic cells, shrivelled and pigmented, with disappearance of nuclei.
B. Cells containing fat spherules.
C. Pigmented capillaries. × 300.

Etiology.—The causes of *partial* chronic red atrophy are tight lacing and pressure from peritoneal effusions and from abdominal tumors. It may also be caused by extensive adhesions to adjacent organs. General atrophy may be due to the contraction of the new connective-tissue developed in the substance and on the surface of the organ, and to chronic malarial infection.

Symptoms.—The symptoms closely resemble those of cirrhosis of the liver. There is loss of appetite, furred tongue, a sense of weight in the right hypochondrium, accompanied by the train of symptoms which attend chronic gastritis. There is profuse and exhausting diarrhœa alternating with constipation, hemorrhoidal tumors, hæmatemesis, intestinal hemorrhages, tympanitis, ascites and emaciation,—all which may be present in interstitial hepatitis.

Physical Signs.—*Palpation.* If the surface of the liver can be reached, it will be found smooth and resistant.

Percussion.—The area of hepatic dulness will be diminished in every direction.

Differential Diagnosis.—The differential diagnosis between chronic red atrophy and *cirrhosis of the liver* is always difficult. In *cirrhosis* there will be the history of spirit drinking, of gout or rheumatism; none of which will form a part of the history of chronic atrophy. In cirrhosis, slight jaundice is common toward the end of the disease; it never exists in uncomplicated red atrophy. Venous stigmata, which are so often met with on the cheeks in cirrhosis of the liver, are absent in chronic atrophy. Diarrhœa is not so common or persistent in cirrhosis as in atrophy. The urine in cirrhosis is high colored and contains albumen, bile pigment, and lithates; while in atrophy it is pale, and bile pigment is rarely present. In cirrhosis the liver is hob-nailed and rough on palpation, while in atrophy it is smooth on its surface.

Prognosis.—Recovery from chronic red atrophy never occurs. Death however, attended with pain. The slightest indiscretion causes an attack of gastric catarrh and diarrhœa, which persists long after the removal of the cause. The patient is anæmic and moody, and there is a general loss of muscular power, with a disposition to sleep. The blood is hydræmic. The skin is sometimes shining, always "velvety" to the feel, and often pasty and smooth, like that of a wax figure. The integument all over the body feels smooth, velvety, and flabby. Dyspnœa results as much from the weakness and anæmia, as from pressure of the enlarged liver. When symptoms of acholia, due to the altered state of the blood, are attended by absence of bile in the intestinal tract, rapid anæmia, exhaustion, delirium and collapse occur, and extensive fatty metamorphosis is then usually associated with some other hepatic degeneration. The fæces are usually normal in color and the bowels are irregular and constipated; in the highest grades of fatty metamorphosis they are pale and clay-colored, and attacks of diarrhœa are frequent. The *urine* is pale, non-albuminous, and of a low specific gravity.

Physical Signs.—*Palpation.* The rounded smooth edges of a uniformly enlarged liver are readily felt below the border of the ribs; the organ has a doughy, soft feel. When fatty degeneration occurs with waxy or colloid disease, the liver is diminished or is of normal size and smooth.

Percussion.—The area of hepatic dulness is increased in all directions, the increase being mainly downward and forward.

Differential Diagnosis.—Fatty and *waxy* degeneration are frequently mistaken for each other. In *waxy liver* a history of syphilis, prolonged suppuration, or disease of bones will be elicited; in fatty liver there is a history of alcoholism, prolonged wasting disease, or one of high living and sedentary habits. In waxy liver, the skin is pale, dry, and has a peculiar odor resembling that of *indigo;* in fatty liver the skin shines with fat, and has a velvety feel. The blood is hydræmic in fatty liver, and is leukæmic in waxy liver. The urinary symptoms in both are distinct: in waxy liver the urine is often increased in amount, is albuminous, and contains casts; in fatty liver it is normal. In waxy liver the fæces are early deficient in bile and pale in color; in fatty liver they are normal until an advanced stage of the disease is reached. A waxy liver is hard; a fatty liver is soft and flabby. A waxy liver may become much larger than a fatty liver, and its edges are sharply defined; while in fatty liver they are smooth and rounded. With a waxy liver the spleen is enlarged, but with fatty liver it is normal in size.

Prognosis.—Fatty infiltration of the liver is not a grave form of disease There is danger only when fatty degeneration of liver-tissue occurs Death may result from fatty heart, pulmonary œdema, acholia, apoplexy, the exhausting diarrhœa, and from the complications already referred to.

Treatment.—When the diet, mode of life, or climate is the main elemen in its causation, the indications for treatment are simple. A restricted diet, with no fat or sugar, and with regular daily exercise in the open air will, in most cases, increase the patient's strength and lessen the size of the liver. Care must be taken not to stop alcoholic stimulants too suddenly, for fatty heart may co-exist. They must be decreased gradually. In all cases, a residence in an elevated temperate region, free from marshes, is important. The vegetable bitters combined with alkalies will aid in restoring the appetite when it is lost. Iron should be administered in the form of the carbonates and lactates. Rhubarb and aloes will best relieve the constipation, and vegetable astringents control the diarrhœa. In syphilis, iodide of potassium is of service. In the fatty liver of phthisis, nothing can be expected from treatment so long as the phthisis is progressive.

PIGMENT DEGENERATION.

The pigment or *melanotic* liver is that form of hepatic degeneration in which there is an abnormal deposit in the liver of pigment derived from the coloring matter of the blood. In pigmentation there must be primarily a fault in the circulation or in the blood-vessels; usually it is the result of slowing of the blood current. The red corpuscles either pass through the walls entire, or liberate the hæmoglobin, which then transudes the capillary vessels. The blood from the spleen, loaded with pigment, passes into the portal vein, is carried through the interlobular veins, then into the veins just within the periphery of the lobule. Hæmoglobin remaining in the portal capillaries soon breaks up into hæmatoidin and, according to some, into melanin, though we are inclined to-day to regard melanin as altered hæmatoidin. This hæmatoidin is first yellowish, later it consists either of brownish-black granules or crystals of an intensely black color. Both hæmatoidin and melanin remain unaltered when once formed. Pigmentation of the liver is confined to the vascular system. Extensive capillary stagnation with a large amount of pigment matter occluding the vessels gives rise to atrophy of the cellular structure.

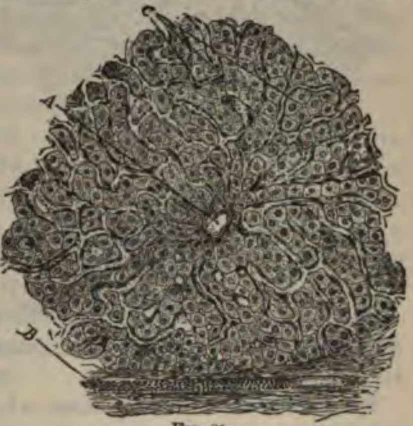

FIG. 81.

Pigmentary Degeneration.

Section of an Hepatic Lobule from a case of pernicious fever.

A. Central vein of the lobule.
B. Longitudinal section of a small hepatic duct.
C. Vessels containing small pigment granules in great numbers. The pigmentation in this case was pretty general throughout the intralobular-capillaries. × 250.

Morbid Anatomy.—The liver is at first enlarged from congestion and the capsule is smooth and tense; afterward the organ becomes smaller than normal and atrophies, its color being much deeper than in the earlier stage and its edges sharply defined.

On section, in the first stage, dark blood flows from the congested parenchyma. If the cut surface presents a mottled appearance there is a steel-gray or black ring around, and slightly encroaching on, each lobule, shading off toward the central vein. In congestion of the liver pigmentation commences about the central vein, and gradually diminishes toward the periphery of the lobule. If the surface is uniform it presents a color which resembles "graphite," a blackened gray color having a slight lustre, and the pigment deposit is seen to have reached the central vein. Occasionally spots of extravasation are found scattered throughout the organ. On section of an atrophied "pigment liver" the whole cut surface is black, and all trace of the lobules is frequently lost. A *microscopical* examination shows the capillaries, not only portal but hepatic, filled with granules or crystals, either throughout their entire extent or in isolated patches. The hepatic cells contain no pigment, but are filled with oily or amyloid material, or sometimes with dark-colored bile. Leucin has often been found in the parenchyma of a pigmented liver. In an "atrophied" liver, the lobules and cells are shrunken, and the capillary system is a mass of pigment. The *spleen* is softened and usually enlarged, never smaller than normal, and is more extensively pigmented than the liver. In some cases of pigment liver, there are evidences of hemorrhages into the various serous cavities. In connection with pigment degeneration of the liver, pigmentation may occur in all the organs of the body.

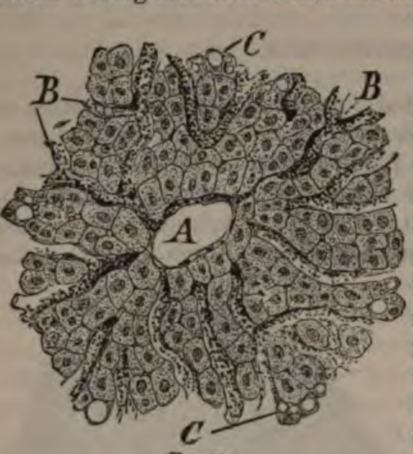

FIG. 92.

Pigment Degeneration.

Section of the same tissue as preceding cut, showing the centre of a lobule more highly magnified.
A. *Central vein of the lobule.*
B, B. *Strongly pigmented capillaries.*
C, C. *Hepatic cells infiltrated with fat.* × 450.

Etiology.—Malarial infection is the only known cause of melanotic liver, but whether a large *amount* or peculiar *kind* of malarial poison is necessary for its development has not as yet been determined.

Symptoms.—Frequently those who have had extensive pigment deposits in all the organs of the body, have given during life no symptoms to indicate their presence. The first effect of extensive pigmentation of the liver is an abnormal secretion of bile. The liver is enlarged and tender to pressure. The skin in the milder forms is ash-colored, and in severer forms it is of a dark-bronze hue. There may be slight jaundice. There is gastro-intestinal catarrh with nausea, loss of appetite, flatulence, painful tympanitis, vomiting, and diarrhœa which may pass into dysentery. In severe cases, hemorrhage from the gastro-intestinal mucous membrane and from the kidney occurs, attended by exacerbations and remissions corresponding to febrile exacerbations and remissions. There is rapid emaciation and extreme exhaustion with giddiness, headache, and ringing in the ears. Occasionally the vertigo comes on so suddenly that the patient falls to the ground without the least warning. Active delirium is often followed by profound coma. The urine and fæces are passed involuntarily during the period of stupor. Coma is the most frequent termination of the cerebral variety of pigment liver. In severe cases which terminate in recovery there is often temporary loss of memory.

Physical Signs.—*Inspection* shows the ashy-gray, jaundiced, or brown colored skin.

Palpation.—The surface of the liver is smooth, and in the first stage the organ is larger, softer, and more tender than normal. In the second stage it is small and hard.

Percussion.—In the early stage the area of hepatic dulness is increased; in the later stage it is uniformly diminished.

Differential Diagnosis.—The liability of confounding pigment degeneration with other diseases of the liver is not so great as is the difficulty of recognizing its existence. If, in intense malarial infection, cerebral or urinary symptoms come on suddenly with hemorrhages from the mucous surfaces, a bronzed hue of the skin, and the physical changes in the size of the liver already referred to, pigmentation of the liver may be suspected; and if, in addition to these, pigment matter is found in the blood, the diagnosis will be established.

Prognosis.—The prognosis is favorable if the patient can be removed from the source of malarial infection. The elements which render the prognosis unfavorable are severe cerebral and renal symptoms combined with signs of extensive portal obstruction. *Death* may occur from exhaustion due to the diarrhœa, dysentery, or intestinal hemorrhage.

Treatment.—The preventive treatment corresponds to the preventive treatment of malarial fever. When the disease is once established, the chief indication is to administer large doses of quinine. The symptoms in all varieties of the disease remit as soon as the individual is brought fully under the influence of this drug. Purges act unfavorably. If the cerebral symptoms are urgent, ammonia may be combined with quinine. Preparations of iron and a change of residence to a non-malarial district are essential to its successful management. The diet should be of the most nutritious character and non-stimulating.

CANCER OF THE LIVER.

Cancer of the liver may be either primary or secondary. It is secondary to cancer of the stomach in one-half of the cases. It has been estimated that one out of every one hundred persons has cancer of the liver. The varieties of cancer met with in the liver are *scirrhus*, *medullary*, *melanotic*, and *colloid* cancer. Infiltrated cancer without any change whatever in the form of the organ has been found.

Scirrhus is usually primary, while medullary is almost always secondary. Scirrhus makes its appearance first as rounded masses. These masses increase rapidly and soon attain their full size, which varies from that of a pea to that of an orange; they then remain stationary for a time until the fibrous tissue contracts. The number of these nodules varies *inversely* with their size. Scirrhus developments usually commence in the interlobular spaces and gradually extend toward the centre of the lobule. As the liver-cells are being crowded upon, the portal capillaries disappear, while the hepatic vessels enlarge and ramify in the cancerous mass as a new and peculiar vascular net-work. The neighboring lymphatic glands may also become infiltrated with cancer, and often exert sufficient pressure upon the bile-ducts to obstruct the outflow of the bile. The cancerous growth sometimes involves the walls of the portal vein, and, extending in the direction of the capillary terminations, fills up their channel. The bile-ducts also may be obstructed, distended, or ruptured. With these changes, the centre of the cancer-nodule becomes harder and harder; or by shutting off its own nutrition, the interior of the nodule becomes fatty, while the periphery is soft and vascular. The obliteration of the capillaries at the exterior of the mass shuts off the nutrition of the adjacent liver-cells, and this induces fatty degeneration. The theory of the development of medullary cancer (the *implantation theory*, as it is called) is that cancer-cells pass through the lymphatics, or blood-vessels, and reaching the interlobular spaces become the starting points of the cancer development. This theory has received much attention, and experiments seem to warrant our adopting it as one method, at least, in which cancer may develop.

Medullary cancer is simply a modification of scirrhus. Rapidity of development is the distinguishing pathological difference,—the line between the two forms often being arbitrarily drawn, for scirrhus may pass into medullary, and *vice versâ*.

Melanotic cancer of the liver is also of rapid growth. The nodules, though very numerous, are small in size. The cancer-cells have a deposit at their centre of yellow, brown or blackish pigment, the "granite" looking spot shading off toward the periphery. Its course is the same as in other varieties of hepatic cancer.

Colloid cancer is of rare occurrence in the liver, appearing only as a degenerated form of scirrhus or medullary cancer. If either of these forms undergoes mucoid or colloid degeneration, a gelatinous, gray, tenacious fluid takes the place of the cancer-juice, while the fibrous framework becomes more distinctly alveolar. *Melanotic sarcoma* has been found; it often pursues as malignant a course as true carcinoma.

Morbid Anatomy.—In nodular *scirrhus cancer* the liver is irregularly increased in size, the right lobe being usually most affected. With medullary cancer it is often so much enlarged as to fill the abdominal cavity. In color it is darker than normal, and it is increased in weight, sometimes reaching twenty pounds. Upon its surface are nodules, hard, elastic, rarely fluctuating, and umbilicated at their centres. Occasionally, there are no nodules on the surface, the cancerous developments being confined to the interior of the organ. The *capsule* of the liver is thickened and sometimes the seat of cancerous development. Adhesions connecting it to the adjacent parts are the result of intercurrent local peritonitis.

On section, if the degeneration is advanced, the liver cuts hard, and creaks like cartilage under the knife. The cut surface is seen studded with nodules, the diameters of which vary from one-eighth of an inch to four inches. Between the nodules the liver-tissue is sometimes congested, and of a dark red color, or it is atrophied. The nodules increase in density from their centre outward, or have a central cavity filled with fatty granules. On pressing them, more or less cancer-juice exudes according to the density of the tumor. The color of the tumor varies from a glistening dirty white to a deep red, according as the vascular net-work is meagre or abundant. If there has been obstruction to the bile ducts the parenchyma will be of a bright yellow color. Evidences of extravasation from distended vessels may be found throughout the liver-tissue and often in the interior of the cancerous growth.

Under the microscope, a cancer nodule will be found to consist of a fibrous framework or "stroma" in which are cancer-cells and cancer-juice. In scirrhus the fibrous stroma is greatly in excess of the other elements. The cancer-juice contains a large amount of fine granular matter, nucleated cells and distinct free nuclei. The cells are of large size and irregular, and the nuclei and nucleoli are often multiple and very distinct. The walls of the capillary vessels in the tumor are thin, and their calibre large. A ring of liver cells at the margin of the cancer-nodule exhibits well-marked degeneration.

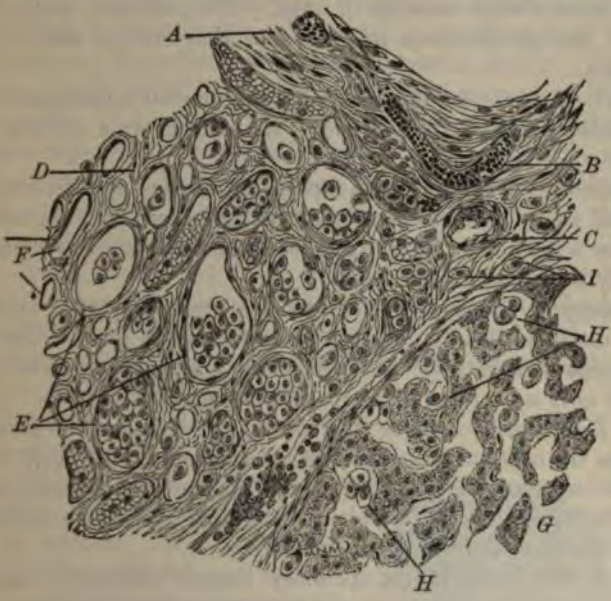

FIG. 83.

Cancer of the Liver.

Section showing part of a cancerous nodule with the contiguous hepatic tissue.

A. *Connective-tissue of a portal canal in which the nodule was developed.*
B. *Hepatic duct in longitudinal section.*
C. *Hepatic artery.*
D. *Stroma of cancer.*
E. *Alveoli of the same filled with "cancer cells."*
F. *Empty alveoli.*
G. *Periphery of an hepatic lobule bordering on the cancer, infiltrated at H H.*
I. *Infiltration of connective-tissue with same.* × 300.

Medullary Cancer.—The gross appearances of the liver are the same as in scirrhus, except that the nodules are fewer and larger. They are very soft and fluctuating, and frequently the more advanced tumors have ruptured through the peritoneal envelope of the liver. In this variety the cancer nodules are often lobulated. Those which occupy the surface of the liver project as large irregular tumors.

On section large nodular masses of curdy-white homogeneous matter resembling fœtal brain-substance are found scattered throughout the liver-tissue. Between the cancer nodules the liver substance is more or less intensely congested. Dark red hemorrhagic spots are seen scattered over its cut surface.

On a *microscopic examination* a small amount of fibrous stroma is found containing a very large number of cells. The cells are much larger than in scirrhus, though the same in kind, and they are the seat of more fatty degeneration.

Melanotic Cancer.—In common with the morbid appearance of all cancerous developments, we find, besides, that the liver is nodular and very dark.

On section the surface presents a peculiar mottled appearance resembling *granite,* and there are numerous small nodules studding the whole gland. On pressure a dark fluid flows from the cancerous mass, varying in color from a gray-brown to a deep black.

A *microscopical examination* shows a stroma varying in amount and color. Sometimes it is colorless, sometimes very dark. The degree of vascularity has wide ranges; the cells at certain spots in the liver often disappear and only a peculiar pigment color remains.

Colloid Degeneration.—The surface of the liver in this form of cancer differs from the other varieties in that it is smooth with large lobulations.

Under the *microscope* the cancerous mass is made up of large and spherical alveoli with thin walls. The alveoli contain mucoid or colloid matter, with fatty material and a few epithelial cells.

Etiology.—The causation of primary hepatic cancer is unknown; in most instances there exists an hereditary predisposition. It is a disease of middle life, occurring oftenest between the ages of forty and sixty-five. Medullary cancer of the liver, especially when secondary, is sometimes met with in early life, even as early as the fourth year. It occurs equally among males and females. Some have dated its development from some great mental emotion or strain, others from the receipt of a blow upon the right hypochondrium. Cancer of the liver is often secondary to cancer of the stomach, mamma, ovary, uterus, pancreas, brain, or portal vein. Clinical experience indicates that extirpation of external cancerous masses is very apt to be followed by cancer of the liver.

Symptoms.—The early symptoms of hepatic cancer are obscure. The more superficial its development, the more marked are the symptoms and the easier the diagnosis. It will be noticed that the individual is gradually losing flesh and strength, he complains of a sense of weight and fullness in the right hypochondrium, he is anæmic, and the surface assumes a doughy hue; with these there may be pain localized over the hepatic region, or shooting up toward the right shoulder, and sometimes to the back. The pain soon becomes lancinating in character, and is localized at some point over the liver which is tender to pressure. There is loss of appetite, flatulence, nausea, vomiting, and constipation alternating with diarrhœa. The vomiting is often profuse and persistent. There is progressive emaciation, and the skin assumes an earthy pallor. Jaundice is present in one-half of the cases, and is due either to compression of the bile ducts or to intercurrent catarrh of the ducts, and when once developed it is permanent. Ascites occurs more frequently than jaundice; the accumulation at first is inconsiderable in amount and increases slowly. It is due to compression of the portal vein by the cancerous tumor or by enlarged glands in the transverse fissure, or to chronic peritonitis. Œdema of the feet comes on late. The temperature is normal or sub-normal. Dyspnœa may become an urgent symptom in the advanced stage of hepatic cancer. The cervical and inguinal glands may be enlarged. Hemorrhages from the stomach, intestines, mouth, and vagina, with petechial and ecchymotic spots, are sometimes accompaniments of hepatic cancer. It is to be remembered that hepatic cancer may run its entire course without pain, without jaundice, and without ascites. In *medullary* cancer, loss of flesh and the peculiar cancer countenance may not appear until the end of the case. The fæces are normal at first, later they are firm and clay-colored. The fluid stools of cancer diarrhœa contain no bile. The *urine* is scanty and high-colored. Deposits of lithates and of bile pigment are rarely absent.

Physical Signs.—*Inspection.* There may be a perceptible bulging in the right hypochondrium and the outlines of large nodules may be visible.

Palpation discloses an enlarged and irregularly shaped liver, tender to pressure. Hard, smooth nodules are felt over its surface, which rarely fluctuate. If the nodules are umbilicated it establishes the diagnosis of cancer. In colloid cancer of the liver, and when the cancerous development is central, no nodules will be felt.

Percussion.—The area of hepatic dulness is irregularly increased and marked by an irregular line of flatness below the free border of the ribs.

Auscultation.—A friction sound, caused by the rubbing of the roughened peritoneal surfaces, is sometimes heard.

Differential Diagnosis.—Cancer of the liver may be mistaken for *hydatids*

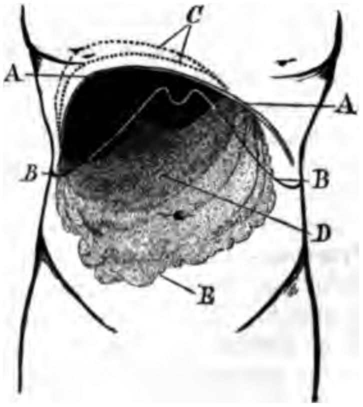

FIG. 84.

Diagram showing enlargements of the Liver as determined by percussion.

A, A. Line of diaphragm.
B, B. Lower border of costal cartilages.
C. Dotted line enlargement upward.
D. Shaded area indicating successive and increasing enlargements.
E. Lower edge of Liver in Cancer, Leukæmia and Adenoma.

of the liver, *abscess* of the liver, *waxy* degeneration with *gummata*, *cancer* of the *stomach*, and an *enlarged gall-bladder*.

In *hydatids* there are no gastric or severe constitutional symptoms. Cancer of the liver is rapid in its development, rarely exceeding one year in duration, while hydatids are of slow growth, lasting from four to eight years. Gastro-intestinal hemorrhages are common in cancer, and do not occur in hydatids. Pain is a prominent symptom in cancer; hydatid tumors are painless. In cancer the nodules are hard, tender, and firm; hydatid tumors are large, soft, smooth and elastic, and can be freely manipulated without pain. The peculiar hydatid fremitus is sometimes obtained by percussing a hydatid tumor. In hydatids (with an exploring trochar) a saline fluid containing the booklets of the echinococci may be withdrawn, which will decide the diagnosis.

In *waxy degeneration* of the liver, there is a history of syphilis, prolonged suppuration, or disease of bone; and in cancer an hereditary cancerous history, or the evidences of carcinoma elsewhere. The progress of waxy liver is slow; that of cancer is rapid. A waxy liver is painless, while pain in cancer is constant. In waxy liver the spleen is markedly enlarged; in cancer it is normal in size, unless it is the seat of cancer infiltration. Jaundice and ascites are rare in waxy degeneration, and frequent in cancer.

In *cancer of the stomach* gastric symptoms are urgent and appear much earlier than in cancer of the liver. In cancer of the stomach there is usually coffee-ground vomiting and cancer-cells in the ejected matter. In cancer of the stomach the pain and gastric symptoms are aggravated after ingestion of food, while in hepatic cancer the pain and gastric symptoms are constant. In cancer of the liver in thin subjects, immovable nodulated tumors may be felt by pressing up under the ribs; while in gastric cancer a single tumor which is movable, and changes its position as the stomach is full or empty, is usually felt. In hepatic cancer there is absolute dulness over the tumor; while in cancer of the stomach the percussion note has a peculiar tympanitic quality.

Cancer of the *right kidney*, *impaction* of *fæces*, and various alterations in the *size* of the healthy *liver* will not long confuse one if the symptoms and physical signs are carefully analyzed.

Prognosis.—Cancer of the liver is a *fatal* disease. The average duration is about one year. Medullary cancer runs its course in from two weeks to four months. The duration of all varieties will be influenced by the presence or absence of complications. Death may result from exhaustion, from the cancerous cachexia, dropsy, diarrhœa, dysentery and hemorrhages, or from peritonitis, pneumonia or pulmonary œdema.

Treatment.—All varieties of cancer of the liver are incurable, hence the absurdity of all the so-called curative measures. The diet should be nutritious, and care should be exercised not to overfeed this class of patients. Easily assimilated preparations of iron are often of service. Diarrhœa, if present, may be checked by such remedies as gallic acid, lead, and opium. The operation of paracentesis should be delayed as long as possible. In the advanced stage of the disease alcoholic stimulants are often necessary and beneficial. In the great majority of cases the principal office of the physician is to relieve pain, and morphia is our most reliable remedy for this purpose; it should be given in sufficient quantities to keep the patient comfortable.

GUMMY TUMOR OF THE LIVER.

This form of *new growth* is perhaps the most characteristic lesion of constitutional syphilis. Some writers group these tumors under the head of *syphilitic disease of the liver*. Those forms of perihepatitis, cirrhosis, and amyloid degeneration which are of evident syphilitic origin, I have preferred to describe in connection with the other corresponding forms, giving at the same time the few differences due to the syphilitic causation.

Morbid Anatomy.—The syphilitic nodules, gummy tumors, or "*gummata*" appear first as small masses of reddish-gray, pulpy, vascular tissue, scattered throughout the liver. Their point of origin I believe to be the wall of the capillaries,—the cells and nuclei of the "syphiloma" being due to the growth of the nuclei of the capillaries. The mass is composed of highly organized granulation-tissue, and is usually spherical in shape. The liver may be enlarged, or may retain its normal size, according to the extent of the waxy change which usually accompanies the development of the gummata. Diminution of its size is due to perihepatitis causing retraction. Under these circumstances the organ is lobulated, and deep, whitish furrows indent it, the result of cicatricial contractions. Fibroid nodules occasionally lie in these cicatrices. The bulgings are soft and smooth to the touch. The *capsule* is firm and opaque, and the seat of fibroid thickening, and is frequently bound to surrounding parts by adhesions.

On section there will be found scattered through the liver rounded masses varying in size from a pea to an orange, yellowish-white in color, either surrounded by congested parenchyma, or as isolated spots in the midst of an infiltrated homogeneous grayish-red mass. They may be encapsulated, a layer of translucent fibrous-tissue surrounding them and shading off imperceptibly into the surrounding liver-tissue. Brown spots in the tumors correspond to obstructed bile-ducts. The liver parenchyma, between the nodules, undergoes various changes: at one time it is congested and hypertrophied, at another it is atrophied and undergoes fatty degeneration. In well-marked cases there are two zones, an outer, red and fleshy, and an inner, dry, grayish and firm. Again, nothing may remain of a previous gumma but a shrivelled cicatrix.

A *microscopical* examination of a fully developed gummy tumor reveals three processes:—*first*, at the periphery, there is a vascular mass of granulation-tissue, embedded in which are cells bearing a striking resemblance to white blood globules, and some larger nucleated ones. *Secondly*, just beneath this zone is found a fibro-nucleated mass, the fibrillations being very dense and cicatricial. *Thirdly*, in the centre of the mass are found fat-granules and broken-down cells, with occasional traces of cholesterin, and sometimes faint evidences of fibrillar tissue. Cheesy and calcareous masses are also sometimes found in the centre of the gumma.

Etiology.—As has been stated, gummata are the most characteristic of the lesions of internal syphilis. They are met with under no other conditions.

Symptoms.—The subjective symptoms of hepatic gummata are few and inconstant. At a post-mortem, a liver may be found studded with gummy tumors, when no symptoms referable to the liver were present during life. There is generally a history of increasing debility, and a feeling of pressure, tightness, and dull pain in the region of the liver. Sometimes the pain is severe and localized, at other times it is dull and diffused over the whole hepatic region. The pain in one case is constant, in another intermittent. If jaundice exists, it is due to the pressure either of the gummata or of an enlarged lymphatic. The temperature is normal, and the pulse-rate is but slightly increased. Ascites may result from pressure on the portal vein, or from chronic peritonitis, which often complicates its development. Both jaundice and ascites are not present until the liver has become very much enlarged. The symptoms which are present in the advanced stage of this disease, such as diarrhœa, loss of appetite, vomiting, hemorrhoids, gastric and intestinal hemorrhage, are due rather to the accompanying hepatic degeneration than to the gummata.

Physical Signs.—*Palpation* may show the liver to be enlarged or normal in size ; a moderate increase in size is the rule. The organ has smooth lobules upon its surface between which run deep fissures. The lobulations are soft and elastic, never fluctuating.

Percussion.—The area of dulness is increased and its outline is irregular below the free border of the ribs. The area of spleen-dulness in the majority of cases is slightly increased.

Differential Diagnosis.—Gummata of the liver may be mistaken for *cancer*, and if the liver is diminished in size, for *syphilitic cirrhosis*. The differential diagnosis of both has been considered.

Prognosis.—Gummata of the liver rarely *directly* destroy life. The prognosis is unfavorable when ascites, gastro-intestinal hemorrhage, persistent diarrhœa, or a marked cachexia exists. Complicating diseases also influence the prognosis ; amyloid degeneration of the spleen and kidneys is a bad complication. The most frequent intercurrent lung diseases are pleurisy, pneumonia, pulmonary œdema, and chronic bronchitis. Death occurs from exhaustion due to the syphilitic marasmus, from diarrhœa, dysentery, and dropsy. Pneumonia and pulmonary œdema often cause it, and sometimes cholæmia, with its peculiar symptoms, ends in coma and death.

Treatment.—The treatment of this affection resolves itself into the treatment of syphilis. As it is a tertiary symptom, our main reliance is on large doses of the iodide of potassium combined with mercurial inunctions. With these iron and cod-liver oil should be constantly taken, and the patient should be placed under the best hygiene. The diet should be nutritious and non-stimulating. Opium combined with nitric acid will always control the diarrhœa if it becomes exhausting.

HYDATIDS OF THE LIVER.

Hydatid tumors are cysts due to the development in the liver of the embryos of the *tænia echinococcus ;* these embryos are called "echinococci," their development "hydatids" ; they are usually single, and for more than two or three to be present in the same liver is a phenomenal event.

Morbid Anatomy.—An ovum of tænia echinococcus, either during mastication or from the action of the digestive juices, has the envelope containing the echinococcus removed, and then by its hooklets it bores its way from the stomach or intestine into the liver. It there becomes encysted: the cyst consists of an external laminated cuticular layer and an internal parenchymatous lining. From the internal layer numerous little heads bud forth in the form of vesicles, and these, the "daughter vesicles," in turn bear a second crop, the "grand-daughter cells," the mother-sac meanwhile enlarging, partly from the increase in the number of the vesicles, and partly from its own secretion, which is clear and watery. As these successive generations of vesicles appear, broods of immature tænia ("scolices") in the form of a grayish granular layer, are developed *first* upon the internal surface of the mother sac and then upon that of the other cysts, in the order of age. While the younger vesicles cling to the parent-walls, the larger and older ones become detached, and float in the interior of the continually enlarging parent-sac. Proliferation of connective-tissue upon the exterior of the sac resulting from the inflammatory process excited by the pressure of a foreign body, develops a fibrous capsule closely connected with the adjacent liver parenchyma ; this is supplied with blood by the hepatic and portal capillaries.

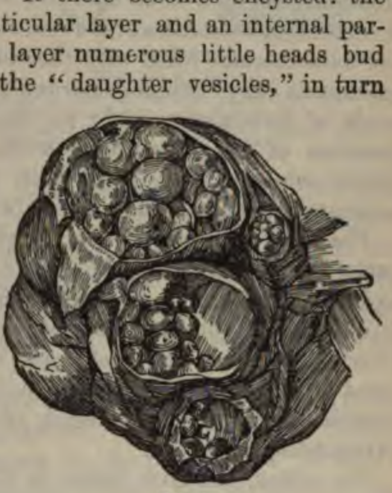

FIG. 85.

Multilocular Hydatid Cysts of the Omentum. (*After Bright.*)

During its enlargement the hydatid tumor loses its spherical shape and becomes indented. As it increases in size, the fibrous capsule becomes thickened, rough and cartilaginous ; sometimes it undergoes ossification. The echinococci may be destroyed by the bile which enters the cysts when the bile-ducts are opened, or by the inflammation which is established between the connective tissue capsule and the wall of the true sac, causing a grayish oily material of variable consistence to be developed. The clear fluid in the cavity of the hydatid becomes cloudy, then opaque, while all traces of the vesicles disappear, and at last only a few hooklets of the echinococci remain. This is a process of fatty degeneration. Sometimes the formation of vesicles is so rapid that their number is beyond all proportion to the fluid, and then they die and collapse, undergoing no degenerative process; again, when neither of these terminations is reached, the hydatid tumor may continue to increase in size until finally it bursts into the adjacent cavities.

The most frequent rupture is into the right pleural cavity. The hydatid tumor, by its pressure upon the diaphragm, causes it to become thin, and to rise upward, sometimes as high as the second rib. Finally the diaphragm ruptures and the sac is discharged into the pleural cavity, or when the pleural surfaces become adherent the tumor ruptures into the lung-tissue or bronchi. These tumors sometimes rupture into the peritoneal cavity, and peritonitis results, or the stomach or intestinal canal may communicate by a small aperture with the hydatid sac. A communication is sometimes established between the bile-ducts and the hydatid tumor, and the ducts become filled with the contents of the hydatid mass; the ductus communis may become obstructed by a large hydatid vesicle. Another mode of termination is by an intense inflammatory action, causing suppuration of the liver-tissue in the vicinity of a ruptured hydatid tumor, which consequently is filled with coagulated blood and pus; more rarely a gangrenous process may be established in it.

The liver is irregularly enlarged and displaced. The increase in size varies with that of the projecting cysts, which are sometimes large enough to fill the abdominal, and a portion of the thoracic, cavity. The bulging is globular if the cyst is simple and is situated in the right lobe of the liver. The tumor is elastic and often fluctuating. A uniform enlargement of the liver results from a centrally located hydatid. The *capsule* covering the cyst is thickened, and adhesions often bind the liver to the surrounding parts.

On section, the liver-tissue in the vicinity of the tumor is found compressed and atrophied, or congested and hypertrophied. The mother-sac is commonly the size of a fœtal head. The true cyst wall is a gelatinous, whitish, semi-transparent membrane, containing the hydatid fluid, floating in which are vesicles from the size of a millet-seed to that of an egg, and varying in number from hundreds to thousands. On the inner walls of the larger ones, and on that of the parent-sac, are younger vesicles about the size of a pin's head. On the inner side of the sac are also found patches of white granular matter. The cysts may be found filled with atrophied and shrivelled vesicles embedded in a débris consisting of fat-granules, cholesterin, hæmaglobin, and bile. Its consistency varies: sometimes it is liquid and watery, then semi-fluid, gelatinous, or like a thick paste; at other times only a few hooklets remain in this gray, putty-like mass. The cyst may contain blood or pus.

A *microscopical examination* shows the sac of the hydatid to be a gelatinous mass made up of concentric hyaline lamellæ. The scolices are from 1-75 to 1-225 of an inch in length; the head is furnished with four suckers and a proboscis, about which are *sickle-shaped hooklets* in number from twenty-five to fifty. The body is striped longitudinally and transversely, and has a groove between it and the head, which latter, being usually retracted into the body, causes the animal to look somewhat like an indented rubber ball, the hooks fringing the depression. The fluid is clear or slightly opalescent, it has a specific gravity of 1010 to 1015, is usually neutral in reaction, and is non-albuminous. It is chiefly water containing chloride of sodium.

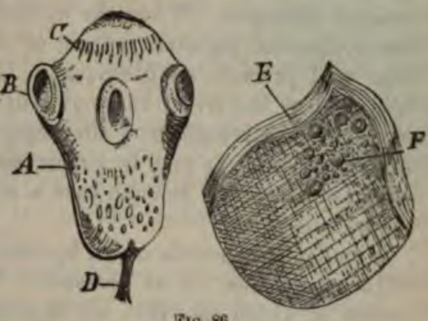

FIG. 86.
Hydatids of the Liver.
A. Head of echinococcus from an hydatid tumor.—B. Discs.—C. Hooklets.—D. Pedicle.
E. Fragment of capsule of hydatid tumor, showing its lamellæ.—F. Germs. × 200.

Etiology.—The essential cause of the development of hydatids is the entrance into the stomach, or intestines, of the tænia echinococcus. If they remain in the intestine they become tape-worms; when they pass into the liver they develop hydatids. Hydatids are chiefly met with between the ages of thirty and fifty. They are rare in childhood and old age. They are most common among the poor and filthy, and in cold climates. It is estimated that one out of every six of the inhabitants of Iceland has hydatids of the liver. Dogs, sheep, pigs, cats, and rats are subject to tapeworms, and as the ova of these parasites are discharged in the excrements of these animals, they can only gain entrance into the human stomach through polluted drinking-water, or the most filthy practices.

Symptoms.—If an hydatid tumor is deeply seated and of small size, it gives rise to no symptoms and cannot be recognized. A large hydatid tumor will cause sufficient functional disturbance by its pressure to be easily recognized. The patient may first see or feel a tumor in the region of the liver, and have a sense of weight and dragging in the right hypochondrium. Symptoms of pressure of the tumor on adjacent organs are the first, and often the only ones which attract attention. Dyspnœa, a dry hacking cough, and bronchial catarrh may result from the upward pressure of the tumor. When the heart is displaced by the tumor, there is palpitation; and when the stomach is encroached upon there is vomiting, dyspeptic symptoms and emaciation. When the portal vein or vena cava is pressed upon by the hydatid tumor, ascites, jaundice and hemorrhoids may result. When the hydatid compresses the bile duct, or when there is intercurrent catarrh of the ducts, or when they have become obstructed by the hydatid vesicles, jaundice sets in and absence of bile in the fæces is noted. A large vesicle may, in passing the duct, give every symptom of gall-stone colic, and thus be confounded with it.

When the pleura is perforated, the symptoms of acute pleurisy are developed, and in most cases the cavity is rapidly filled with pus containing hydatid vesicles. Peritonitis may result from spontaneous or traumatic rupture of an hydatid cyst. The opening into the stomach or intestines being usually very small, it is rarely attended either by peritonitis or secondary abscess; when a cyst is evacuated in this way the case usually terminates in recovery. When shreds of hydatid vesicles and echinococci are found in the urine, it indicates that the rupture has taken place into the urinary passages. When the hydatid tumor is to discharge itself through the abdominal parietes, redness of the skin, tenderness, pain, and fluctuation will precede its discharge. If, in a patient who is known to have hydatids of the liver, there is pain, elevation of pulse and temperature, extreme sensitiveness over the hepatic region with a peculiar friction sound on auscultation, it may be suspected that inflammation in and around the sac has occurred. In such case abscess may be excluded by the absence of rigors and sweats.

Finally, the growth of an hydatid of the liver is in rare cases attended with pain caused by its pressure. The fæces are normal unless jaundice exists, in which case they are firm and clay-colored. The urine is generally normal, but if pus or albumen is found in it, pyelitis exists as a result of the pressure of the tumor on the renal vein.

Physical Signs.—*Inspection* may show a distinct bulging in the right hypochondrium, which has the appearance of a globular elevation over the right or left lobe of the liver. The ribs often project, and respiratory movements on the right side are interfered with.

Palpation discovers an enlarged liver, elastic to the touch when the tumor is deeply seated; when it is superficial, fluctuation may be detected. The tumor is smooth, but if two, three or more cysts exist, the liver will have a lobulated outline below the free border of the ribs.

Percussion.—The normal area of hepatic dulness is increased in some one direction. When the tumor is superficial, the hydatid thrill or "fremitus" is elicited by firm percussion. This sign, peculiar to hydatids of the liver, is elicited in the following manner: place three fingers, slightly separated, firmly over the most prominent part of the tumor; give a sharp blow upon the middle one, and a vibration or fremitus will be communicated to the other two.

Differential Diagnosis.—Hydatids of the liver may be mistaken for *cancer, abscess, abdominal aneurism, enlarged gall-bladder, pleurisy,* rarely *multilocular hydatids,* and a *cyst* of the right *kidney.* In *abdominal aneurism* there will be severe and constant pain in the back; the tumor is soft, doughy, and compressible, has an "expansive" pulsation and is immovable, while an hydatid tumor moves up and down with the respiratory movements and fluctuates. A "bruit" synchronous with the heart and often double will be heard over an aneurism, while neither of these is ever present in hydatids. The femoral pulse will be altered in an abdominal aneurism, but normal in hydatids of the liver.

When a *pendulous hydatid* cyst is attached to the liver by a pedicle, we may readily mistake it for an *enlarged gall-bladder.* An enlarged gall-bladder is usually preceded by jaundice, biliary colic, or symptoms of catarrh of the ducts, while an hydatid has no such previous history. On palpation it will be found that an hydatid does not correspond exactly to the position of the gall-bladder. The gall-bladder is pear-shaped and evades manipulation or pressure, while an hydatid tumor is globular and readily manipulated.

When hydatids extend into the pleural cavity so as to be mistaken for *pleurisy*, the heart will be displaced much more than ever occurs in pleurisy. Percussion in pleurisy marks out a line of dulness which is transverse when the patient is erect, and which changes with his position, while in hydatids the upper limit of dulness is irregular and stationary, being lower near the median line of the body than toward the axilla. This is an important point. In hydatids the lower edge of the liver is below the free border of the ribs and rises and falls with the respiration; in pleurisy the liver occupies nearly its normal position and is stationary.

In a *cyst of the right kidney*, there is the history of a growth from below upward, while in hydatids the tumor grows from above downward. In cystic kidney the colon lies in front of the tumor, while in hydatids of the liver the colon is behind the tumor. An hydatid of the liver rises and falls with respiration, while a cyst of the kidney is motionless. In hydatids of the liver, an exploring needle will withdraw a non-albuminous, salty fluid, containing hooklets of the echinococci, while from a cyst of the kidney it will withdraw an albuminous fluid with chlorides and perhaps pus.

Prognosis.—Hydatids are dangerous in proportion to their size and the direction of their growth; if they cease to enlarge, they may be regarded as harmless. Their average duration is about four years. They have been known to exist twenty-five years. If they rupture into the pleura, lung, peritoneum, pericardium, or through the abdominal walls, the prognosis is unfavorable. When the discharge takes place into the intestines, stomach, or bronchi, the prognosis is favorable. *Death* occurs from exhaustion caused by the pressure of a very large hydatid, rarely from that caused by ascites through pressure on the vena cava. Suppuration of the cyst, or an abscess developed secondarily to phlebitis may induce fatal exhaustion. Any one of the pulmonary complications referred to may cause death. A fatal result has, in some few cases, followed hemorrhage from the sac through an external opening. Peritonitis, pericarditis, and uræmia are infrequent causes of death, and when the pulmonary artery is plugged, when the vena cava is opened, or when a large vesicle is lodged in a bronchus, asphyxia is the immediate cause of death.

Treatment.—Prophylactic measures consist in preventing the drinking-water from being contaminated by the evacuations of animals, and in not allowing dogs to feed upon the offal of sheep. Chloride of sodium and iodide of potassium have been proposed as internal remedies to destroy the echinococci. The chief solid ingredient of hydatid fluid is chloride of sodium, but no trace of iodide of potassium has ever been found in the fluid after the administration has been continued for months.

If the tumor is of large size, and is still increasing in size, *operative interference* is necessary. Select the point where the hydatid tumor is most prominent, and puncture with a fine aspirating needle. The dangers which have been feared in this procedure are peritonitis, and the entrance of air into the peritoneal cavity. Peritonitis may be avoided by pressing the parts about the puncture firmly against the tumor as the aspirating needle is withdrawn, so that no fluid can escape into the peritoneal cavity. All danger of the entrance of air is obviated if a small aspirating needle is used; all of the fluid should not be withdrawn from the cyst at the first aspiration. It is important to enjoin absolute rest after the operation for two or three days; febrile symptoms and pain will follow the withdrawal of the fluid, and the tumor will decrease in size; usually a second puncture will be required. It is not essential to wait for adhesions to form between the tumor and abdominal wall, though it is much safer if they exist.

Where simple puncture is not sufficient to destroy the echinococci, iodine or bile may be injected into the cavity of the sac. When the fluid withdrawn is pus, or when the symptoms are indicative of a suppurating cavity, it is best to establish adhesions by caustics. Vienna paste is to be preferred for this purpose, and the same precautions are to be exercised as in the opening of an hepatic abscess. Puncture of the cyst by insulated needles,—electrolysis,—has been claimed to be very successful in those cases where it has been resorted to, but it seems to me that it is the puncture, rather than the electric influence, which produced the favorable result claimed for it. Never hesitate to aspirate an hydatid tumor when it is well developed and elevated above the level of the abdominal walls; the nearer the cyst is to the surface, the better the result of the aspiration.

MULTILOCULAR HYDATIDS.

Multilocular hydatids in their *pathology* are similar to the ordinary hydatid, except that in the one case a cyst is formed, and in the other it is wanting.

Morbid Anatomy.—The liver is enlarged, the right lobe being out of all proportion to the rest of the organ; it is hard, and sometimes has a cartilaginous feel. There are large nodules on its surface, the tumors often being as large as a child's head. *The capsule* is thickened and opaque over the tumor, and adhesions may be formed with surrounding parts.

On section a tumor is usually found embedded in the right lobe, varying in size from that of an orange to a cocoanut, with a reticulated surface. The stroma is connective-tissue, dull-yellow in color, and the spaces are rounded, oval, or caudate, from the size of a pin's head to that of a pea, usually communicating with one another by small apertures, and containing a clear or semi-transparent gelatinous substance. The stroma generally contains some hepatic parenchyma, which, at the periphery of the mass, is dark-colored from pigment deposit, and nearer the centre is in a state of partial or complete fatty degeneration. In the centre of the mass is usually found a large spot of suppuration, varying in size, or a cavity filled with a light-brown fluid or a greenish fetid pus. This cavity is well defined, having a wall whose lining membrane is sacculated at points, with openings into neighboring cavities. This membrane frequently has clusters of hydatid vesicles upon it. The surface cavities are much smaller than the central one, and are strung along like "strings of pearls," compressing and occluding the branches of all the vessels of the liver at the point whence the growth develops. The rest of the liver is congested or hypertrophied.

Microscopically, the gelatinous contents in one of the alveoli are found to be made up of the laminated structure of the vesicle and containing echinococci with their circlets of hooklets ; it is rare, however, to find perfect "scolices." The cyst-wall may exhibit calcareous, fatty, or pigmentary degeneration. Between the cysts small globules, consisting of calcareous matter, are sometimes found, and also granular and crystalline hæmatoidin. The *spleen* is usually enlarged.

Etiology.—The etiology of multilocular hydatids is the same as that of a single hydatid tumor. When the ova of the tænia reach the liver, the question arises: what is it that causes the peculiar alveolar or colloid arrangement of the vesicles ? This has been variously explained : some claim that when the embryo enters either the lymphatics, blood-vessels, or bile-ducts, the growth extends along the channels of either system, since it cannot extend concentrically because of the exterior pressure. I am inclined to agree with those who think that when a cyst-wall is developed around the embryo and no connective-tissue encapsulates the cyst, it can grow in all directions. Their development is yet obscure.

Symptoms.—The insidious approach and slow progression which mark the course of single hydatid tumors are present here. The effects of pressure may be shown by vomiting and dyspepsia when the stomach is encroached upon ; constipation and difficulty in defecating when the intestines are pressed upon ; and ascites, œdema, hemorrhoids, and enlarged spleen, when the portal trunk or vena cava is compressed. There are no subjective signs. When suppuration occurs there will be a slight rise in temperature, increased frequency in the pulse, and other symptoms which attend the formation of pus in the liver. Gastro-intestinal hemorrhages, and hemorrhages from mucous surfaces rarely occur. When the bile-ducts are involved, jaundice is the first, or an early symptom ; the jaundice gradually deepens and is persistent. The fæces are clay-colored.

Physical Signs.—*Palpation* shows the liver early enlarged, having slight elevations, with a smooth feel. Later it is hard, resistant, nodular, and uneven. The tumors are confined to the right lobe, or, at least, are best marked there.

Percussion.—The area of hepatic dulness, in the region of the right lobe especially, is increased, and the spleen is enlarged with multilocular hydatids. There is neither fluctuation nor the "hydatid thrill."

Differential Diagnosis.—When the bile-ducts are involved a diagnosis may be made by exclusion ; in other cases the affection is liable to be confounded with *cancer, cirrhosis,* and *nodular* (syphilitic) *waxy* liver.

In *cancer of the liver* there is a history of a gradually developing cachexia and lancinating, almost constant, pain, with evidences of cancer elsewhere, or an hereditary tendency ; while in multilocular hydatids there are no constitutional symptoms and *no pain,* the hepatic region merely being tender. The duration of cancer is rarely more than a year, while the development of multilocular hydatids is protracted to two or three years. A cancer nodule, on palpation, is usually umbilicated and soft at the centre; while a multilocular hydatid is smooth and elastic. An exploring trochar will remove all doubt.

In *cirrhosis* of the liver there is a history either of alcohol drinking, rheumatism, gout, or syphilis; in multilocular hydatids no such history necessarily exists. In cirrhosis the liver is smaller than normal and hob-nailed; in multilocular hydatids it is enlarged and the nodules are of a larger size. Ascites is common in cirrhosis; while it is rare in hydatids.

In a *nodular waxy liver* there is a well-marked history of syphilis; there is a complicating diarrhœa, albuminuria, and the characteristic waxy cachexia, which are absent in multilocular hydatids.

An *hydatid* tumor can only be diagnosticated from multilocular hydatids by the exploring trochar. The fluid in multilocular hydatids contains pus in varying quantities, a few hooklets of the echinococci, and *numerous small* vesicles; while that from an ordinary hydatid is nearly pure water, containing the hooklets and a *few* vesicles. In many instances the hydatid thrill is present in a single hydatid tumor, but *never* present in multilocular hydatids.

Prognosis.—In those cases in which the jaundice is intense, the disease may run its course in six months; in other cases, the course is as prolonged as in single hydatids. Peritonitis is a frequent complication, and amyloid degeneration of the liver may also occur with it. Death results from the exhaustion due to the suppuration or from the complicating peritonitis.

Treatment.—There is no medicinal treatment which can in any way affect the course of this disease; and operative interference has thus far proved unsuccessful. This class of patients must be sustained during the exhaustion of the supervening suppuration. The pain, if severe, may be relieved by hypodermatics of morphia.

TUBERCULOSIS OF THE LIVER.

Tubercle of the liver is always secondary to tubercle elsewhere. It is probably more common than is usually supposed, from the fact that hepatic tubercle is always *microscopic.*

Morbid Anatomy.—The liver is slightly but uniformly enlarged in size. On close inspection the surface is seen to be irregularly elevated and depressed, and looks and feels, in this respect, like the surface of an orange.

On section the liver cuts hard, the parenchyma being tense and tough. The tissue is pale and yellow, resembling a fatty liver. The bile-ducts at points are expanded, the walls being thinned. They contain a turbid fluid mainly composed of mucus and bile. There are also small cavities filled with pus and bile. When the tubercle has undergone retrograde metamorphosis, small gray masses the size of a pin's head are seen, "yellow tubercle," or larger yellow masses the size of a pea; these changes are usually best marked just beneath the capsule.

A *microscopic examination* shows miliary tubercles scattered between the lobules. When, as a result of obliteration of blood-vessels, tubercles undergo fatty degeneration, the so-called "yellow tubercle" is the product.

Etiology.—Hepatic tubercle occurs as part of acute miliary tuberculosis, and is secondary to tubercle in the lungs, peritoneum, spleen, and lymphatics.

Symptoms.—There are no symptoms indicative of hepatic tuberculosis, independent of those of general tuberculosis.[1]

JAUNDICE.

Jaundice is a yellow discoloration of the skin, due to the presence of bile or blood pigment. There are two varieties, ***hepatogenous*** or obstructive jaundice, and ***hematogenous*** or non-obstructive.

Hepatogenous jaundice is the more common variety, and is caused by the absorption of bile, its *passage into the ductus communis* or *intestine* being prevented by some mechanical obstruction.

Hematogenous jaundice results, probably, from a *change* in the blood, whereby its coloring matter is set free in excess.

Morbid Anatomy of hepatogenous jaundice. In a normal state, the liver-cells are constantly manufacturing bile, which flows along the bile-ducts into the ductus communis. The cause of its outward flow is the *vis a tergo*,—the secretion of the bile in the hepatic cells,—for there are no muscular fibres except in the larger bile-ducts; the respiratory movements also assist slightly in its outward flow. When from any causes the bile cannot enter the common duct or the duodenum, the small ducts and then the hepatic cells become overfull and distended. In consequence of this increased pressure, bile passes through the wall of the smaller ducts into the blood-vessels and lymph channels. If the normal tension of the capillary system in the liver is diminished, then the passage of bile through the walls of the vessels is favored and jaundice results. Bile pigment with serum exudes and stains the tissues, even the bones, the teeth and pathological new formations. In both hepatogenous and hematogenous jaundice, the staining occurs in the same way.

Hematogenous Jaundice.—In health the bile pigment is formed within the liver, by transformation of the coloring matter of the blood, and after it has been poured into the intestine, it is partly absorbed by the blood and appears, after another change, as one of the coloring matters of the urine. Under abnormal conditions, and as the result of processes that are not fully understood, coloring matter is either set free in excess or is not excreted with the bile, and is then deposited in the tissues producing jaundice. As this variety of jaundice is thought to have its origin in morbid conditions of the blood, it is called hematogenous.

The anatomical lesions which are associated with hematogenous jaundice have already been considered in connection with the history of the different hepatic affections in which it occurs.

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[1] Lymphatic formations, simple cysts, dermoid cysts, erectile cavernous tumors, and benign fibrous growths occur in the liver, but are only of pathological interest.

Etiology.—The causes of *hepatogenous* jaundice may be included under three heads:

I. Those which obstruct the larger hepatic *ducts*.
II. Those which obstruct the hepatic *radicles*.
III. Those which diminish *capillary tension*.

Those obstructions of the larger hepatic duct which have their seat *within* the duct are:

(1) Inflammations of, or inflammatory exudations from, the lining membrane of the duct, that which accompanies duodenal catarrh being the most frequent. (2) Biliary calculi. (3) Inspissated bile and mucus. (4) Hydatid vesicles. (5) Distomata. (6) Foreign bodies from the intestinal canal, such as stones of fruits and round worms. (7) Congenital occlusion, or plugging of the duct. (8) Cicatrices from ulcers on the mucous membrane of the duct. (9) Carcinomatous growths from the lining membrane of the ducts.

The causes which obstruct the duct by *external pressure*, are·

(1) Contraction from perihepatitis, or from inflammation of the hepatico-duodenal ligament. (2) Tumors of the pyloric extremity of the stomach, of the head of the pancreas, and of the kidney. (3) Pressure from a pregnant uterus, from ovarian and fibroid tumors, from omental tumors, and from large impaction of fæces. (4) Enlarged lymphatic glands in the transverse fissure from waxy, cancerous, or tubercular change, abdominal aneurism, and the new tissue in hypertrophic cirrhosis of the liver.

Slight hepatogenous jaundice may be caused by compression or obliteration of the hepatic *radicles*, such as occurs in cirrhosis and the other atrophies of the liver, in active and passive hyperæmia, in hydatid tumors and multilocular hydatids, in cancerous and syphilitic tumors, in abscess of the liver, in adhesive pylephlebitis, and perhaps in acute yellow atrophy.

Finally, the bile may be prevented from *entering* the intestine in its normal amount when *capillary tension* is *diminished*. This may occur in severe right diaphragmatic pleurisy, in perihepatitis, in thrombosis of the trunk or of the larger branches of the vena portæ, and in exhausting hemorrhage from the radicles of the portal vein.

The causes of *hematogenous jaundice* are fevers, especially yellow, typhus, typhoid, and the malarial fevers. It is often an attendant of pyæmia, puerperal fever, septicæmia, and suppurative pylephlebitis. The poison of snake-bites, phosphorus, mercury, copper, antimony, and the excessive use of ether and chloroform may cause it. Pneumonia, probably by its action on respiration, and ulcerative endocarditis induce it; it may follow a fright, a fit of anger, great anxiety, or cerebral concussion. A long continued hepatogenous jaundice may lead to a hematogenous jaundice; and it is yet undecided whether the icterus in yellow atrophy belongs to the first or second named group.

Differential Diagnosis.—*Hematogenous* jaundice accompanies acute infectious fevers and other conditions of blood poison, while *hepatogenous* jaundice can be traced to some mechanical interference with the outflow of the bile. The yellow staining is slight in hematogenous jaundice; while the discolorization in hepatogenous jaundice is more intense and may appear suddenly without constitutional disturbances. A feeble and irregular heart-action, a small pulse, and a tendency to hemorrhages attend hematogenous jaundice; while an unimpaired heart-action, a slow pulse, and a low temperature mark the development of hepatogenous jaundice. There is great itching of the surface in hepatogenous jaundice which is absent in the hematogenous variety. The fæces are dark in hematogenous jaundice, and white or clay-colored in hepatogenous. The urine is albuminous, contains a small amount of bile pigment, and deposits a sediment of uric acid in the hematogenous variety, while it is rarely albuminous in hepatogenous jaundice and contains bile-pigment in considerable amount, the quantity varying with the intensity of the jaundice.

DISEASES OF THE GALL-BLADDER AND GALL-DUCTS

will be considered under the following heads:

I. *Catarrhal Inflammation of the Biliary Passages.*
II. *Exudative Inflammation of the Biliary Passages (croupous or diphtheritic).*
III. *Cancer of the Gall-Bladder.*
IV. *Enlargement of the Gall-Bladder.*
V. *Gall Stones.*

CATARRH OF THE BILE-DUCTS.

Morbid Anatomy.—Catarrhal inflammation of the mucous membrane of the larger bile-ducts, the ductus communis, and the gall-bladder is similar to that of other mucous surfaces. There is hyperæmia followed by an abnormal secretion of mucus and muco-pus which more or less obstructs the outflow of bile. The catarrhal process usually begins in the duodenum and extends inward, and in severe cases may be so rapid that pus will be the product of the inflammation, in which case the deeper tissues are involved and numerous little ulcers may form, and when the duct is perforated by them, cavities of varying sizes, resembling small abscesses, result. When the catarrh becomes chronic the deeper tissues are infiltrated, causing thickening and induration of the ducts from the consequent obstruction to the exit of the bile. Dilatations occur at points along the bile ducts; these dilatations often become very large and occasionally form cysts; at other times the alternate dilatations and constrictions give the appearance of a string of beads. The lymphatics often become involved, and their enlargement gives a nodular appearance to the mucous membrane. Ulcerative processes are more frequent in chronic catarrh of the bile passages than in acute. The *liver* is uniformly enlarged and its margins are firm and sharp.

On section, its substance presents a mottled appearance, resembling a nutmeg, and varies in color from a deep yellow to an olive green. The color is deeper at the centre of a lobule and shades off toward its periphery. The gall-ducts commonly have their mucous membrane pale and covered with a thick, purulent mucus; and plugs of mucus and epithelial débris are found in them, most frequently near or at the opening of the duct into the duodenum. The gall-bladder is enlarged, and the cystic and common ducts often attain immense size; in one case this diameter reached an inch and a half.

In *chronic catarrh* the liver is normal or diminished in size, and is soft, flabby and shrivelled.

On section it is greenish-black in color, the hepatic ducts are dilated, forming cysts, and little points of ulceration are formed on the mucous surface of the duct, often extending into the adjacent parenchyma which is atrophied. The ramifications of the vena portæ are compressed by the ducts, and thickened bile may cause these ducts to present the appearance of a dark brown tube. The *gall-bladder* is enlarged in size, and sometimes there are spots of ulceration upon its walls which may also undergo calcareous changes.

Etiology.—The most frequent cause of biliary catarrh is extension of a gastro-duodenal catarrh. Most of the structural diseases of the liver may lead to or be attended by catarrhal inflammation of the bile ducts. Thoracic disease where the venous return is impeded (as in cardiac valvular lesions and emphysema) may cause catarrh of the biliary passages. General blood diseases, syphilis and pyæmia prominently, and mineral poisons, phosphorus, and perhaps arsenic, cause it. A gouty diathesis causes or predisposes to a catarrh of the biliary passages, just as it does to catarrhal inflammations of the mucous membranes elsewhere in the body—bronchitis, for instance. Foreign bodies, as calculi and parasites, in the bile passages may cause biliary catarrh. Exposure to cold and an altered condition of the bile may induce it.

Symptoms.—The subjective symptoms of biliary catarrh are at first obscure. It is usually preceded by the symptoms of gastro-duodenal catarrh, and hence for a few days there will be loss of appetite, furred tongue, flatulence, nausea and vomiting. There is also some pain and tenderness in the epigastrium, and in most cases the temperature will be slightly raised, and the pulse accelerated. The bowels are constipated, unless it is accompanied by extensive intestinal catarrh, when diarrhœa will be present. The fæcal discharges are of a light clay color and contain no bile. The urine is of a dark green color, and contains bile pigment. The liver is enlarged and tender, especially over the region of the gall-bladder. The absence of bile from the intestine favors gaseous distention of the bowel. The sclerotic becomes yellow, and gradually the entire surface assumes a yellow hue. The *temperature* falls to *normal* and the pulse is slowed. As the jaundice deepens, there is a noticeable loss of strength, the patient becomes apathetic and disposed to sleep during the day. There is headache, vertigo, and great depression of spirits; itching of the surface becomes exceedingly troublesome.

All these symptoms remit, the appetite returns, and the fæces and urine return to their normal color; or the catarrh becomes *chronic* and continues for months, the jaundice deepening, exhaustion and emaciation becoming extreme. Then gastric and intestinal hemorrhages frequently occur, and ascites may be followed by general anasarca ; coma closes the scene. The last stage of chronic catarrh is accompanied by evidences of atrophy of the liver.

Physical Signs.—*Inspection* reveals a jaundiced condition of the skin and conjunctivæ, and perhaps a bulging in the right hypochondrium.

Palpation discovers an enlarged, smooth and tender liver. The gall-bladder is enlarged, and sometimes there is a pear-shaped fluctuating tumor at its anterior margin. The gall-bladder is tender on firm pressure. Late in chronic catarrh the liver is diminished in size.

Percussion shows a uniform increase in the area of hepatic dulness, which, however, in chronic disease may be normal or diminished.

Differential Diagnosis.—This condition may be mistaken for *suppurative pylephlebitis* and *exudative inflammations of the ducts*. The former has already been considered, the latter will be considered under exudative inflammations.

Prognosis.—This is good ; catarrh of the bile ducts is not a dangerous disease. The jaundice usually continues from three to five weeks, but sometimes it continues for months. The prognosis is rendered unfavorable when oft-repeated biliary catarrhs lead to permanent closure of the ducts and atrophy of the liver. Catarrh of the bile-ducts may be complicated by peritonitis, pleurisy, pneumonia, dysentery, suppurative hepatitis, or acholia. Death then results from exhaustion, from faulty nutrition, or dropsy, from intercurrent diseases, rupture of the ducts, or with brain symptoms—"acholia."

Treatment.—It should be remembered that the jaundice is only a symptom, and requires no treatment. The treatment of this catarrh is mostly symptomatic ; it is usually self-limiting and will subside without remedial measures. If the hepatic pain is severe, leeches followed by an anodyne poultice over the points of greatest tenderness will usually relieve it. When the bowels are constipated, "blue-pill," or a saline purgative is first demanded, after which old cider or tamarinds will regulate the bowels for the remainder of the attack ; nitro-muriatic acid acts favorably in most cases. If there is diarrhœa, ipecacuanha or Dover's powder will readily control it. When the urinary secretion is much diminished the salts of potash in combination with diaphoretics may be administered. The diet throughout should contain no carbo-hydrates ; the food should consist principally of lean or prepared meats, vegetables, and skimmed milk.

When there is a gouty diathesis, colchicum and iodide of potassium are often of service. In a syphilitic diathesis, chloride of ammonium and the bi-chloride of mercury are indicated. Emetics rather aggravate the gastric intestinal catarrh than cause the expulsion of a hypothetical plug in the common duct, and should not be administered. Finally, if the hepatic parenchyma become involved, a tonic and diuretic plan, similar to that adopted in cirrhosis, may be adopted. The use of mineral waters must be determined by the influence which they exert on each patient ; in some cases increased appetite results, while in others they seem to hasten the wasting process.

EXUDATIVE INFLAMMATION OF BILIARY PASSAGES.

Under the head of *exudative inflammation* of the biliary passages I include both a *croupous* and a *diphtheritic* process. Both are rare and seldom recognizable during life.

Morbid Anatomy.—The commencement of croupous inflammation is the same as catarrh; but the inflammatory product is fibrinous.

In diphtheritic inflammation, the deeper tissues of the walls of the gall-bladder and bile-ducts are involved, and large gray sloughs, more firmly adherent than in croupous inflammation, are formed upon their walls. The liver is usually enlarged.

On section the ducts within the liver are seen clogged with inspissated bile, and occasionally there are abscesses. When constriction and occlusion of the ducts exist, they become dilated behind the narrowed portion, and resemble cysts, containing a pale yellow fluid with loose coagula floating in it. The gall-bladder is sometimes filled with a gray-white liquid, neutral, albuminous, and sometimes containing leucin; at other times the liquid is purulent, or thick and dark like tar. On the mucous membrane of the gall-bladder and common duct is a yellowish-white fibrinous layer, varying in thickness and tenacity, having all the anatomical characteristics of a diphtheritic exudation. The walls of the gall-bladder and larger ducts are thickened and sometimes ulcerated. The ulceration may lead to perforation and fistulous openings. Adhesions sometimes bind the gall-bladder to the surrounding parts. If the diphtheritic process extends to the venous coats pylephlebitis may result; sometimes the bile-ducts open into branches of the vena portæ.

Etiology.—These inflammations occur with typhus and typhoid fevers, cholera, diphtheria, pyæmia, septicæmia, bilious fever, and from the irritation produced by biliary calculi.

Symptoms.—The first symptom of exudative inflammation of the biliary passages is a sense of constriction in the right hypochondrium. This is soon followed by pain, increased by pressure in the region of the gall-bladder, and vomiting. There are active febrile symptoms, but these are usually not marked. If ulceration of the ducts or implication of the branches of the portal vein occurs, then chills, sweats, and the other symptoms of pyæmic abscesses of the liver result, or the symptoms of pylephlebitis are developed. When an opening into the peritoneal cavity occurs, rapidly fatal peritonitis is the result. If there is no obstruction to the outflow of bile, neither jaundice nor alteration in the color of the stools will be present.

Physical Signs.—*Inspection* may show a slight elevation of the free border of the ribs.

Palpation discovers a pear-shaped, tender, movable tumor at the normal site of the gall-bladder. Slight pressure over it gives pain.

Percussion.—The area of liver dulness is normal or slightly increased; over the enlarged gall-bladder the percussion note is dull and somewhat tympanitic in character.

Differential Diagnosis.—Exudative inflammation of the bile-ducts may be mistaken for *simple biliary catarrh*. The points which will aid in a diagnosis are the occurrence of intense pain, active febrile symptoms, and a careful study of the etiology of each case.

Prognosis.—This is determined by the disease which it accompanies. It usually terminates in death.

Treatment.—Absolute rest is important. To relieve the pain leeches may be applied over the tumor, followed by poultices and, later, by counter-irritation. The diet and saline purgatives should be the same as in simple catarrh, unless the primary disease contraindicates their use. If symptoms of pus formation are present, quinine may be given in large doses and tonics are indicated. If the tumor becomes large, so that there is danger of its rupture, it may be aspirated, the same rules being observed as in hydatids and abscess.

CANCER OF THE GALL-BLADDER.

Cancer of the gall-bladder is usually associated with cancer of the liver substance, and is often the primary seat of the development of scirrhus or medullary cancer of the liver.

Morbid Anatomy.—The gall-bladder is enlarged, nodular and adherent to the surrounding parts; sometimes there are spots of ulceration on its surface, and there may be fistulæ from the gall-bladder to the intestine. *On section* its wall is found thickened, and the cavity sometimes filled with a cancerous mass in which are embedded numerous concretions.

Etiology.—This is the same as that of cancer of the liver. It is often secondary to cancer of the stomach. Concretions are so often found that some have ascribed its development to gall-stones.

Symptoms.—The subjective symptoms are few: none are constant except the gastric derangement and the paroxysms of lancinating pain; vomiting is common and severe, because of the pressure of the tumor on the pylorus. Jaundice may be present when the common duct is involved. While the tumors often increase very rapidly, the cancerous cachexia and emaciation are slow in their development. In some cases the symptoms undergo marked exacerbations and remissions. Swelling of the glands in the inguinal and axillary regions may occur.

Physical Signs.—*Palpation* will discover over the site of the gall-bladder a hard, nodular and immovable tumor. It is tender, and sometimes fluctuates at the centre.

Percussion shows an increase in the area of hepatic dulness below the free border of the ribs.

ENLARGED GALL-BLADDER.

Dropsy of the gall-bladder is a term used to include those cases where, on account of some obstruction, bile is prevented from entering the natural reservoir, and an increased secretion from its mucous surface leads to its distention.

Morbid Anatomy.—The gall-bladder is found enlarged, sometimes reaching the size of a cocoa-nut. The walls are thickened, at some parts more than at others, and occasionally sacculations render its outlines uneven. The cystic wall is often tense, and now and then plates of calcareous matter are found upon it. On opening it there may be a discharge of gas from its interior, but more commonly a curdy white fluid fills its cavity. This fluid contains whitish flakes of albuminous matter resembling synovial fluid; it may contain bile, and then it is dark and viscid. On close examination the mucous surface resembles a serous membrane, and the muscular fibres of its wall are attenuated and wide apart. Later on, the fluid contents of the cavity may disappear, and only a mass of pultaceous matter remains. When the obstruction has been near the opening into the duodenum, the ductus communis and the cystic duct are dilated and their walls thickened.

Etiology.—*Hydrops cystidis fellæ,* as it is sometimes called, may be caused by a catarrhal, croupous, or diphtheritic inflammation of the cystic duct, which obstructs the passage of bile into the intestine. Plugging of the common or cystic duct, or of the neck of the gall-bladder, by a calculus, may cause dropsy of the gall-bladder. Multilocular hydatids or hydatid cysts may plug the cystic duct and induce it. Pressure by tumors outside of the duct, as enlarged glands in chronic peritonitis, aneurisms, impacted fæces, and cancerous growths of the adjacent parts, occasionally leads to it.

Symptoms.—When the *cystic duct* alone is pressed upon or in some way plugged, there are few subjective symptoms. The patient may notice a bulging in the hepatic region, which steadily increases, and is accompanied by pain, nausea, vomiting, loss of appetite, and constipation. But the color of the skin, urine, and fæces exhibits no change. If a calculus is the cause of the obstruction, there is usually a history of "bilious colic," and if abdominal tumors press upon the cystic duct there will be the physical evidences of their existence.

Physical Signs.—*Inspection* may reveal a globular tumor near the rectus muscle, at the free border of the ribs. *Palpation* discovers at the normal site of the gall-bladder a pear-shaped, extremely movable tumor, which is elastic and rarely fluctuating. When the ductus communis is obstructed, jaundice is a prominent symptom, and the other symptoms which have been described under "catarrh of the bile-ducts" are present. Occasionally the tumor suddenly disappears, the stools become dark, and the skin regains its normal color. This denotes that the obstruction, which is then commonly a calculus, has been temporarily removed. When external openings are formed, or rupture into the peritoneal cavity occurs, there are, in the latter case, evidences of a rapidly developed peritonitis, and, in the former, a remission of symptoms with a biliary fistula discharging externally.

Differential Diagnosis.—Dropsy of the gall-bladder may be mistaken for *abscess, hydatids,* and *medullary cancer* of the liver. In *medullary cancer,* there is, in nine-tenths of the cases, an hereditary predisposition or a history of cancer of the stomach or heart; while in dropsy of the gall-bladder we get a history of previous biliary catarrh, or of the passage of gall-stones

In cancer, the constitutional symptoms and cachexia are marked, while persistent gastric symptoms, ascites and hemorrhages from mucous surfaces are absent in enlarged gall-bladder. Cancer growths are slow, and precede jaundice if it exists, while a gall-bladder enlarges rapidly, and follows jaundice. Palpation, in cancer, discovers a nodular, uneven, *immovable* mass below the free border of the ribs. An enlarged gall-bladder gives rise to a smooth, pear-shaped, elastic, or fluctuating tumor, which is movable and projects below the free border of the ribs in the direction of the gall-bladder.

Prognosis.—This varies with the cause. When inflammatory products or gall-stones induce the dilatation, it is better than when it is due to external pressure ; it is always attended with more or less danger.

Treatment.—The treatment, when it is the result of catarrhal inflammation of the ducts, has already been considered. When it is due to the presence of gall-stones, the treatment appropriate to such conditions is indicated. If the enlargement is very great and shows no indications of becoming stationary or diminishing in size, aspiration should be practised.

GALL-STONES.

When bile is retained in the gall-bladder for a long time it decomposes, and the cholate of soda and other bile salts, with cholesterin, globules of bile-resin, and granules are precipitated. These materials combine to form concretions, which are called *biliary calculi.* Catarrh of the gall-bladder always accompanies this retention and decomposition of bile.

Morbid Anatomy.—The *number* of gall-stones varies : single calculi are rare ; eight thousand were found in one case. Their usual number is about thirty. Their *size* varies from that of a pin's head to that of a goose egg. In *shape* they are originally spherical, ovoid, or pear-shaped ; but when there are many and they lie in contact with one another for a long time, they have numerous facets developed on their surface; six, or even twelve are sometimes found on a single calculus. Warty or "mulberry" calculi are occasionally met with ; solid or hollow casts of the larger bile ducts, and those which resemble rhomboidal crystals, and the star-like calculi with blunt points are rare forms of gall-stones. These calculi are commonly of a light-brown or greenish-yellow *color ;* they may be white, green, blue, red, or black. The *specific gravity* of fresh calculi is about 1.02, and it may reach 1 09, so that they will not float in water. In most cases a fresh biliary calculus can be

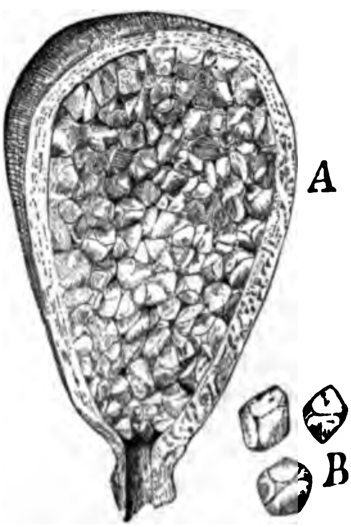

FIG. 87.

Sketch of a gall-bladder filled with biliary calculi. This bladder contained 260 gall-stones. At B are single calculi showing facets.

crushed between the fingers. Gall-stones may form in the smallest radicle of the hepatic duct.

On section a biliary calculus will rarely be found *homogeneous* throughout. Its substance, if it breaks down like clay, consists of cholesterin and lime. If it has a saponaceous fracture, it consists of bile-resin and cholesterin. The *ingredients* of biliary calculi are cholesterin, the coloring matter of the bile, bile-resin, lime salts, mucus, epithelium, biliary acids, margarin and traces of iron. A gall-stone usually has a nucleus, an external crust, and an intermediate portion. The *nucleus* may be formed of crystals of cholesterin, cholate of lime, mucus, a distoma, blood-clot, round worm or foreign body. Most nuclei are formed of casts of the hepatic ducts. Sometimes small calculi form the nuclei of larger ones, and in very rare instances multiple nuclei are observed. The *external crust* varies in thickness at different points, and is distinguished from the intermediate portion by its color; it is commonly composed of cholesterin, and its color is due to a mixture of cholesterin and biliary pigment; carbonate of lime gives a rough, whitish crust. The *intermediate structure* usually consists of crystalline radiations of cholesterin, which substance forms about *eighty per cent.* of all gall-stones. In this radiation can be seen evidences of a lamellar deposit, and sometimes, when there is no radiation, the layers are concentric, like those of an onion. Again, light layers of cholesterin alternate with deeper ones of pigment; gall-stones are rarely found to undergo a process of erosion or disintegration.

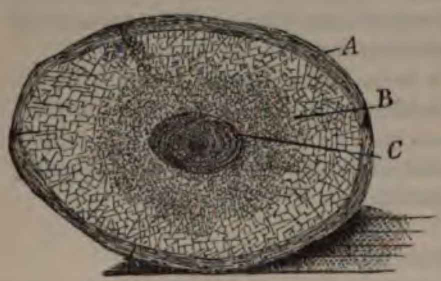

FIG. 88.

Section of a large Gall-stone, showing successive layers. A. External crust.—B. Intermediate portion.—C. Nucleus. Natural size.

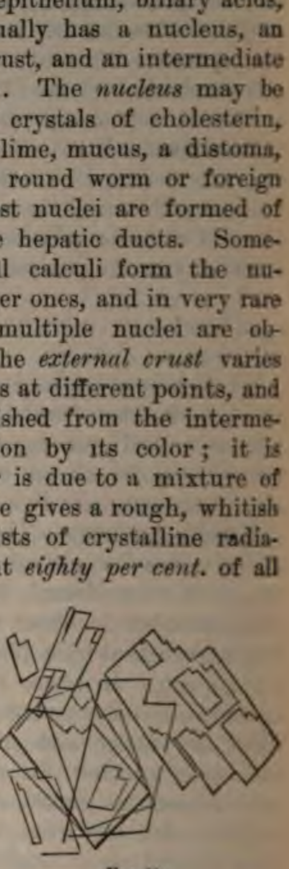

FIG. 89.

Crystals of cholesterin from gall-stones, showing the characteristic plates, with one corner wanting. × 200.

The *gall-bladder* may be normal, or enlarged and sacculated, and is often adherent to the intestine, abdominal wall, and adjacent organs. Its walls are thickened, and there are evidences of a local or general catarrh; late in the disease there may be fibroid contraction and calcareous degeneration in the cystic walls. Ulceration of the walls is frequently found in a bladder distended with calculi. When a gall-stone becomes impacted near the entrance of the ductus communis into the duodenum, the duct may become enormously dilated, and have its walls thickened, hypertrophied, or calcareously degenerated.

When the ulcerative process extends through the walls of the gall-bladder or of the larger ducts, we may have openings externally through the abdominal walls, usually about the umbilicus, called "biliary fistulæ."

These fistulous openings may lead from the gall-bladder or ductus communis to the duodenum, stomach, colon, right ureter, trunk of vena portæ, pleura, or vagina. When calculi are found in the smaller ducts, they may excite abscess of the liver, local fatty degeneration, inflammation of the ducts or pylephlebitis. Either by rupture into the cavity, or by extension of inflammation, peritonitis may be caused by the presence of gall-stones. They may also excite ulceration and gangrene of the intestines, and there are rare cases where gall-stones, having escaped into the intestines, have caused death by intestinal obstruction.

Etiology.—Gall-stones may be formed at any period of life, but are most frequent after thirty-five. A sedentary, physically inactive life is a great factor in their etiology, and I regard the greater prevalence of calculi in women than in men as due to their less active mode of life. Those who have to pass the greater part of their lives in bed, and prisoners who are confined in cells for a long time, are especially liable to the formation of gall-stones. A diet over-rich in fats, animal food, or alcoholic beverages, predisposes to the formation of biliary calculi. Cancerous growths in the liver and gall-bladder, catarrh of the gall-bladder, and in fact any morbid condition interfering with the excretion of bile and favoring its retention in the gall-bladder, predispose to the development of calculi. I have been able in a few cases to make out an hereditary predisposition to the formation of gall-stones. The menstrual epoch seems to have some peculiar influence upon their formation.

Symptoms.—Small gall-stones,—"gravel,"—in the *hepatic ducts* may cause hepatic congestion, but without enlargement of the liver, and give rise to dull pain, a sense of weight and constriction in the right hypochondrium, with nausea and the other symptoms of gastric disturbance. Jaundice in these cases is of rare occurrence. When the hepatic and *larger ducts* are occluded, the liver becomes enlarged, and there is jaundice, sharp pains, colic, and sometimes rigors and sweats. If the hepatic duct is closed, the gall-bladder is normal in size. Fatal rupture of the ductus hepaticus sometimes, though rarely, is the result of the impaction of a calculus in it.

When *small calculi* are formed within the *gall-bladder*, they often cause no inconvenience; when they reach a large size they excite inflammation, which may ultimately cause closure of the neck of the gall-bladder. When the gall-bladder contains a large number of calculi, violent physical exertion causes pain, which disappears during rest. Sometimes the patient may actually "feel something rolling around" in the vicinity of the gall-bladder, which on a physical examination is found enlarged, more or less tender, hard, and nodulated, and by a stethoscopic examination gives to the ear the impression of a number of pebbles being grated together in water.

If biliary calculi in this situation cause perforation of the gall-bladder, a fatal peritonitis follows, or a biliary fistula may be formed between the gall-bladder and the stomach, which will be attended by sudden intense pain, with obstinate vomiting; sometimes one or more calculi are found in the vomited matter. The vomiting of a gall-stone cannot be accounted for on the ground of reversed peristaltic action after the stone has passed the ductus communis into the duodenum. Again, when calculi are formed in the gall-bladder, a fistulous opening into the duodenum may occur, followed by vomiting and signs of a local peritonitis, or of intestinal hemorrhage and hæmatemesis. An opening from the bladder into the colon is exceedingly rare, for the colon is very movable. The symptoms which attend such a perforation are obscure. The gall-bladder may open into the pelvis of the right kidney, and then biliary concretions will be voided in the urine. There is an instance on record where, during pregnancy, a communication was made between the gall-bladder and the uterus, the discharge of the calculi taking place at the birth of the infant. If an opening from the gall-bladder into the vena portæ occurs, symptoms of pyæmia will develop very rapidly. If perforation of the left pleural cavity occurs, fatal pleurisy will result. A single, rarely a double, fistulous canal may connect the gall-bladder with the external surface; the opening is usually near the umbilicus, and may discharge for months. It may cicatrize, and form a mass of fibrous induration; or abscesses may form when a large calculus plugs the fistula formed by previous perforation. If perforation occurs, recovery is most frequent when an external opening is established.

When a gall-stone has by any means entered the intestinal canal, it may be voided *per anum* or it may lead to an intestinal obstruction, ulceration, or gangrene of the intestine. Obstruction in the common duct may be temporary or permanent. If temporary there is no jaundice; if the obstruction is complete and is continued for twenty-four hours, jaundice is added to the other symptoms; this jaundice increases and is persistent when the obstruction is permanent.

Biliary colic, or the passage of gall-stones, is the name applied to the peculiar and severely painful symptoms produced by the passage of one or more calculi along some one of the larger biliary ducts. Usually after a hearty meal, or after some jolting exercise, as horseback riding, the patient is suddenly seized with a severe pain in the epigastrium, which is increased by change of position or pressure. Sometimes slight rigors, nausea, eructations, and attacks of yawning precede the colic. The pain is paroxysmal, and has its seat at a point where a line from the right nipple to the anterior superior spinous process of the left ilium crosses the free margin of the ribs. It radiates backward and upward, often as far back as the right shoulder, and *may* extend over both hypochondriac regions. It has been described by patients as boring, tearing, piercing, or lancinating. It is often so agonizing that patients will roll about the floor or bed, double themselves up, and groan with the pain. The face is pale and covered with cold sweat, and the pulse is very small. The abdominal muscles are rigid, and pressure greatly augments the pain. Vomiting, hiccough, a distended and tympanitic abdomen are often present during an attack, and a weak or feeble subject may faint, or pass into convulsions, which are epileptiform in character. Fatal syncope has occurred during an attack of gall-stone colic. After a few hours, sometimes a day, of exhausting and intense pain, the patient experiences sudden relief, and the pain entirely disappears;

often the pain remits, but does not cease until the calculus enters the duodenum; an exacerbation occurs at the moment the calculus enters the intestinal canal. Jaundice is often present, but not until the attack has continued for twenty-four hours. During the colic, the gall-bladder is very sensitive to pressure; during and after the attack, the patient is very much exhausted, and shows great lassitude. When jaundice is present the fæces are clay-colored, and the bowels are apt to be constipated. After the attack, gall-stones may be found in the fæces. It is to be remembered that fresh gall-stones are slightly heavier than water. The urine, if jaundice exists, contains bile-pigment and is mahogany in color; after the colic, it deposits lithates and lithic acid.

Differential Diagnosis.—Gall-stone colic may be mistaken for *cardialgia, intestinal* and *renal* colic. *Cardialgia* may be mistaken for biliary colic when there is no jaundice present. In cardialgia, pain comes on immediately after eating; gall-stone colic has no necessary connection with taking food. In cardialgia, the symptoms are referred to the epigastrium alone, while in biliary colic the pain shoots to the right shoulder and back. In cardialgia, the pain gradually diminishes; in biliary colic it suddenly ceases. In gall-stone colic, the presence of a gall-stone in the fæces is pathognomonic.

In *intestinal colic,* the pain begins at the umbilicus, and radiates over the abdomen; in gall-stone colic it has its seat at the free border of the ribs, and shoots to the back and upward to the right shoulder. In intestinal colic, pressure relieves the pain; in gall-stone colic it aggravates it. In intestinal colic, the pain is intermittent; in gall-stone colic it is constant, though paroxysmal. In intestinal colic, jaundice is *never* present, while it may exist in biliary colic. Intestinal colic accompanies or is followed by diarrhœa; in gall-stone colic, the fæces are firm and may be clay-colored.

With *renal colic,* the pain shoots from the region of the affected kidney to the inner part of the thigh and end of the penis, and *the testicle is retracted;* in gall-stone colic, the direction of the pain is upward and backward. In renal colic there is a constant desire to micturate. There is no urinary disturbance in biliary colic. In renal colic, after the cessation of pain, pus, blood and epithelium are found in the urine; after gall-stone colic, bile-pigment is found in the urine. Jaundice and clay-colored stools frequently containing gall-stones may be present in biliary colic; they are all absent in renal colic. The gall-bladder is very tender after biliary colic; while there may be dull pains in the region of the loins after the passage of a renal calculus.

Cancer of the head of the pancreas may readily be mistaken for gall-stones in the common duct.

Prognosis.—The sudden and unexpected terminations and varied consequences due to the formation of a gall-stone, render it impossible to give any rule for the prognosis. When a large stone, without facets, has been voided, in any manner, from the bile passages, the prognosis is better than when small facetted calculi are found. Oft-repeated attacks of biliary colic are bad. Catarrhal and exudative inflammations of the bile passages are frequent accompaniments of gall-stones; and pulmonary gangrene, empyema, and pneumonia may sometimes *complicate*. Though it is not necessarily a fatal disease, death may result from peritonitis, ulceration, gangrene or obstruction of the intestines, pyæmia, pylephlebitis, abscess of the liver, from exhaustion, or from the escape of bile through an external opening. Death may occur during an attack of colic, from unexplained causes.

Treatment.—An attack of biliary colic demands that attention be given, first, to the pain: this is best relieved by morphine, which should be given hypodermically, but never in such amounts as would be toxic were the patient free from pain. Inhalations of chloroform or ether may be employed to relieve the severity of the spasm. The application of two or three leeches over the gall-bladder is often followed by relief, and diminishes the chances of inflammation of the bile-ducts. Large draughts of warm water, containing bicarbonate of soda, often relieve the pain at the onset of the attack. At the same time put the patient in a tepid bath, or wrap warm cloths about the abdomen. In mild cases, and when opium is contraindicated, belladonna will be sufficient, in connection with anodyne fomentations over the region of the gall-bladder. If the patient shows signs of collapse, stimulants, ammonia and brandy should be administered.

A patient who has passed gall-stones must be put on a restricted diet; wines or fats should be prohibited; exercise in the open air, and an entire change in the mode of life, are important. Mineral waters, whether by giving an alkaline bile or by an increase in the amount secreted, cause the number of gall-stones to diminish, and also allow them to be passed with less pain. A prolonged course of alkaline mineral water has been found the best remedy against the formation of gall-stones. Ether, turpentine, chloroform and hydrate of chloral have been proposed as specifics, it being thought that they have the power of dissolving the gall-stones.

FUNCTIONAL DERANGEMENTS OF THE LIVER.

The terms *biliousness* and *torpid liver* were more frequently used twenty years ago than now. Many, indeed, have denied that any such conditions exist, but there is undoubtedly a variety of symptoms (such as constipation, yellow and itching skin, dark urine, headache, lassitude, furred tongue, bitter taste in the mouth, etc.), which can properly be classed as dependent upon functional derangement of the liver. Writers describe ten varieties; I shall only briefly consider those which are the most common. In these functional hepatic derangements there are no morbid appearances in the organ itself to account for the symptoms.

Etiology.—Functional derangement of the liver may be due to structural diseases (*e. g.*, cirrhosis, abscess, and acute yellow atrophy), to dyspepsia, both gastric and intestinal, to atony of the bowels, to obstructive diseases of the heart and lungs, to the specific fevers, malaria especially, to faulty diet, the food being too rich, to the daily use of alcoholic beverages, especially ales and sweet wines and liquors (not from whiskey, brandy or gin, unless in the form of a hot toddy or sweet punch), to badly ventilated, hot, and moist apartments, sedentary habits, a deficient supply of oxygen, a warm climate (India, for instance), and finally to anxiety and prolonged mental labor. In many cases the tendency to "liver complaint" is inherited; the children of the diabetic or gouty are very prone to functional derangements of the liver.

Symptoms.—Few cases are exactly alike. The prominent symptoms which usually first attract the patient's attention are anorexia, a bitter taste in the mouth (due to taurocholic acid in the blood), flatulency, "acidity" and pyrosis.[1] The tongue is large, pale, and flabby, with indentations of the teeth along its edges. It may be white, showing elongated papillæ-like villi. The fæces are pale, unless they have remained long in the large bowel, when they are blackish. Constipation and diarrhœa may alternate. When bile is in excess the fæces are semi-fluid and contain more bile than normal. It is a question whether melæna ever occurs as a *sole* result of hepatic derangement, but hemorrhoids are very common. There is often a sense of weight, fulness, tightness, burning, or even actual pain over the liver. Those who suffer from functional derangement of the liver may become very fat, or they may emaciate rapidly. Emaciation results either from deficient production of bile or from derangement of the glycogenic function of the liver. Bile may saturate the texture of the body for months, and yet no symptoms of blood poisoning occur so long as the eliminating function of the kidneys is not impaired.

A deficient elimination of cholesterin may give rise to "biliousness," and thus be a part of functional derangement of the liver.[2] "Cholesteræmia" is said to be associated with obstinate constipation, and Dr. Murchison regards this as "torpor of the liver," or at least one, and a frequent, form of it. Lithates and pigments deposited in the urine should always be regarded as signs of functional derangement of the liver arising from causes sometimes temporary and sometimes permanent. Murchison says "lithuria, like glycosuria, must be classed as a functional derangement of the liver," and he calls the antecedent morbid blood state *lithæmia*. In many, who by heredity are predisposed to "liver troubles," the liver is capable of performing its healthy functions only under the most favorable circumstances, and functional derangement is at once induced by articles of diet which most persons can easily digest.

"Gouty dyspepsia," "latent gout," suppressed, anomalous or irregular gout, are terms which in many instances should be dismissed, and "functional derangement of the liver" substituted for them, for the symptoms which have been ascribed to them occur in those who neither inherit nor ever have shown any gouty tendencies.[1] Biliary calculi (cholesterin and bile-pigment) may result from hepatic derangement. Frerichs regards the coincidence of gall-stones and urinary calculi in the same individual as purely accidental. Since the kidneys eliminate certain products of the liver, renal derangements may be a consequence of faulty hepatic digestion. Hence Murchison places lithæmia among the chief causes of Bright's disease of the kidneys.[2] Many suppose that albuminuria may be induced by functional derangement of the liver, independent of any morbid kidney change, and this accords with the modern theories of albuminuria.

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[1] The functions of a healthy liver are, *first*, sanguinification; *second*, the re-combination of albuminous matter derived from the food and tissues; *third*, the formation of urea and lithic acid, both of which are afterward eliminated by the kidneys; *fourth*, the secretion of bile, most of which is reabsorbed; *fifth*, the glycogenic function. Among the most constant results of functional hepatic derangement is imperfect formation of urea evidenced by the deposit of lithic acid or lithates in the urine. When a great part of the liver has been destroyed by disease the urea is lessened or disappears from the urine. Destructive nitrogenous metamorphosis is unquestionably an important function of the liver.

[2] Virchow's Archiv. Bd. 65, p. 410. 1875.

After the functions of a liver have been interfered with for some time, the structure of the liver is very liable to become diseased. Fatty liver and cirrhosis are common sequelæ, and their causes are closely allied to functional derangement of the liver.[3] Senile decay (sometimes premature), fatty, calcareous and atheromatous arterial changes are very frequently direct sequelæ of functional hepatic derangement. It is questionable whether the rheumatic hyperinosis is due to non-destruction of fibrin in the liver, as Murchison would have us believe. But the anæmia of this cachexia is undoubtedly often due to it.

Symptoms.—Those who suffer from torpor of the liver complain of lassitude, drowsiness, pain in the limbs, dull pain in the right hypochondrium often shooting up the right side to the shoulder, and not infrequently of sciatica and lumbago. Circumscribed patches of skin, usually on the extremities, often become hot and burning. Headache, usually frontal, is very common, and when induced by indiscretions in diet it is called "bilious" or "sick headache," and the patient states that he has had another "bilious attack." Dizziness, dim vision, and muscæ volitantes are frequent results of over-eating in those whose livers are functionally deranged. Convulsions, paresis and cramps in the legs are rare, but they may occur. Melancholia, insomnia, hypochondriasis, irritability of temper, and moodiness are consequences of deranged liver-function. The term "bilious temperament" has passed into common use. In some cases there are cardiac palpitations, an irritable, irregular or even intermittent pulse, cold extremities, and slight lividity or cyanosis, and, according to Sir James Paget, venous thrombosis may result from functional derangement of the liver. Paget and Murchison regard lithæmia, *i. e.*, functional derangement of the liver, as causing acute urethritis (non-specific) in many instances.[4]

After prolonged hepatic derangement psoriasis, lichen, eczema, lepra, urticaria, boils, carbuncles, pigment-spots (popularly called *liver spots*), and pruritus are liable to appear. Frequently the same individual will within a year have three or four of the above-named skin diseases as a direct result of functional derangement of the liver.

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[1] Gout is one result of lithæmia; and urinary calculi are frequently but an exhibition of functional derangement of the liver.

[2] Clin. Lec. Dis. Liver, pp. 572-573.

[3] Trousseau describes a chronic gouty hepatitis that comes under this head.

[4] British Med. Journal, 1875, i. 701.

CHRONIC PANCREATITIS.

Morbid Anatomy.—The changes are identical with cirrhotic processes elsewhere, *e. g.*, in the liver and spleen. This process may lead to complete disappearance of the gland substance or to closure of the duct and the consequent formation of cysts. The head of the gland is most involved in the cirrhotic process. Interstitial hemorrhages may occur, or little cysts may stud the gland. Adhesions generally bind the organ to the adjacent parts. In chronic suppurative pancreatitis the pus may infiltrate the gland, or there may be one or more small abscesses. The contents of the latter may become cheesy or calcareous.

Etiology.—The causes are similar to those of cirrhosis of the liver;—calculi, the pressure of an adjacent tumor, or extension of inflammation from adjacent parts, especially ulcers of the stomach and duodenum. It may be associated with syphilitic infection.

Symptoms.—The only symptoms that could lead to a diagnosis are fatty stools, intercurrent mellituria, neuralgic pains, and the presence of a transverse tumor in the epigastrium. Abdominal dropsy and signs of intestinal obstruction may be caused by the pressure of the hard gland which acts as an abdominal tumor. A peculiar cachexia is usually present.

FATTY DEGENERATION.

Two forms are recognized:

I. Fatty infiltration of the connective-tissue investing the gland and surrounding the acini, where the new growth of fat-tissue causes atrophy and disappearance of the gland cells by its pressure. The whole gland may look like a mass of fat with only a central canal.

II. Fatty degeneration affects the gland cells and ultimately destroys them; the acinous structure is preserved in the midst of a soft, flaccid and wasted gland. A more or less abundant fatty emulsion is found in the ducts.

Etiology.—The first form occurs in general obesity and in chronic alcoholismus. The second is due to the same causes, and also to heart disease or obstruction to the outflow of the pancreatic secretion.

WAXY DEGENERATION.

The vessels of the pancreas and the cells of the acini may exhibit amyloid change. This is the rarest disease of the pancreas.

CANCER OF THE PANCREAS.

This is the most frequent form of primary disease of the pancreas. In one hundred cases of cancer the pancreas was found involved in five. Scirrhus is the most frequent variety of cancer found here. Pancreatic cancer tends to involve adjacent organs; the bile-duct and left ureter may be pressed upon and obstructed, and the mass may compress the splenic or superior mesenteric vessels, the vena portæ, the inferior cava, or even the aorta. The cancer may ulcerate into neighboring structures. The canal of Wirsung may be obstructed, and then cysts will form.

Etiology.—It occurs chiefly in men after the fortieth year; further than this little can be said.

Symptoms.—The symptoms are varied, because the neighboring organs are so frequently and extensively involved. Neuralgic and paroxysmal pain, is an important symptom. The presence of a tumor with enlargement of the adjacent lymphatic glands is essential for a diagnosis. Vomiting, jaundice, dyspepsia, dropsy, œdema of the feet,—all may be present. Sometimes the stools may be fatty. There may be constipation or diarrhœa, or the two may alternate. The general symptoms are those of anæmia.

Differential Diagnosis.—A pancreatic cancerous tumor may pulsate and have a bruit conducted from the aorta, and therein simulate *aortic aneurism.*

Prognosis.—Cancer of the pancreas usually causes death within a year. The treatment is symptomatic.

Small-celled Sarcoma usually occurs as a melanotic tumor. It is very rare, and clinically indistinguishable from carcinoma.

Tuberculosis of the pancreas, secondary to that of the lungs and peritoneum, develops in the connective-tissue between the acini. Caseous nodules are oftener met with than diffuse miliary tubercles. It is denied that the pancreas ever is involved in acute miliary tuberculosis.

Syphilitic Gummata are usually found in connection with syphilitic interstitial pancreatitis. They have been described by Rostan and Rokitansky. It is believed that more frequent examinations of the gland would reveal a larger number of gummata.

CYSTS IN THE PANCREAS.

Cysts in the pancreas are due to retention of the pancreatic secretion, from obstruction of the duct by calculi, or from external pressure of tumors. Hemorrhagic cysts are very rarely found. When the duct is closed near its mouth, the canal and its branches look like a bunch of currants. Atrophy and cirrhosis of the gland may result from these cysts, which at first contain a normal secretion which afterward becomes purulent, hemorrhagic, or albuminous. Hæmatoidin crystals, lime-salts and urea have been found in these cysts. The cyst-walls thicken from connective-tissue developments.

Etiology.—Any tumor, either of the pancreas itself or of neighboring parts, calculi, cirrhosis of the pancreas, or *angular displacements of the pancreas* may cause it.

Symptoms.—The only symptom is the discovery of a smooth lobulated tumor in the region of the pancreas.

CALCULI OF THE PANCREAS.

Calculi of the pancreas are usually gray-white, rounded masses of carbonate or phosphate of lime. They are situated anywhere in the pancreas, are either free or embedded, vary in size from microscopic dust to a walnut, and also vary in number, but rarely exceed fifteen or twenty. Laminated protein concretions are described by Virchow.

Etiology.—Anomalies in the pancreatic juice itself, catarrh of the ducts, and retention cysts are the most frequent causes.

Symptoms.—There will be no symptoms except when interstitial inflammation is excited, or the common duct is pressed on so as to cause jaundice. *Round worms* have been found in the pancreas.

DISEASES OF THE SPLEEN.

Diseases of the spleen will be considered in the following order:

I. *Hyperæmia.*
II. *Inflammation, or Splenitis, including Embolism and Infarction.*
III. *Hypertrophy, or Chronic Enlargement.*
IV. *Degenerations, Waxy or Sago-spleen.*
V. *Morbid Growths.*
VI. *Parasites.*

HYPERÆMIA.

Splenic hyperæmia may be active or passive.

Morbid Anatomy.—The accumulation of blood in the vessels and intervascular spaces of the spleen, causes the enlargement which occurs in hyperæmia; the organ may be increased to five or six times its normal size and yet retain its normal shape. Its color is darker than normal, its capsule is usually tense and shining, and its consistency is often diminished, being sometimes as soft as pulp. A *microscopic* examination shows no new elements in the spleen, only an increase in the number of its normal ones.

Etiology.—A physiological congestion of the spleen takes place after every meal. A pathological engorgement occurs: (1) when there is any obstruction to the venous flow from the spleen, as happens in certain cardiac, hepatic, and pulmonary diseases; (2) in the acute infectious diseases, such as typhus, malarial fevers, and pyæmia; (3) at the menstrual epoch, depending upon an abnormality of menstruation; (4) as the result of injuries and inflammation, when the hyperæmia will be circumscribed.

Symptoms.—The symptoms of simple hyperæmia of the spleen are usually not well marked; the patient may complain of a sense of weight in the left hypochondrium and more or less tenderness on pressure over the splenic region. Palpation and percussion will discover a tumor in this region of greater or less size; this tumor extends obliquely downward toward the umbilicus, rises and falls with each respiratory movement, and has the outline of the spleen, with the characteristic notches on its lower rounded edge. The pale and anæmic appearance often met with in those-having splenic hyperæmia is due to the overloading of the spleen with blood at the expense of the rest of the body, rather than to any change in the composition of the blood.

Prognosis.—The prognosis is good, although in certain rare cases death has occurred from rupture of the distended organ.

Treatment.—The treatment is directed rather to the disease which gives rise to the hyperæmia than to the condition itself. Quinine in large doses has been found in most instances to remove the splenic congestion and relieve the accompanying symptoms.

INFLAMMATION OF THE SPLEEN.

(*Splenitis.*)

Primary splenitis is exceedingly rare; it is generally due to injury, embolism, or infarction, especially when occurring with pyæmia or septic diseases. It may occur in connection with morbid growths and abscesses in the spleen. There is a condition of the spleen resembling cirrhosis of the liver, called by some *chronic* or *interstitial* splenitis.

Morbid Anatomy.—The anatomical arrangement of the splenic arteries renders the spleen a favorable seat of metastatic inflammation. In acute splenitis a part or all of the organ may be attacked; the involved portions are congested and swollen, and the peritoneum over them is injected and covered with a fibrinous exudation. The spleen is of a deep purplish color and friable, being broken down as easily as coagulated blood. When a hemorrhagic infarction is formed in the spleen, it is usually without rupture of the blood-vessels, and is encircled by a zone of sero-hemorrhagic infiltration. These infarcts are at first of a brownish red color and of a firm consistency; later, they become dirty yellow in color, and either undergo fatty degeneration and become absorbed, or remain as cheesy and calcareous masses; or, lastly, the infarctions soften and abscesses form, which are single or multiple, sometimes fusing together, and again increasing by peripheral extension.

In a few instances these abscesses are found incapsulated in a proliferation of connective-tissue, but in most cases this is not the case; the connective-tissue breaks and a large sac is formed filled with pus; as much as thirty pounds have been found in one of these sacs. At last the capsule becomes involved, *perisplenitis* is set up, and, adhesions having formed between the spleen and adjacent parts, the abscess may open into some adjacent organ, as the stomach and colon, into the thorax or abdominal cavity, or an external fistulous opening may be formed. Localized suppurative inflammation of the small Malpighian bodies in the spleen has been found to occur in typhus and other fevers.

"*Necrotic splenic softening*" may occur from an infarction caused by atheroma and endocarditis: the latter is not necessarily ulcerative. In these cases the lymph-cells in the fibrinous reticulum of the clot become fatty, and then they mass themselves into spheres of fat-crystals. The whole infarction softens and becomes a fatty, pulpy mass. The capsule over such a spot is villous and appears covered with vegetations. A gangrenous condition of the left lower lobe of the lung has been caused by such a form of metastatic splenitis; with the intense engorgement which accompanies acute splenitis, hemorrhage into the organ may occur, the capsule may be ruptured, and a fatal peritonitis may be induced.

Chronic splenitis is the result of long-continued splenic congestion. The spleen is of a brownish-red or slate color, its capsule is thickened, and covered with very firm vegetations and new connective-tissue formations, which are highly vascular. The organ is more or less pigmented, owing to the pigmentary deposit in the endothelia of the veins. This whole process is analogous to that which occurs in cirrhosis of the liver, except that a spleen which is the seat of interstitial inflammation is larger than normal.

Etiology.—It is very doubtful whether idiopathic splenitis ever occurs. Blows and severe muscular exercise are said to have caused it. Splenitis is usually metastatic, the embolic plug, the result of endocarditis and valvular disease, most often having its origin in the left heart, although it may be induced by thrombotic changes in the aorta. Rarely the embolus comes from the lungs, having passed through the pulmonary vein and left heart. In pyæmia and its allied states hemorrhagic infarctions of the spleen are of frequent occurrence, and a similar condition has been noticed in Bright's disease and in the infectious diseases. Extension of inflammation, especially the result of ulcerative changes in the stomach, is an occasional cause of acute splenitis.

Symptoms.—These are vague; often there is nothing except the local changes to direct attention to the spleen as the seat of disease. There is no pain, unless the capsule is involved; if pain is present it will be increased by a full inspiration. There may be hectic fever, but, as splenitis is secondary to some febrile disease, the fever may be attributed solely to the latter. There may be a sense of weight and pain in the left hypochondrium, and even shooting pains in the left shoulder and arm.

Vomiting of blood and pus, or the simultaneous passing of blood and pus with the fæces is indicative of rupture of a splenic abscess into the stomach; this, however, is an exceedingly rare event. The recognition of a splenic abscess will depend upon its attaining a sufficient size to form an appreciable fluctuating tumor in the splenic region.

Physical Signs.—*Inspection* will show a marked enlargement in the splenic region.

Palpation may discover a fluctuating mass. The "notchings" on the anterior margin of the spleen are usually readily made out. The mass is more or less movable, unless adhesions have formed between the splenic capsule and adjacent parts.

Differential Diagnosis.—Acute splenitis may be mistaken for *cancer of the stomach*, disease of the *pancreas*, or *hepatic* disease, especially that involving the *left lobe* of the liver. *Ovarian* tumors will rarely be confounded with it. In stomach diseases the absence of fever, the vomiting, pain and discomfort dependent *on the ingestion of food*, the long dura-having splenic hyperæmia is due to the overloading of the spleen with blood at the expense of the rest of the body, rather than to any change in the composition of the blood.

Prognosis.—The prognosis is good, although in certain rare cases death has occurred from rupture of the distended organ.

Treatment.—The treatment is directed rather to the disease which gives rise to the hyperæmia than to the condition itself. Quinine in large doses has been found in most instances to remove the splenic congestion and relieve the accompanying symptoms.

INFLAMMATION OF THE SPLEEN.

(*Splenitis.*)

Primary splenitis is exceedingly rare; it is generally due to injury, embolism, or infarction, especially when occurring with pyæmia or septic diseases. It may occur in connection with morbid growths and abscesses in the spleen. There is a condition of the spleen resembling cirrhosis of the liver, called by some *chronic* or *interstitial* splenitis.

Morbid Anatomy.—The anatomical arrangement of the splenic arteries renders the spleen a favorable seat of metastatic inflammation. In acute splenitis a part or all of the organ may be attacked; the involved portions are congested and swollen, and the peritoneum over them is injected and covered with a fibrinous exudation. The spleen is of a deep purplish color and friable, being broken down as easily as coagulated blood. When a hemorrhagic infarction is formed in the spleen, it is usually without rupture of the blood-vessels, and is encircled by a zone of sero-hemorrhagic infiltration. These infarcts are at first of a brownish red color and of a firm consistency; later, they become dirty yellow in color, and either undergo fatty degeneration and become absorbed, or remain as cheesy and calcareous masses; or, lastly, the infarctions soften and abscesses form, which are single or multiple, sometimes fusing together, and again increasing by peripheral extension.

In a few instances these abscesses are found incapsulated in a proliferation of connective-tissue, but in most cases this is not the case; the connective-tissue breaks and a large sac is formed filled with pus; as much as thirty pounds have been found in one of these sacs. At last the capsule becomes involved, *perisplenitis* is set up, and, adhesions having formed between the spleen and adjacent parts, the abscess may open into some adjacent organ, as the stomach and colon, into the thorax or abdominal cavity, or an external fistulous opening may be formed. Localized suppurative inflammation of the small Malpighian bodies in the spleen has been found to occur in typhus and other fevers.

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Cancer, abscess, or *cirrhosis* of the liver would give *physical signs* which could hardly be confounded with those of acute splenitis—the position of the area of dulness would of itself be sufficient for a diagnosis. The condition of the stools, the urine, and the color of the skin are often sufficient to lead to a diagnosis of hepatic diseases. If the tumor is of considerable size, the diagnosis of splenic abscess can always be safely reached by the aid of the exploring trochar.

Prognosis.—Suppurative splenitis is always a dangerous disease, and is rarely recovered from, even when an extensive opening is established.

Treatment.—No special treatment is called for unless the pain is severe, when anodynes and fomentations may be used. When an abscess can be made out it should be treated in the same manner as an hepatic abscess.

HYPERTROPHY OF THE SPLEEN.

(*Enlarged Spleen.*)

Enlargements of the spleen are of two kinds: (1) acute and transient; and (2) chronic and permanent. The first class occurs in fevers, *e. g.*, typhoid; the second from long-continued and repeated congestion, as in chronic malarial infection.

Morbid Anatomy.—In *acute enlargement*, the spleen may reach four or five times its normal size. The capsule becomes thin and tense. The spleen pulp varies in consistence, and may even be completely diffluent; its color varies from a bright pink to a red black. Hemorrhagic foci independent of embolism may occur. Many lymphoid cells contain one or more red corpuscles, the latter either being normal in size or smaller (1–8000 of an inch). This microscopical appearance is best marked in typhoid fever.[1]

Chronic Enlargement of the Spleen.—The organ is enlarged without any obvious change in texture; there is an increase in all its elements, and it acquires a more or less fleshy consistency. It sometimes reaches a very great size, filling the left side of the abdominal cavity from the ribs to the pelvis; it may be increased to twenty pounds in weight. Its normal shape is unchanged and the notches on its edges are distinctly retained. In rare cases chronic enlargement occurs as a multiple nodular hyperplasia.

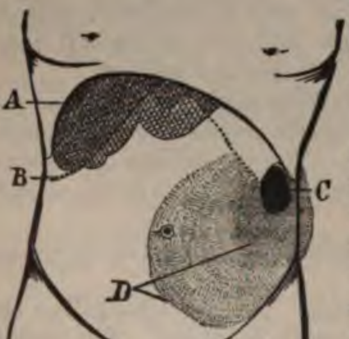

FIG. 90.
Diagram illustrating the abdominal areas of percussion-dulness in Splenic Enlargement.

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[1] The color of the spleen in congestive enlargement is always *paler* than that of the other viscera, on account of the presence of a larger quantity of white blood corpuscles than are found elsewhere in the body.

Etiology.—Acute enlargement of the spleen is met with most frequently in acute infectious diseases. Many regard the accumulation of micrococci in the spleen and their retention within the splenic protoplasm as a cause of acute swelling.[1] Of all the diseases that cause sudden and great enlargement of the spleen, the most frequent are typhoid and intermittent fevers.

Chronic enlargement of the spleen is frequently associated with cirrhosis and other chronic affections of the liver, and with chronic cardiac and pulmonary diseases that induce long-continued or repeated congestion of the organ. But chronic malarial infection is its most common and constant cause. Chronic enlargement of the spleen is part of the history of leucocythæmia; it is never idiopathic. Chronic mercurialism disposes to enlargement of the spleen.

Symptoms.—Acute enlargement of the spleen is part of the history of acute general diseases; its natural symptoms are few: pain on pressure, dyspnœa, and an increased area of splenic dulness are the most constant and prominent. Chronic enlargement of the spleen is attended by no symptoms except the physical signs of splenic enlargement.

Treatment.—Quinine and iron are always indicated, but they should be given alternately and never at the same time; they may be combined with arsenic and cod-liver oil. In malarial hypertrophy of the spleen the patient should reside in a non-malarial district. Inunctions of the biniodide of mercury are strongly recommended by English authorities, but I have never seen any beneficial results from their use.

AMYLOID DEGENERATION OF THE SPLEEN.

(*Sago Spleen.*)

Waxy or lardaceous degeneration of the spleen, also called the "sago spleen," is a part of a general cachexia in which other organs are primarily involved.

Morbid Anatomy.—It occurs in two forms; in one it is limited to the Malpighian bodies, in the other it is diffused. In both the organ is enlarged, rounded and doughy. The capsule is tense, but not thickened, and is usually smooth and glistening.

On section the first variety presents the appearance of a number of sago granules, the Malpighian bodies being enlarged to 1-25 or 1-12 of an inch in diameter, and filled with waxy material which gives the characteristic reaction with iodine. The corpuscles through which the arteries pass are involved, but the wall of the in-going vessels may remain normal. The "adenoid" or "cytogenic" tissue, the lymph-corpuscles, and the capillaries of the spleen are, however, infiltrated with waxy material, massed together, and channelled by healthy capillaries. The veins near the diseased Malpighian bodies are sometimes involved. In diffuse lardaceous degeneration, the spleen, *on section,* is pale, homogeneous, glistening and anæmic; all the vessels, the trabeculæ, and capsule are involved.[2]

Etiology.—The causes of waxy spleen are identical with those of waxy liver; it is met with in chronic bronchitis with bronchiectasis, in phthisis, chronic Bright's disease, chronic peritonitis, cirrhosis of the liver, chronic alcoholismus, and intermittent fever. Sago spleen frequently accompanies chronic intestinal catarrh in children. Syphilis is probably its most frequent cause.

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[1] Mosler and Birch-Hirschfeld.

[2] Rokitansky regards this form as but a later stage of "sago spleen."

Symptoms.—As the liver and intestines are generally involved in the same change, the waxy cachexia will not be characteristic of splenic changes. There will be anæmia, accompanied by a great increase in the area of splenic dulness. Late in the disease there is usually anorexia, vomiting, and hemorrhages, but it is not possible to determine to what extent these various symptoms depend on the splenic disease.

The *diagnosis* rests mainly on its etiology.

The *prognosis* is unfavorable.

Treatment.—It never calls for independent treatment. Niemeyer regards iodide of iron as the most efficacious drug. Our first efforts should be to cure or remove the causes or conditions which have led to its development, and to improve the general condition of the patient by tonics, hygienic measures, and a carefully regulated, nutritious diet.

MORBID GROWTHS OF THE SPLEEN.

Cancer of the spleen is very rare; it may be secondary to cancer of the stomach, mamma, liver, or brain. When disseminated, it is generally of the encephaloid variety. It may develop in the hilum by contiguity of cancer in the other organs. Secondary isolated growths may be scattered through its substance, or in many more instances it may be the seat of primary cancer. In *pigment-cancer* of the spleen, the organ rapidly enlarges to nearly double its size; in other forms it is but slightly enlarged. The symptoms are obscure and of little clinical importance.

Gummata, or syphilitic tumors of the spleen, are only met with in connection with amyloid changes, and are accompanied by similar developments in the liver. Syphilis thus shows itself in the spleen in one of four ways,—waxy degeneration, gummata, inflammation of the spleen-pulp, or hypertrophy of the spleen with increase in interstitial tissue. Syphilomata are of no clinical importance.

Tubercles in the spleen develop in the spleen-pulp. The nodules may be small and gray, or large, yellow and cheesy. In acute tuberculosis, the spleen rapidly enlarges as the tubercles develop. Tubercular formations are very common in young children. Yellow tubercular masses, varying in size, are frequently found in the spleen in connection with similar formations in other parts of the body; occasionally they soften and form abscesses. The small splenic vessels are often clogged with lymph and fibrin. Tubercles of the spleen cannot be recognized during life.

Cysts have been found in the spleen. They are associated with cystic developments in the liver and omentum.

Hydatids, when occurring in the spleen, usually accompany similar developments in the liver and peritoneum. The enlargement of the spleen may cause a sense of weight in the splenic region, and if the splenic capsule becomes inflamed their development will be accompanied by sharp pains in the left side. An hydatid tumor in the spleen usually fluctuates, but it rarely gives the hydatid fremitus. An exploratory puncture decides its character.

The prognosis and treatment are the same as in hydatids of the liver.

SECTION III.

DISEASES OF THE HEART, BLOOD-VESSELS AND KIDNEYS.

Diseases of the heart may be classified as follows:

I. *Pericarditis.*
II. *Endocarditis.*
III. *Valvular Lesions.*
IV. *Cardiac Hypertrophy.*
V. *Cardiac Dilatation.*
VI. *Myocarditis.*
VII. *Cardiac Degenerations.*
VIII. *Cardiac Atrophy.*
IX. *Cardiac Thrombosis.*
X. *Cardiac Aneurism.*
XI. *Morbid Growths and Parasites.*
XII. *Tuberculosis of the Pericardium.*
XIII. *Cardiac Neuroses.*
XIV. *Hydro-pericardium.*
XV. *Pneumo-hydro-pericardium.*

PERICARDITIS.

The pericardium is a fibro-serous sac; the fibrous layer is firmly adherent to the diaphragm and is attached to the large vessels about two inches above the heart; it forms a closed sac. The serous layer is in close apposition to the internal surface of the fibrous layer, is reflected from the large vessels, and completely invests the heart itself. This shut serous sac, when diseased, behaves in all respects like the pleura. Inflammation of the pericardium may be acute or chronic. Chronic pericarditis is usually the sequel of acute.

ACUTE PERICARDITIS.

Acute pericarditis is perhaps more frequently overlooked than any other acute disease, for its subjective symptoms are rarely, if ever, well marked.

Morbid Anatomy.—At its commencement, the serous surface of the pericardium becomes more or less reddened, with here and there ecchymotic spots of irregular shape. The reddening may be circumscribed about the roots of the great vessels, or it may involve the whole visceral and parietal pericardium. The reddening is due to hyperæmia of the subserous capillary vessels. With the redness there are swelling and infiltration of its serous and sub-serous tissue. Following the hyperæmia and infiltration the epithelium desquamates and the membrane loses its natural glistening appearance. If the inflammatory action is continued, an exudation is poured out on its free surface: it may consist of but a few shreds of lymph, or a fibrinous layer may cover the whole of its cardiac or parietal surface. It varies in thickness from a line to three-fourths of an inch, or even more. This exudation is composed of fibrin, a few pus cells and detached epithelia; it causes the free surface of the pericardium to assume a roughened appearance; it is this appearance which has given rise to the expression "hairy heart." When there is only a very small amount of plastic exudation, it will usually be confined to that portion of the pericardium which covers the blood-vessels.

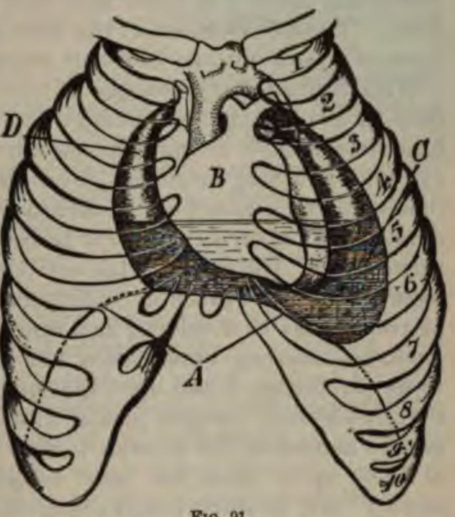

Fig. 91.

Diagram Illustrating the Morbid Anatomy and Physical Signs of Sero-plastic Pericarditis.

A. Line of diaphragm.
B. Heart.
C. Serous effusion into lower portion of pericardial sac.
D. Plastic exudation upon both visceral and parietal layers of the pericardium.

With or following the plastic exudation there may be a fluid effusion which varies in quantity and in quality. It may be sero-albuminous, sero-fibrinous, hemorrhagic, or purulent. It varies in quantity from three fluid ounces to several pints. In most instances it will be sero-fibrinous in character; it is rarely sero-albuminous. When it is small in amount it will gravitate to the most dependent portion of the pericardial sac. When it is large in quantity, the entire pericardial sac is filled, and the adjacent lung-tissue compressed, and the surfaces of the membrane have a reticulated or honey-combed appearance. It is always turbid from the molecular fibrin suspended in it, and may be yellow, green, brown, or red in color.

Hemorrhagic pericarditis is rare, except with purpura, scurvy, cancer of the lung, and tuberculosis. In this variety the line of demarcation between the false membrane and the pericardium is very indistinct.

Tuberculous pericarditis is attended by the development of tubercles in the pericardium and in the substance of the heart; the blood effused forms ochre-colored masses in the exudate. The exudations and effusions in pericarditis may all undergo absorption. The serous effusion is removed rapidly, the hemorrhagic with less facility, the plastic and purulent with still greater difficulty. The fluid disappears first, then the granular, and last the coagulated fibrin. The lymph and purulent exudations may undergo fatty metamorphosis and be absorbed, or remain in a cheesy, mortar-like mass, and finally become calcareous after the absorption of the more fluid portion of the degenerated mass. The calcareous material with connective-tissue formations may form ossified plates upon the surface of the heart and pericardium.

New connective-tissue formations may take place upon the surface of the pericardium under the layer of plastic exudation; if the inflammatory process is continued sufficiently long, these are converted into a firm fibrinous mass, causing either a permanent thickening of the pericardium, or adhesions between its two surfaces. Sometimes these adhesions are by bands stretching across from one portion to the other; at others there is complete agglutination of the two surfaces, and an entire obliteration of the pericardial cavity; in either case more or less complete organization takes place. The adhesions about the base are the most dense. Those at the apex are drawn out into fibrinous strings.

With inflammatory changes in the visceral pericardium, there will be more or less inflammatory change developed in the muscular tissue of the heart immediately beneath the pericardium. If the pericarditis has been extensive and long continued, the walls of the heart will become weakened; indeed, they are somewhat weakened in every attack of pericarditis. The development of myocarditis will be considered more fully under its appropriate head. Dilatation of the cavities of the heart may take place in consequence of the weakened condition of the cardiac walls, and cardiac hypertrophy may be developed as a result of this weakening and dilatation. Upon post-mortem examination not infrequently smooth, opaque, pearly-white patches are found upon the external surface of the heart. They are slightly elevated, variable in size, have irregular sinuous margins, and are usually located on the anterior surface of the ventricle. As to the nature of these spots there has been considerable discussion. These "*milky patches*" are, however, nothing more than growths of white, laminated connective-tissue with elastic fibres, immediately beneath the cardiac pericardium, and indicate the previous existence of a localized pericardial inflammation which has been recovered from without adhesions. In rare instances the two surfaces of the pericardium will become firmly agglutinated throughout their entire extent, and the pericardial sac will remain completely obliterated, and if attempts are made to separate them the cardiac muscle is torn. Under such circumstances, the movements of the heart carry with them the pericardium, and with each cardiac pulsation there is a lifting of the diaphragm.

Etiology.—Acute pericarditis rarely occurs as a primary affection, but is usually secondary, or is developed during the course of some other disease.[1] It may be produced by injuries to the pericardium,—by extension of inflammation from neighboring organs, as when it occurs with pleuro-pneumonia, pleurisy, necrosis of the sternum, ribs, rupture of abscesses, etc. It occurs most frequently in connection with that class of diseases which depend upon well-recognized blood-changes, and especially with those due to a specific infection; under this head are included pericarditis which accompanies acute rheumatism, Bright's disease, acute infectious diseases, as scarlatina, small-pox, typhus and typhoid fever, pneumonia, tuberculosis, syphilis, chronic alcoholismus, etc. Occasionally it is developed in connection with scurvy and purpura; then it is of the hemorrhagic variety. Cancer of the lung and tuberculosis also cause "hemorrhagic pericarditis."

When pericarditis occurs in connection with pyæmia and septic conditions, the effusion is purulent in character and accumulates rapidly. It is of most frequent occurrence in connection with acute articular rheumatism, Bright's disease and pneumonia. Often in rheumatic pericarditis, the articular rheumatic development occurs subsequent to the pericarditis. In rheumatism it is an *early*, in Bright's disease a *late* occurrence. Pericarditis occurring in connection with scarlet fever is especially liable to be overlooked, for its presence is not revealed until a large fluid effusion takes place.

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[1] A case of "*Idiopathic Pericarditis.*" Glasgow Med. Journal, Sept., 1878.

Symptoms.—The symptoms of acute pericarditis are rarely well defined. It is very difficult to give a clear description of the rational symptoms which attend its development, for it is usually associated with some other affection whose symptoms tend to obscure those of pericarditis; more than one-half of the cases are latent, and come on so insidiously that they would go unrecognized were it not for the physical signs which attend them.

The two prominent rational symptoms are pain in the precordial region and cardiac palpitation. The pain is usually confined to the precordial space; occasionally it involves the brachial plexus, and extends down the left arm; under such circumstances it is probably reflex in character. The pain may be increased in severity by pressing the left lobe of the liver against the diaphragm. It varies in severity; sometimes it is very slight, again it is of a sharp, lancinating character, and sufficiently severe to demand immediate relief. With the pain there is always more or less cardiac palpitation, a dry irritable cough, and a sense of constriction over the whole chest, with more or less dyspnœa; the intensity of the dyspnæa will vary with the amount of the fluid effusion. When the effusion is considerable and there is orthopnœa the patient becomes restless and the countenance assumes an anxious expression, with a painful look of suffering somewhat characteristic; he assumes the half-sitting posture, leaning somewhat toward the left side. Lying on the back with the head elevated, is the position usually preferred when the effusion is not large. The face is often livid.

At first the pulse is full and strong, ranging from 90 to 120 beats in the minute,—after the fluid effusion has taken place, it becomes feeble, suppressed and sometimes delayed. If the effusion is abundant the pulse has a tendency to become irregular, and not infrequently intermitting; it is always out of proportion to the activity of the heart and strongly *dicrotic*. The temperature usually rises one or two degrees,—in some cases it may rise as high as 104° F. In fatal cases the temperature falls toward the close of life, sometimes below normal. Jaundice sometimes occurs and headache and dizziness are frequently present; in the severe forms of the disease there is often delirium, the patient sometimes becoming so furious as to require restraint; at other times it is low and muttering. The delirium is often accompanied by delusions, tetanic or clonic spasms, and in rare cases convulsions occur, rapidly passing into coma and followed by death. Usually when the fluid effusion takes place, the acuteness of the symptoms subsides, and the patient experiences a sensation of oppression referable to the precordium,—he is disinclined to make any movement, for the least motion of the body gives rise to a sinking sensation with a tendency to syncope. Painful hiccough accompanies this symptom. The patient is now constantly in danger of sudden and fatal syncope from pressure of the pericardial accumulation upon the heart. Some maintain that sudden and fatal syncope never occurs in primary pericarditis, but that it is met with only after several attacks have occurred, and more or less extensive pericardial adhesions have taken place. This is not necessarily the case, for whenever large fluid effusions are developed, with the attendant weakening of the cardiac walls from superficial myocarditis, patients are constantly in danger from sudden syncope.

The severity of the symptoms in pericarditis corresponds to the intensity of the inflammation and the amount of the effusion ; if the inflammation is slight and the effusion moderate, the plastic exudation predominating, none of these symptoms will be present, and the subjective symptoms will only serve to attract attention to the heart as the seat of disease.

The subjective symptoms in many cases of pericarditis being so obscure, often altogether wanting, the physical signs become all important. In fact, in all cases of acute articular rheumatism, for the first two weeks it is an imperative duty each day to make a careful physical examination of the heart, especially if its action becomes irritable and the apex-beat is increased in force. Delirium in acute rheumatism ought at once to direct attention to the heart. The same care in examination should also be exercised in Bright's disease when convulsions or coma occur ; and in severe acute infectious disease the heart will often be found implicated.

Physical Signs.—These vary with its different stages. In the early stage the only sign furnished by *inspection* and *palpation* is an irritable, turbulent, forcible, and sometimes irregular action of the heart.

Palpation gives a friction-fremitus in a few cases. There is no change in the normal area of precordial dulness.

On auscultation the first positive physical signs of pericarditis are the *pericardial friction sounds.* They may be grazing, rubbing, or creaking in character. These friction sounds may be single or double, and may accompany the heart sounds or occur independently of them. They are always superficial in character and are generally restricted to the precordial space. Their point of maximum intensity is usually at the junction of the fourth rib with the sternum on the left side ; occasionally they will not be audible at this point, but will be heard over the large vessels at the base of the heart ; when this is the case it indicates that only a small extent of the pericardium is involved, and that the inflammatory changes are confined to that portion of the pericardium which covers the large vessels. When *absent,* as they sometimes are, their absence may be due to softness of the fibrin, feebleness of heart action, or alteration in, or abnormal position of, the lungs. Pericardial friction sounds may be increased in intensity by changing the position of the patient : when the body is thrown forward the heart will be brought nearer to the anterior wall of the chest and the friction sound will be more distinctly audible. These friction sounds will also be increased in intensity by a full inspiration, for the distended lung will press the two pericardial surfaces together and thus intensify the rubbing sounds. In this way a single friction sound may become double. These sounds are usually of short duration, disappearing after a few hours, or at most in a few days.

As soon as the *stage of effusion* is reached and liquid is poured into the pericardial sac, the friction sounds disappear and another class of physical signs are developed which mark the effusive stage of pericarditis.

Inspection now shows a diminution in the respiratory movements over the precordial space, and if the pericardial sac is distended—especially in children and young persons—there will be arching forward of the precordial region; this arching forward may extend from the second to the sixth intercostal space. On lying down the apex-beat often becomes more prominent. This bulged portion does not move with the rest of the thorax in respiration.

Palpation shows the point of the apex-beat to be raised and carried to the left of its normal position. This raising of the apex-beat is never actual —only apparent—for as the fluid accumulates the apex is pushed further back from the anterior wall of the chest, and the portion of the heart that is nearest to the chest wall appears to strike it and cause an "apex-beat," which is nearer the base the more fluid there is in the pericardium —a simple physical phenomenon dependent upon the "conicity" of the heart and the pear-shaped sac in which it hangs. The cardiac excitement and friction-fremitus which might have been present before the effusion occurred disappear, and if the effusion is large the apex-beat becomes imperceptible. Sometimes in extreme pericardial effusion an undulating impulse is communicated to the hand as it rests on the chest walls, by the action of the heart in the fluid.

On *percussion*, if the pericardium is distended with fluid, the area of precordial dulness is found to be increased in every direction, especially laterally and vertically. The shape of the enlarged area corresponds to the pyramidal form of the pericardial sac. Recent experiments prove that the triangular or pyramidal dulness is *not* due to the shape of the pericardial sac, but to the retraction of the edges of the lung. In a lateral direction the precordial dulness may extend from one nipple to the other; it may extend upward as high as the second or the first rib, or above the clavicle, and downward somewhat beyond the normal limits. A small amount of effusion is denoted by an increase in the width of the precordial area

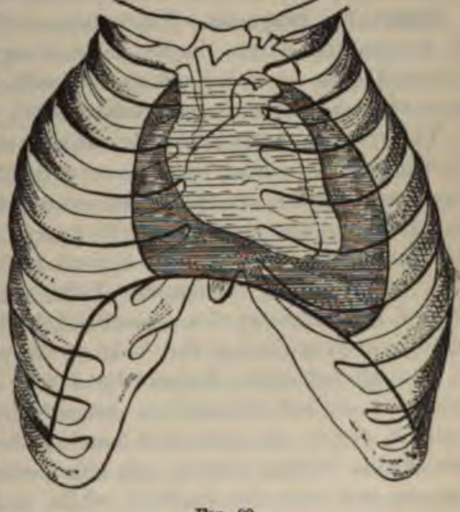

FIG. 92.

Diagram illustrating the Physical Signs of Pericarditis when the pericardial sac is distended with fluid.

of dulness at the lower portion of the precordial region; if emphysema exists the changes in the area of dulness will be less marked.

Upon *auscultation* an absence of respiratory murmur is noticed over all that space which is normally occupied by lung tissue, the lungs being pushed to the right and left by the distended pericardial sac. The friction sound which may have been present before the occurrence of the effusion disappears and the heart sounds become feeble or indistinct. In most cases the fluid disappears rapidly within a week or ten days.

Stage of Absorption.—As recovery takes place and the effusion is absorbed, the area of precordial dulness decreases and the pericardial surfaces again come in contact and the friction sound reappears, the heart sounds will become more distinct, the apex will assume its normal position, the cardiac impulse will regain its normal force, and the respiratory and vocal sounds are again heard over the space formerly occupied by the distended pericardium. If the anatomical changes developed in the substance, and on the surface of the pericardium, have been extensive, as the two pericardial surfaces come together they may become firmly adherent and all motion between the heart and pericardium cease. This condition cannot be recognized by physical examination—it is only to be inferred from the history of the case. If one who has had all the symptoms of pericardial effusion which has been followed by a friction sound that has gradually disappeared, leaving a slight intermittent action of the heart, suffers on active exertion from a sense of constriction above the precordial region, it may be inferred that the two surfaces of the pericardium have become adherent. Pericardial adhesions, whether general or in bands, may undergo absorption, and if a second attack of pericarditis is not developed motion between the two surfaces will be restored, and the only evidence of the disease will be the milky patches on the pericardial surface found at the autopsy.

Differential Diagnosis.—The existence of pericarditis can never be positively determined except by its physical signs; even when attention has been directed to the heart it is not always easily recognized. Its physical signs may be confounded with those of *endocarditis*, *pleurisy*, and *cardiac hypertrophy.*

The friction murmurs of pericarditis may be distinguished from *endocardial* murmurs :—*first*, by their superficial character. *Second*, by their limited area of diffusion, their maximum area of intensity being over the right ventricle and the junction of the fourth rib with the sternum ; while endocardial murmurs are audible beyond the pericardial limits to the right and left, upward along the course of the vessels and sometimes in the back. *Third*, the intensity of a pericardial friction sound may be increased or diminished by inclining the body of the patient forward or backward, and it is rendered more distinct by a full inspiration ; whereas endocardial murmurs are not changed in intensity by a change in the position of the patient, nor by the period of time of the respiratory movement. *Fourth*, pericardial friction sounds are not necessarily synchronous with the heart sounds and may be *double;* while endocardial murmurs always precede, take the place of, or follow heart sounds. Pericardial sounds are more grating, rubbing or creaking in character than endocardial.

Pericardial friction sounds may be distinguished from the friction sounds of *pleurisy* when the pleurisy occurs over the precordial space, by directing the patient to hold his breath for a moment; if the friction sound is pericardial it will continue during the suspension of the respiratory act—if it is pleuritic the friction sound will cease during the arrest of respiration. Occasionally, however, where there is consolidation of the lung directly over the heart, accompanied by a pleuritic friction, and firm adhesions having taken place between the two surfaces of the pericardium, a distinct friction sound may be produced in the pleura by the motion of the heart. This is of rare occurrence and is hardly to be taken into consideration. In this case the general bodily condition and the state of the pulse may aid us.

The abnormal area of percussion dulness produced by *hypertrophy* or dilatation of the right ventricle very closely resembles that produced by pericardial effusion, and it is often exceedingly difficult to draw a distinct line between them. There is one point which may be regarded as diagnostic: that is, in enlargement of the right heart the precordial dulness never extends to the left beyond the apex-beat, while in pericardial effusion it may extend one or two inches beyond the apex-beat. The fact that cardiac dulness extends to the left of the apex-beat proves that there is more or less fluid in the pericardial sac. The outline of dulness is quadrilateral in dilatation, and triangular in pericardial effusion. Besides, in cardiac hypertrophy there is an increase in the force of the apex-beat, and an abnormal intensity to the heart sounds; in pericarditis both will be diminished in intensity. Pericardial effusion is distinguished from hypertrophy or dilatation of the left heart by the fact that in left cardiac hypertrophy the apex-beat is carried downward and to the left, and the area of precordial dulness is increased in the same direction and not to the right. The force of the heart's action is greatly increased in left ventricular hypertrophy.

Prognosis.—In most instances pericarditis ends in complete recovery. The exceptions to this rule are met with almost exclusively in connection with Bright's disease and septic or pyæmic conditions. In connection with either of these diseases there is always more or less danger; if it occurs in connection with pyæmia, the danger is very great, for the exudation in such cases is usually purulent and its absorption can hardly be expected, although it does occur. A large amount of fluid may compress, and cause paralysis of the heart, death resulting in a few hours. The nature of the exudation determines to a great extent the prognosis; when it is hemorrhagic or purulent, the prognosis is bad. Rheumatic pericarditis is rarely fatal. Occasionally, acute pericarditis passes into chronic, or rather is accompanied by a large serous effusion, which disappears slowly, and is especially liable to be accompanied by relapse, and thus the disease goes on for months. During its progress the patient suffers from repeated attacks of extreme dyspnœa; in rare instances a fatal syncope occurs.

As a result of the long continuance of the fluid effusion the substance of the heart becomes softened and its muscle undergoes more or less degeneration, on account of which its propelling power is diminished, and death by œdema of the lungs may occur ; or any sudden effort may result in instant death. This form of subacute or chronic pericarditis is generally associated with blood changes attended by a loss of red corpuscles and fibrin, and must always be regarded as a grave disease. The most frequent sequelæ of acute pericarditis are adhesions of the two surfaces of the pericardium, cardiac dilatation, and hypertrophy. Cardiac dilatation occurs as the result of the weakening of the cardiac walls from myocarditis. The hypertrophy of the cardiac walls which follows this dilatation is compensatory. Occasionally the pericardial exudation is abundant, and extensive pericardial adhesions take place at the base of the heart, which, by their contraction and pressure, interfere with the current of blood through the coronary arteries, and as a result the nutrition of the heart is impaired and fatty degeneration of its walls may be developed. The duration of pericarditis is from one to three weeks ; some cases end fatally in a few hours from sudden heart failure.

Treatment.—We have to deal with an inflammation of considerable severity, yet from our knowledge of its etiology and morbid anatomy we are not warranted in the use of a single antiphlogistic measure. Blood-letting, hydragogue cathartics, diuretics and blisters, which at one time were almost universally employed, are now abandoned ; the tendency is toward a supporting plan of treatment. As soon as it is discovered that pericarditis exists, endeavor to determine its cause, and, if possible, remove it ; if this is not possible, endeavor to counteract it. If the pericarditis is due to uræmia, employ those means which favor elimination of urea. If it accompanies articular rheumatism it must be treated as a rheumatic affection. In those acute diseases marked by great depression the occurrence of pericarditis is an indication for an increase in stimulants. Under all such circumstances, especially in connection with septic and pyæmic developments, supporting measures are early called for. The favorite local applications in its early stage are hot anodyne poultices over the precordial space in connection with the internal administration of opium ; absolute rest in bed must be enjoined. If the pulse exhibits dicrotism, stimulants in small quantities may be given.

Opium is the most valuable internal agent. It should never be given in large doses, but only in sufficient quantities to relieve pain and arrest or allay an irritable action of the heart. The largest doses administered should be given at night, in order that the patient may secure quiet sleep ; the heart is more liable to become irritable at night, and the patient usually becomes more restless. Great care should be exercised in the administration of opium ; it should never be carried to semi-narcotism. Chloral is thought to be equally good, since it does not *interfere with secretions ;* my experience is against its use.

The means usually employed for removal of the fluid are hydragogue cathartics. diuretics and blisters. I am convinced that this plan of treatment will not hasten, but will rather delay the removal of the fluid. Experience teaches that pericarditis is an inflammation which occurs in the weak and feeble, and not in the strong and vigorous ; it is met with among the young rather than in healthy persons in the prime of life. In almost all instances it is associated with those diseases that are especially marked by a loss of vitality; consequently all measures that have a tendency to depress the patient are to be avoided. Blisters are apt to accelerate the heart's action and should never be applied directly over the precordial space; leeches are less painful and more efficacious, and may be applied over the precordial space. The same general rules which were given as guides in promoting the absorption of the inflammatory product in pleurisy are to be followed in the treatment of pericarditis. Iron, stimulants, and a highly nutritious and readily digestible diet are the most efficient remedies. Anything which accelerates the heart's action should be avoided.

The surface of the chest must be carefully protected from changes in temperature; any exposure incident to a physical examination of the chest must be carefully avoided. The Germans advocate *cold* to the precordia; it is said to diminish pain and frequency of the heart's action. They direct that an ice-bag shall be kept over the precordial space until all evidences of pericarditis have disappeared. I cannot recommend this plan of treatment.

During the period of convalescence the patient must be very strictly guarded, for the walls of the heart are in a weakened condition, and should not be overtaxed. Everything which will have a tendency to increase the action of the heart must be carefully avoided. Children should not be allowed to go up and down stairs or to play with other children during the period of convalescence. Patients convalescing from pericarditis must be placed under the very best hygienic conditions for two or three months after the disappearance of the pericardial symptoms.

Sometimes the symptoms which attend a large fluid effusion become very urgent, and the question presents itself:—shall *aspiration of the pericardium* be performed? It has been claimed that little danger attends its performance, but it should never be rashly undertaken. When it is positively determined that pus is in the pericardium aspiration should be practised. When the effusion is sero-fibrinous it must be remembered that the urgent symptoms, for the relief of which aspiration would be resorted to, are usually of short duration, and patients rarely die from the pressure produced by the effusion. Whether aspiration shall be performed under such circumstances is a question for most careful consideration.[1]

CHRONIC PERICARDITIS.

Chronic pericarditis is rare except as a sequela of acute; occasionally it may be sub-acute from its commencement. When, after three or four weeks, acute pericarditis does not terminate in recovery, it becomes *chronic*. In some cases of chronic pericarditis the pericardial sac contains several pounds of fluid. In others firm adhesions form between the pericardial surfaces, binding them more or less closely to each other; mingled with these adhesions are chalky débris and calcareous plates. The adhesions which form in acute pericarditis are not regarded as a part of the history of chronic pericarditis. The fibrous changes of chronic pericarditis only occur when the sub-pericardial tissue is involved. The heart may then be encased in a calcareous wall, and a fibrous degeneration of the cardiac muscles result, which may lead to local aneurismal dilatation.

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[1] In a monograph by Dr. Roberts, who gives an account of sixty cases with twenty-four recoveries (forty per cent.), he states that the best points to tap are in the fossa between the ensiform and costal cartilages on the left side, or in the fifth left interspace near the junction of the sixth rib with its cartilage. In Huidenlang's case 1000 c. c. were withdrawn at two tappings and recovery followed. (Archiv. f. klin. Med. 24, p. 452.)

The **Symptoms** of chronic pericarditis are those which give evidence of obstructed circulation with signs of enlargement of the heart. This enlargement may be due to hypertrophy, but is more frequently dependent upon dilatation of the cardiac cavities. There is dyspnœa sometimes amounting to orthopnœa and uneasiness or a sense of weight in the precordial region. In some instances this condition is associated with attacks of angina pectoris. The heart's action is easily disturbed, and cardiac palpitation is present on slight physical exertion or mental excitement.

The **Physical Signs** of chronic pericarditis closely resemble those of eccentric cardiac hypertrophy; in both cases there is increased dulness in the precordial region, but in pericarditis the apex-beat is indistinct and is raised above its normal position; while in hypertrophy the apex-beat is distinct and is carried downward and to the left of its normal position. A friction murmur is usually heard even when a large amount of fluid is present. There is no murmur in hypertrophy. Bulging sometimes occurs, and there may be fluctuation when the fluid is large in amount. If the two surfaces of the pericardium are closely agglutinated, and the pericardium is adherent to the costal pleura, so that firm adhesions are formed between it and the chest wall, there will be more or less depression of the precordial region—so-called "*systolic* depression;" the cardiac impulse will be permanently displaced upwards, and will be unaltered either by change of posture or by a full inspiration, and there will be an irregular jogging motion of the heart during both its systole and diastole. Sometimes there is a depression over the scrobiculus cordis, caused by adhesion of the two layers of the pericardium to each other and to the pleura covering the diaphragm, and concomitant adhesion of the diaphragm with the liver.

Although the **Diagnosis** of chronic pericarditis is always difficult, and its existence is rarely, if ever, positively determined unless there is a large amount of fluid effusion in the pericardial sac, still, if the symptoms and physical signs already detailed follow an attack of acute pericarditis, there is presumptive evidence of its existence.

The **Prognosis** in this affection, as regards complete recovery, is always unfavorable, and when it is accompanied by degeneration of the cardiac walls, and dilatation of the cardiac cavities with or without valvular insufficiency, sudden death is liable to occur during violent physical exertion.

The **Treatment** consists in limiting physical exercise so as not to overtax the embarrassed heart; at the same time in furnishing the patient with a most nutritious but non-stimulating diet, and in administering daily some preparation of iron. When there is a large amount of effusion the same rules for the performance of paracentesis are to be followed as in acute pericarditis. The operation will be resorted to in chronic cases with better success than in acute.

ENDOCARDITIS.

Endocarditis is an inflammation of the endocardium. I shall describe it under three heads, grouping the first two together :—

I. *Acute Non-infectious Endocarditis.*
II. *Infectious or Ulcerative Endocarditis.*
III. *Interstitial Endocarditis.*

In most instances this disease depends upon a constitutional dyscrasia characterized by alterations in the vital, physical, or chemical properties of the blood. Etiologically, all the different varieties are closely connected; clinically and pathologically, they are distinct. They cannot be classified as *acute* and *chronic* in the ordinary acceptation of the terms, for some cases are at no time acute, and even in so-called chronic endocarditis the changes are but an advanced stage of an acute process.

So-called *acute endocarditis* is accompanied by a fibro-cellular exudation into the substance of and underneath the endocardium, causing elevations of its surface. A common term for this variety is "acute exudative endocarditis," it being understood that no *exudation* occurs *on* its free surface, but underneath it. This form may be entirely recovered from, or may lead to interstitial formative changes. Acute non-infectious endocarditis may become ulcerative as the result of septic infection, giving rise to changes known as *acute infectious endocarditis.*

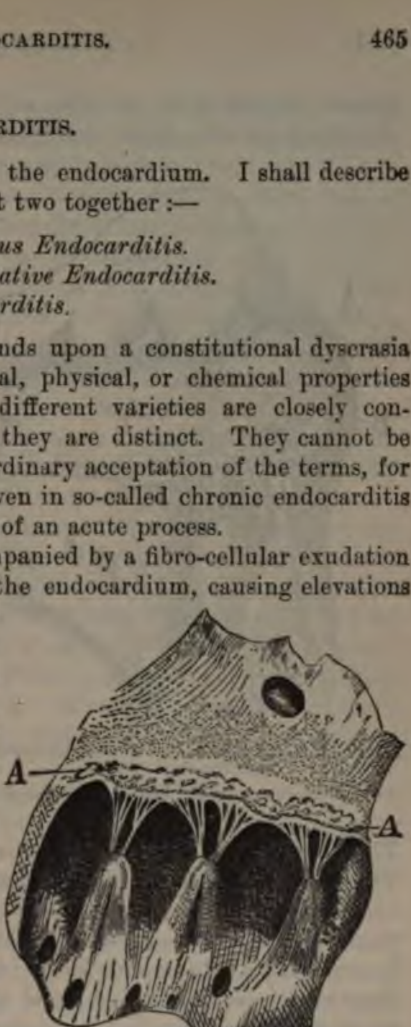

Fig. 98.
A drawing showing the changes in a case of Diphtheritic Endocarditis.
A, A. Thickening of the Endocardium on the free border of the mitral valve, with papillary elevations surmounted by fibrinous deposit.

Chronic or *interstitial* endocarditis is the form in which the endocardial and myocardial tissues are involved in sclerotic or cirrhotic changes, giving rise to the many varieties of valvular disease. It may be a sequela of the acute exudative variety, or the inflammation may have been interstitial from its commencement, for the valvular changes due to this form are often found in those who never have had either acute articular rheumatism, or acute endocarditis, but who are subjects of chronic rheumatism, syphilis, or gout.

ACUTE ENDOCARDITIS.

In adults the left, and in intra-uterine life the right, heart is oftenest affected. The process rarely extends beyond the valves and valvular orifices; but it may involve the whole or any part of the ventricular or auricular portions of the endocardium.

Morbid Anatomy.—The endocardium becomes infiltrated with cells, the process beginning in the layer of flat cells. The new formative cells are developed in the layer *underneath* the endocardium. This hyperplasia is accompanied by softening of the intercellular structure, which is soon destroyed. The endothelial elements take part in the process. The masses of new cells push out the endocardium, and papillary elevations are formed. These conical elevations are surrounded in the deeper layers of the endocardium by a zone of proliferation which is never distinctly limited, but which exhibits progressive hyperplasia from the periphery toward the centre. All these changes may have taken place in non-vascular tissue. Where the capillaries are most numerous, a punctate or arborescent vascularity is seen, after which the part becomes opaque. *There is no exudation upon the papillary elevations,* but fibrin from the blood is deposited on them as on foreign bodies. These coagula are most numerous on the surface opposed to the blood current. They have a cauliflower-like, bulbous extremity connected by a constricted neck with a firm, hard base which is continuous with the subjacent tissue. Each mass is covered by a thin hyaline layer. At first these vegetations are so small and numerous that the membrane has a granular look. Later, they enlarge, sometimes to the size of a pea, and have a conical or raspberry-like shape. They are arranged on the borders of the aortic valve near the edge, their seat being determined by the limit of the vascular network. The bands of tissue passing from the attached valvular border to the *corpus Arantii* in the centre show the granulations most distinctly. Near the insertion of the tendons upon the auricular surface of the mitral valve are irregular wreaths of vegetations enclosing attachments of the chordæ tendineæ. Friction of these vegetations or coagula upon the endocardium may excite an endocarditis at points remote from the valves. The chordæ tendineæ may adhere to one another. From these adhesions stenosis may result, by the flaps becoming agglutinated to each other, or regurgitation by their adhering to the heart-walls. As a result of these changes, new vessels appear in the substance of

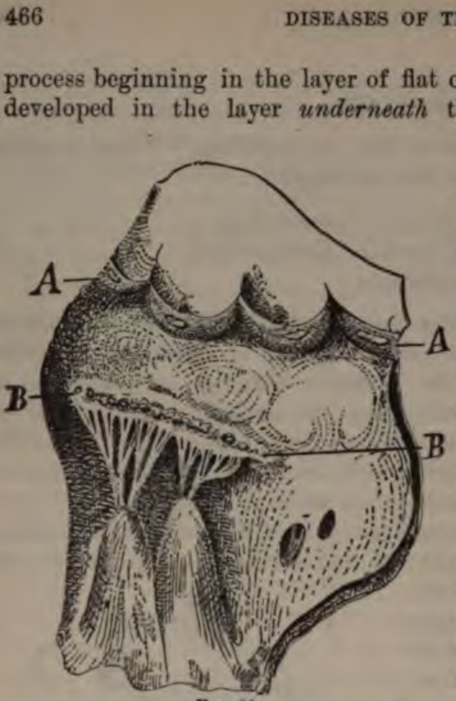

FIG. 94.

Drawing from the previous case showing similar changes upon the Aortic Valves.

A. A. Aortic, B. B. Mitral Valves showing papillæ and fibrinous deposit.

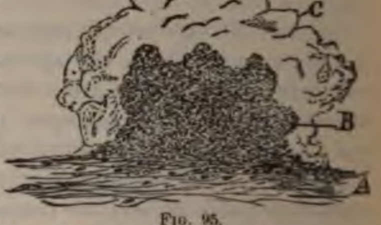

FIG. 95.

Vertical Section of an Aortic Valve in Acute Endocarditis.

A. Endocardium.
B Papillary elevation.
C. Fibrinous deposit. × 60.

the mitral valve, or existing ones become more apparent. The more rapid the course, the more marked are these changes. The largest vegetations are found on the valves. Young vegetations are translucent, soft, and friable.

Infectious or Ulcerative Endocarditis.—Necrotic changes with loss of tissue may occur in non-infectious endocarditis, but by general usage the term "*ulcerative*" has come to signify the infectious form of the disease.

It is oftenest met with in pyæmia, puerperal fever, scarlatina and diphtheria; it has been called "septic" and "diphtheritic" endocarditis. When ulceration of the endocardium is of specific origin, the primary changes are associated with the entrance of bacteria, which invade the membrane from the blood-current, and induce necrotic changes that may be well advanced before there are any distinct evidences of reactionary inflammation. White cell infiltration, however, of both the necrotic mass and the surrounding tissue, rapidly supervenes. This is followed by disintegration and sloughing of the affected portions. The margins of the valves are irregular, but well defined. The edges are swollen and thick, and their floor is infiltrated with pus. In some case the apices of the vegetations which are formed are swept off by the blood-current, and an ulcerated surface is left. If their removal causes great loss of substance, perforation of the valve may occur. These perforations are sometimes closed or hidden by a fibrinous exudation. Micrococci and other bacteria are found in ulcerative endocarditis of a septic origin, and have given to it the name of "*mycosis endocardii.*"[1]

The valvular ulcerations in this form of endocarditis may give rise to various lesions. If small masses are detached from the cardiac orifices, either from deposits on the valves or from ulcerations, and enter the blood-current, they originate morbid processes in the organs to which they are carried. It is important to distinguish between the results produced by displacements into the blood-current of *large masses*, and those arising from the entrance of *molecular* fragments. Masses from the vegetations, or from the ulcerated valves in infectious endocarditis, being stamped with a septic element, lead to the development of suppurative infarctions in different organs. The size and site of the emboli are important; they may be so large as to obstruct vessels of the largest size, as the external iliac. When arteries in the lungs are thus plugged, the result is generally an *ischæmia*, often terminating in gangrene. Capillary embolism may occur in a number of organs simultaneously.

When the cutaneous capillaries are obstructed, ecchymotic spots are formed, followed by cellulitis. If in cerebral embolism the occluded vessel is large, instantaneous hemiplegia and secondary softening will result; if it is very small, softening develops without evidence of obstructed circulation. Infarctions and suppuration of the spleen (splenitis, so called) are not uncommon. The kidneys may be similarly affected. Septic phenomena are very important. When typhoid symptoms, deep jaundice, and symptomatic intermittent fever are associated with endocarditis, it establishes its "ulcerative" character. When the inflammation develops rapidly, the valves soften, lose their resisting power, and in time become stretched, other form of endocarditis. The *spleen* is much oftener the seat of embolisms than the kidney or brain, but they may occur in any organ and give rise to characteristic local symptoms. The occurrence of hemiplegia with aphasia, or of severe cerebral disturbance, during the course of an endocarditis, is indicative of cerebral embolism.

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[1] Jaccoud. Klebs.

Physical Signs.—*Inspection* sometimes shows the area of cardiac impulse greater than normal; the impulse is irregular and often tumultuous. Later the apex-beat and the impulse grow more indistinct, but never so suddenly or so markedly as in pericarditis. In children the vessels of the neck may exhibit venous stasis.

Palpation.—At the onset of the disease, the cardiac impulse is more forceful than normal, and the heart's action is frequently irregular. Sometimes the heart thumps violently against the chest-walls. The force of the cardiac impulse varies from day to day, being stronger when pain is present. If, during the disease, there is no increase in the force of the apex-beat, we infer that the muscular power of the heart is deficient. When acute endocarditis supervenes upon long-standing valvular disease, there is alternate increase and diminution in the area and force of the impulse. When the heart walls are weakened by myocarditis, or when the endocarditis is itself very extensive, the force of the apex-beat is diminished; an endocardial thrill is often present.

Percussion.—The area of cardiac dulness is normal unless changes at the valvular orifices retard the outflow of blood from the lungs, and then the cavities in the right heart become engorged and the area of dulness will be abnormally increased. But this increase is always slight, except in those cases where sudden and extreme distention of the heart cavities results from the presence of masses of fibrin. Extensive myo- or endo-cardial inflammation may so weaken the heart that dilatation results, and then percussion will show marked increase in the area of cardiac dulness.

Auscultation reveals a murmur, or murmurs, over the various cardiac orifices. The fact that valvular disease may have pre-existed makes it important to carefully examine the heart at the first visit to one suffering with acute rheumatism, chorea, Bright's disease, etc. When hypertrophy and old valvular disease of the heart exist, the advent of an attack of acute endocarditis generally passes unrecognized, and even its presence is often undetermined. The most important constant sign of endocarditis is a *systolic murmur,* heard with greatest intensity at the apex; this soft, blowing, or "bellows" murmur may be ventricular or valvular. In all cases it is due to *roughening* or *thickening* of the endocardium. It often changes its point of maximum intensity during the acute period of the disease. It is developed early, and when one is on the lookout for endocarditis this will be the first evidence of it. In some instances *no* murmur is at any time present.

A *mitral murmur alone* occurs in fifty per cent. of cases of rheumatic endocarditis; it is developed early and is preceded by prolongation of the first sound, a "transition" sound, so to speak, feeble and wavering in character, extending over the slight interval which normally exists between the first and second sounds. Other changes, not murmurs, but which precede hem in many cases—are loud, ringing normal sounds;—muffled first ound;—feeble first, intensified second sound;—doubling of the first sound; —"roughening" of the first sound;—and a "humming" over the right heart. Complete absence of the heart sounds is a rare but possible antecedent of an endocardial murmur. A mitral murmur, in acute endocarditis, is usually audible over a limited area. It is the exception to hear it both in front and at the back; very frequently it is heard most distinctly over the stomach. When the pulmonary circulation is greatly obstructed, it causes an extra strain on the pulmonary valves, and then the second sound will be accentuated, while the first pulmonic sound may be feeble or absent. Reduplication of the second sound in mitral endocarditis is probably due to the difference in time occupied by the ventricles in emptying themselves.

A *tricuspid murmur* occurs in fifty per cent. of the cases of acute mitral endocarditis; a *pulmonic murmur* in about one-third of the cases. They are superficial and "scratchy" in character, and indicate a relaxed condition of the vessels and a thin state of the blood. They are *never* permanent. Mitral endocarditis is accompanied by *aortic* murmurs in about sixteen per cent. of the cases, and these murmurs are usually soft and blowing, but may be "musical," "whistling," or "twangy."

In *aortic endocarditis* the second sound is usually lost over the carotids. In about twelve per cent. of all cases of acute (rheumatic) endocarditis a regurgitant murmur will be heard over the *tricuspid* orifice. Tricuspid murmurs are present in fifty per cent. of all cases of recent mitral murmurs; in forty per cent. of recent aortic murmurs, and in twenty-five per cent. of mitro-aortic murmurs. They are due to an increase in the slight (normal) insufficiency of the tricuspid valves. Such murmurs are of short duration, vibrating in character, and heard over the right ventricle. In children aortic endocarditis is rare; at this period obstruction at, and regurgitation through, the mitral orifice commonly occur together.

Differential Diagnosis.—Acute *non-infectious endocarditis* may be mistaken for *pericarditis*, and its murmur may be mistaken for that produced by *aortitis* or for those friction murmurs that develop during *fevers*. The friction-sounds of *pericarditis* are superficial and limited to the precordial space, while those of endocarditis are distant, and each murmur will have its area of diffusion *beyond* the precordial space. A pericardial sound is distinctly a friction, creaking, or rubbing sound, and it has a "to-and-fro" character, while that of endocarditis is soft and blowing. Endocardial murmurs accompany the heart sounds, while pericardial friction sounds are not always rhythmical with the heart sounds. The intensity of a pericardial sound is increased when the patient bends forward, at the end of a full inspiration, or when the stethoscope is pressed firmly over the precordial region, and in the last instance it becomes "grazing" and "rubbing" in character. As soon as effusion occurs in pericarditis, alteration in the character of the pulse, increase in the area of precordial dulness, and the disappearance of adventitious sounds, will decide the diagnosis.

Aortitis has many of the symptoms of endocarditis, but in addition the pulse is more rapid, the respirations are more hurried, and pain is present in the precordial region, shooting down the spine and increased by motion. Aortitis is often accompanied by cutaneous hyperæsthesia. Acute aortitis is very rare.[1]

The *functional* murmurs which occur in *fevers* are usually heard only at the base of the heart; while those of endocarditis are most frequent and distinct at the apex. There are no signs of obstruction present with febrile murmurs, while they are frequent with endocarditis.

It is difficult to tell whether a murmur is of *old* or *recent* origin. If during an attack of rheumatism a murmur is developed under daily examination, it indicates acute endocarditis. If a murmur exists at the first examination, systolic, soft, blowing, and unaccompanied by cardiac hypertrophy, there is reason to believe that it is due to an acute endocardial inflammation; but should it be rough, diastolic, and accompanied by cardiac hypertrophy, it is probably *not* due to acute endocarditis.

Prognosis.—Acute *non-infectious endocarditis* is rarely a direct cause of death, and is seldom completely recovered from. Acute mitral endocarditis ends in permanent valvular disease in twenty-five per cent. of the cases. The prognosis is rendered unfavorable when the signs of embolism or metastasis occur. Sudden splenic enlargement, with tenderness, albuminuria, or hemiplegia, when accompanied by the physical signs of acute insufficiency or perforation of a valve, with cyanosis, dyspnœa, and disturbance of the cardiac rhythm, will render the prognosis bad. All these symptoms suggest infectious endocarditis, so that when the symptoms of this disease appear during the course of septic diseases, the liability to its occurrence must be borne in mind. Typhoid symptoms in acute endocarditis render the prognosis unfavorable. In children bronchitis, lobular pneumonia, and intercurrent diarrhœa may cause death. It may result from acute insufficiency of the heart.

In *cardiac aneurism* death may result from rupture of the sac, apoplexy, or from secondary disease in organs.

Treatment.—The treatment of both varieties of acute endocarditis must be determined by the conditions under which they occur. The patient must have absolute rest in bed in a room whose temperature is never below 70° to 75° F. The chest should be covered with flannel, and during the physical examination it should be exposed as little as possible. Some claim that an ice-bag over the heart during the acute period will arrest or limit the inflammation, but my own experience does not sustain this statement. In rheumatic endocarditis anti-rheumatic remedies are indicated. The joints must be kept absolutely at rest in the most comfortable position, and the pain relieved. If the urine is kept alkaline, the liability to endocarditis is diminished. To ensure rest, small doses of opium may be given, but opium cannot be administered as freely as in pericarditis. The patient's strength must be sustained by the judicious use of concentrated nutriment with some preparation of iron.

When endocarditis, accompanied by typhoid symptoms, occurs with septic lesions, alcohol, quinine, and iron must be freely administered; when it complicates Bright's disease, the rapid elimination of urea must be established. The pain over the precordium is often relieved by the application of a few leeches. The (internal) use of mercury, with the external application of blue ointment to "lessen the plasticity of the blood," and even the use of iodide of potassium—"for the absorption of the fibrinous exudation"—are harmful, and the theory of their use has no foundation. A single case of recovery from infectious endocarditis has been reported, in which the treatment consisted in the use of sulpho-carbolate of sodium and inunctions with carbolized oil.

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[1] Lebert and Rindfleisch doubt its existence.

INTERSTITIAL ENDOCARDITIS.

Kreizing first traced the relationship between chronic valvular diseases of the heart and interstitial endocarditis.

Morbid Anatomy.—Interstitial (or chronic) endocarditis may be a sequela of the acute, or it may be interstitial from its commencement, and be so insidiously evolved as to escape notice. Sometimes its lesions are confined to the edges or base of the valves; at others the entire valve may be involved.

The affected valves may be *thickened, indurated, contracted, adherent,* or *degenerated.* It is more closely allied to rheumatism, gout, and chronic interstitial changes in other organs than either of the other varieties; no part of the endocardium is exempt from interstitial changes, but the endocardium over the valves and that at the apex of the left ventricle are its favorite sites. The mitral valves may become three or four times thicker than normal. Sometimes their functional activity is unaffected even after they have undergone extensive pathological changes. White, thickened, opaque spots, the results of interstitial endocarditis, are often found irregularly scattered over the internal wall of the heart cavities. When vegetations are developed in interstitial endocarditis, they differ from those of the acute form, for they are firmer and less prominent, and rest upon an indurated base. In, and underneath, the endocardium there is tissue-increase, and fibrin is deposited on any prominence of the endocardium. These deposits are of various forms, and may extend for one-half an inch or more into adjacent vessels or cavities. They are usually globular or wart-like in form, and are situated on the ventricular surface of the aortic, and upon the auricular surface of the mitral and tricuspid valves.

A *microscopical examination* of a cross section of an indurated valve shows flat cells arranged in irregular layers, having between them a fibrinous material, which has in it, here and there, a few elastic fibres. The new formation always originates in the layer of *flat cells.* These changes are best marked in the fibrous zone of the valvular orifices, upon the surfaces of the valves, and in the chordæ tendineæ. After a time the new tissue becomes organized, and contracts, and this contraction is progressive. Gradually the rigid valves, whose edges are rounded and hard, are drawn together toward their base, and thus assume a puckered appearance. Similar processes in the chordæ tendineæ cause them to hypertrophy, become rigid and shortened. In this way the valves are diminished in depth, and sometimes their free edges become approximated to the cardiac walls, so that extensive valvular insufficiency is the result. This does not always happen; for a thickened cartilaginous valve may have so much fibrinous or papillary growth upon it, that the inward current is obstructed, and stenosis results without insufficiency. As this thickening and rigidity increase, the mobility of the valvular flaps is diminished, and adhesions occur between their edges, beginning at their base and extending toward their apex. So adherent may they become that all evidences of a valvular outline is lost and a fibrous diaphragm is stretched across the orifice, having only a small slit at its centre, looking and feeling like a button-hole, hence the term "*button-hole slit.*" The mitral opening, which normally will admit the ends of three fingers, may be so narrowed that the end of the little finger will scarcely pass through it, and the aortic opening may not even admit a small quill. These retractions and adhesions cause the mitral valves with their columns and cords, to assume the form of a perforated cone.

Long gelatinous vegetations on the aortic valve sometimes form adhesions with the aortic walls, and thus a sudden and extensive regurgitation is induced. Insufficiency and stenosis are often found at the same valvular orifice as the result of the valvular thickening, adhesion, and retraction. Such changes at the aortic orifice usually occur after middle life, and cause more thickening, adhesion, and retraction than those at the mitral valve. In children and early adult life, the mitral valves are the most frequent seat of interstitial endocarditis. The tendency of this lowly organized tissue is to undergo fatty and calcareous changes. The minute patches of fatty degeneration in the imperfectly organized tissue underneath the endocardium sometimes form atheromatous masses, containing more or less granular débris. The endocardium over these patches may be destroyed, or they may soften, ulcerate, and cause extensive destruction of the valves.

A valvular aneurism may form in the same manner as has been described in ulcerative endocarditis. The formation of calcareous granules and plates is a very frequent termination of interstitial endocarditis. The aortic orifice is the most frequent seat of these calcareous degenerations. So extensive may this process become that little beads of chalky material are seen studding the free edges of the valve and even extending into the cardiac cavities.

When interstitial endocarditis has its seat in the endocardium of the heart cavities, it will undergo changes similar to those of the valves, and the muscular walls of the heart will become the seat of interstitial changes. As a result the walls of the heart become thin and less resistant than normal, and depressions occur on its inner surface. The process is a fibrous overgrowth, which occurs in spots varying in size from one-half an inch to one inch in diameter. When it extends through the entire heart-wall, the columns and cords may be so shortened as to cause valvular insufficiency. If the cardiac walls yield to the internal blood pressure, a well-defined pouch is produced. This condition is called "*aneurism of the heart,*" and is usually situated at the apex of the left ventricle ; the pouch may be as large as the closed fist, and may communicate with the ventricle by a funnel-shaped or ring-like aperture. The walls of the sac are firm and rigid, the internal surface is generally smooth, but it may be irregular, in which case clots adhere to its walls. Cardiac muscular fibres are found in the walls of the aneurismal sac. Aneurisms at the base and in the intervenlished. The pain over the precordium is often relieved by the application of a few leeches. The (internal) use of mercury, with the external application of blue ointment to "lessen the plasticity of the blood," and even the use of iodide of potassium—"for the absorption of the fibrinous exudation"—are harmful, and the theory of their use has no foundation. A single case of recovery from infectious endocarditis has been reported, in which the treatment consisted in the use of sulpho-carbolate of sodium and inunctions with carbolized oil.

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At the end of a cardiac diastole all the heart cavities are filling; just before the cardiac systole, blood is forced from the lungs and cavæ through the auricles and ventricles, while the mitral and tricuspid valves are pressed against the ventricular walls, thus offering no obstruction to the blood current. Should any obstruction exist at either of the auriculo-ventricular orifices, the blood while passing through the opening will impinge *on* such obstruction and cause a *presystolic* murmur. During a cardiac systole, the filled ventricles contract and blood is thrown through the arterial openings, the flaps of whose valves are pressed against the walls of the vessels so that no obstruction is offered to the outgoing current. At the same instant, the auriculo-ventricular valves close their orifices, so that blood may not flow back into the auricles. If the semilunar valves obstruct the outgoing current, or if the mitral or tricuspid valves do not wholly close the auriculo-ventricular orifices, then, in the one case, the blood-current as it passes over the obstruction at the semilunar orifices, will produce a *systolic* murmur, and in the other a systolic murmur will be produced by the backward current through the abnormal opening at the auriculo-ventricular orifices. If the pulmonary and aortic system—which are filled at the systole—have back of them a semilunar valve that does not completely close that end of the circuit, the blood will regurgitate into the ventricles during the period of cardiac rest, so that semilunar incompetence causes a *diastolic* murmur.

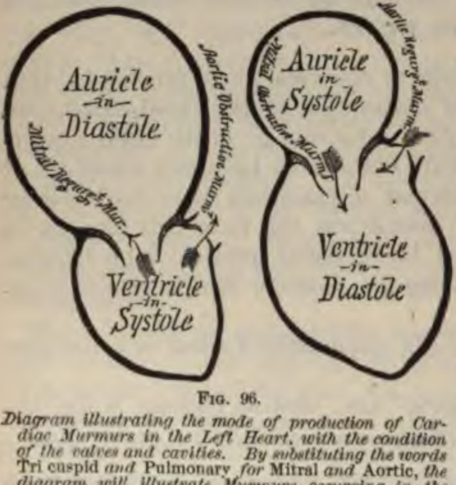

FIG. 96.

Diagram illustrating the mode of production of Cardiac Murmurs in the Left Heart, with the condition of the valves and cavities. By substituting the words Tri cuspid *and* Pulmonary *for* Mitral *and* Aortic, *the diagram will illustrate Murmurs occurring in the Right Heart.*

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[1] The factors that determine the character of a murmur—its pitch, quality and intensity—are physical, as the force with which the jet is propelled, and the physical properties of the media of conveyance; and they are the same as those which determine the quality of other sounds.

VALVULAR MURMURS.

RHYTHM.	SITUATION.	ORIFICE.	NATURE.
Systolic, 1	Basic.	Aortic.	Obstructive.
2	"	Pulmonary.	"
3	Apical.	Mitral.	Regurgitant.
4	"	Tricuspid.	"
Diastolic, 1	Basic.	Aortic.	"
Presystolic, 1	Apical.	Mitral.	Obstructive.

Pulmonary (diastolic) regurgitant murmurs and tricuspid (presystolic) obstructive murmurs are so rare, clinically, that they may be disregarded.

The following is the order of *relative frequency* of cardiac murmurs: (1) mitral regurgitation; (2) aortic obstruction; (3) aortic regurgitation; (4) mitral obstruction; (5) tricuspid regurgitation; (6) tricuspid obstruction; (7) pulmonary obstruction; and (8) pulmonary regurgitation.

The most frequent *combinations* of murmurs are: (1) aortic obstruction and regurgitation; (2) mitral obstruction and regurgitation; (3) mitral obstruction and tricuspid regurgitation; (4) aortic obstruction and mitral regurgitation; (5) double valvular disease at aortic and mitral orifices (four murmurs).

Having appreciated the existence of a cardiac murmur, it is often very difficult to determine its *rhythm*. This difficulty may be lessened by remembering that the first sound of the heart is synchronous with the carotid and radial pulse and the apex-beat, and that it may be wholly replaced by a systolic murmur; the second sound is, however, almost always heard, for the pulmonic and aortic valves are rarely diseased at the same time.

After determining the rhythm, pitch, intensity, and quality of a cardiac murmur, we next find the point of its *maximum intensity*. Murmurs arising at the *mitral* valve are loudest at the apex of the heart, or *just above it; tricuspid* murmurs are loudest over the lower part of the sternum; *pulmonary* murmurs, in the second left intercostal space close to the sternum, and *aortic* murmurs in the second right intercostal space at the edge of the sternum.

Valvular diseases, causing murmurs, consist in a condition of the valves allowing either of regurgitation or obstruction.

Valvular insufficiency results when extensive retraction, perforation, or partial detachment of the valves prevents them from completely closing their respective orifices; or when the chordæ tendineæ have been ruptured, or calcareous degeneration has made the valves rigid, the backward current in such conditions giving rise to a *regurgitant murmur*.

When the valves are thickened, retracted, adherent, hypertrophied, or degenerated, they obstruct the outward current of blood and give rise to *obstructive murmurs*.[1] Both conditions, viz., stenosis and insufficiency, are often found co-existing, but rarely to the same extent. The lesions which induce these murmurs are *acute*, when they occur during the course of acute endocarditis, and *chronic* when they depend upon the presence of some firm tissue, such as connective, fibroid, calcareous, or atheromatous tissue, which alters the form and impairs the function of the valves. Both the above varieties may produce the same murmurs.

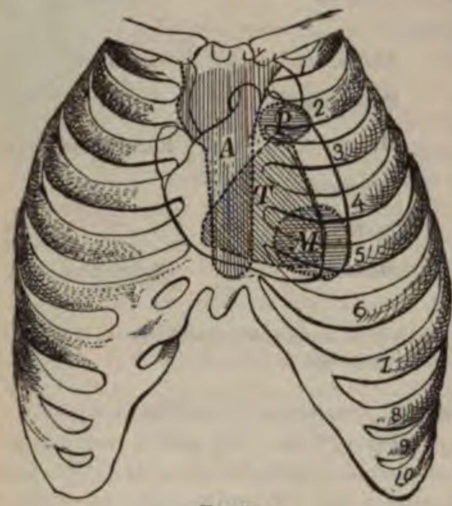

Fig. 97.

Diagram showing the Areas of Cardiac Murmurs.

A. Area of Aortic Murmurs; M. Mitral; T. Tricuspid; P. Pulmonary.

Since physical signs are here the most important factors in diagnosis, the normal (physical) relation of the heart must be borne in mind: the apex of the heart is normally felt between the fifth and sixth ribs on the left side, about two inches below the nipple and one inch to its sternal side. The highest part of the base of the heart is on a level with the third costal cartilage. The tricuspid orifice is situated at the junction of the fourth left costal cartilage with the sternum. The mitral orifice is to the left of the tricuspid, immediately behind the left border of the sternum, at the junction of the third costal cartilage with that bone. The aortic orifice is one-half an inch lower than and to the right of the pulmonary orifice, behind the sternum on a level with the third interspace. The tricuspid orifice is the most superficial, then the pulmonary, next the aortic, and deepest of all the mitral. Ranged from *above downwards*, the pulmonary orifice comes first, then the aortic, then the mitral, lastly the tricuspid.

AORTIC OBSTRUCTION, OR STENOSIS.

This is a common cardiac lesion, and is always accompanied by more or less hypertrophy of the left ventricle.

Morbid Anatomy.—The valves will be found to present some or all of the changes described in the history of interstitial endocarditis, together with degenerative changes due to atheromatous, calcareous, fibroid, fatty, or connective-tissue metamorphosis; they may be covered with thick, warty, irregular excrescences, that cause loud murmurs and yet do not seriously obstruct the out-going blood-current. Or the aortic orifice may be almost completely occluded, and then the extent of the lesion is measured more by the resulting hypertrophy and its effects on the systemic circulation, than by the loudness or harshness of the murmur. The valves are often so rigid that they cannot be pressed back, and then they present greater obstruction to the outgoing current than when vegetations exist; as the result of adhesions, the valves may become fused into a mass, so that they project into the blood-stream in the form of a funnel, irregular in shape and studded with calcareous nodules. The line of attachment of the valves to the aorta frequently becomes obliterated.

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[1] Some call obstructive murmurs *direct*; and regurgitant murmurs *indirect*, from the current that causes the sound.

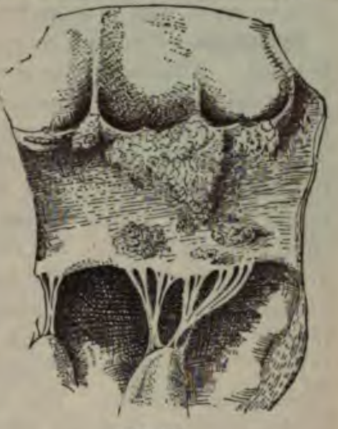
FIG. 98.
Vegetations on the Aortic Valves giving rise to Aortic Obstruction.

Aortic stenosis is frequently accompanied by atheromatous changes in the aorta, called "*Arteritis deformans.*" As a result of aortic stenosis, hypertrophy of the left ventricle occurs, which is gradual in its development and called "compensatory" hypertrophy, because it is due to the increased force required to propel the blood through the constricted orifice. Mitral insufficiency is apt to occur later, either from extension of the inflammation from the aortic valves, or from forcible pressure of blood upon the ventricular surface of the mitral flaps. Slight thickening and roughening of the aortic valves lead to no serious results.

Etiology.—Aortic stenosis is most frequently met in middle and advanced life; the mean age being forty-seven years. It is occasionally met with in children under two years of age. It may be the result of defective aortic development and perhaps of imperfect development of the trachea, causing imperfect expansion of the chest.[1] Interstitial endocarditis of *rheumatic* origin is its most frequent cause. Chorea and chronic Bright's disease may cause it. Atheroma or arteritis deformans extending to the valves sometimes gives rise to it. Increased aortic tension indirectly causes aortic stenosis.[2] Men suffer from aortic stenosis oftener than women, for in them the valves are subject to greater tension, and hence non-rheumatic aortic valvular disease is common in men and rare in women. Occupations that involve repeated sudden and severe muscular effort induce it. In old age, the aortic walls are weakened, and when aortic disease is met with in the young, it is often the result of premature vascular senility.[3] Disease of the aortic valves is oftener non-rheumatic in origin than mitral lesions. It is slower in its development, and is more frequently met with in advanced life.

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[1] Guy's Hosp. Reports, S. I., vol. vi., p. 235.

[2] The coexistence of cardiac valvular disease and cancer is a remarkable coincidence, possibly with a causal relation.

[3] Dr. Allbutt says that in Leeds quite young men have aortic valvular disease; and Dr. Peacock mentions several cases where it has occurred in young girls who have been placed at service before they were fully developed. Corvisart and Virchow both admit the possibility of syphilis being a cause of aortic valvular disease, but clinically this is not yet proven.

Symptoms.—The subjective symptoms of aortic stenosis are rarely well marked. Although extensive, it may cause no discomfort, for as the obstruction increases, compensatory hypertrophy prevents pulmonary congestion; but when this no longer compensates for the obstruction, the arteries are inadequately filled, the left auricle cannot empty itself, and consequently the pulmonary vessels and the venous system are abnormally full. The scanty arterial supply causes pallor of the face, and syncope may occur from cerebral anæmia, but these are late symptoms, not usually appearing until after the mitral valve has become secondarily involved. The pulse is normal in frequency, diminished in volume and fulness, and, as a rule, regular in rhythm, though it may be intermittent, compressible, and "jerky" in character. Signs of arterial anæmia usually precede those of venous engorgement. The sphygmograph gives a slanting or oblique upstroke, showing that the influence of percussion is lost, and the tracings may show considerable separation between the "percussion" and "tidal" waves. The pulse is rarely slowed. There may be slight palpitation and paroxysmal pain in the chest.

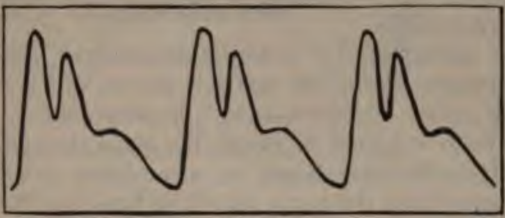

FIG. 99.

Sphygmographic tracing in a case of Aortic Obstruction, with marked separation of the percussion and tidal waves.

Aortic stenosis is more often associated with cerebral embolism than any other valvular lesion, and the splenic and renal vessels are frequently the seat of emboli. The left middle cerebral artery is the most common seat of cardiac emboli; and the left lower limb is more subject to embolism from aortic valvular disease than the right. Embolism may be due to small auricular or ventricular clots that form behind the obstruction; such clots have occluded the aortic orifice and caused sudden death.[1]

Physical Signs.—The physical signs of aortic stenosis are usually distinctive and easily appreciated.

Inspection shows the area of cardiac impulse to be abnormally increased. Very extensive increase of this area is often accompanied by lifting of the chest over the precordial region.

Palpation.—The impulse is felt to be forcible, and may be accompanied by a heaving or lifting sensation. The apex is displaced to the left and slightly downward. An indistinct thrilling sensation is often imparted to the hand during the systole. This systolic *fremissement* is nothing more than an intensified endocardial thrill, and it generally radiates to the ensiform process, being most intense in the second right intercostal space.

Percussion.—The area of cardiac dulness increases in proportion to the displacement of the apex beat to the left.

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[1] Path. Trans., vol. ix., p. 9.

Auscultation.—Aortic obstructive murmurs are loudest and most distinct at the second right intercostal space and at the sternal insertion of the third left costal cartilage. They are systolic, and oftener accompany, than replace the first sound of the heart. The maximum intensity of this murmur is at the second sterno-costal articulation of the right side, but it may be heard with equal intensity over the whole upper part of the sternum, and may be audible at the xiphoid cartilage. It is always a harsh murmur, heard most distinctly at the commencement of the systole. In uncomplicated aortic stenosis, the aortic second sound may be inaudible; it is always feeble, but the pulmonic second sound is always audible. The area of diffusion of this murmur follows the law that a murmur is propagated in the direction of the blood-current. It is conveyed along the aorta into the carotids, and one of its characteristics is that it is heard in the great vessels of the neck. It may be heard in the thoracic and abdominal aorta. When an aortic obstructive murmur is heard at the apex, its intensity is diminished, and when heard behind it is most distinct at the left of the third and fourth dorsal vertebræ, near their spines, and frequently extends downward along the spine in the course of the aorta, but with diminished intensity. It is to be noted that a systolic murmur, audible at the base and traceable along the ascending arch toward the end of the right clavicle, is by no means limited to cases of aortic stenosis, although this lesion always produces a murmur with these characteristics. When the mitral or tricuspid valves are thickened or incompetent, or when the myocardium undergoes fatty degeneration, this murmur will entirely replace the first sound of the heart.

Differential Diagnosis.—Aortic obstruction may be mistaken for *mitral* and *tricuspid regurgitation,* an *anæmic bruit,* or the *murmur of a thoracic aneurism.* Both mitral and tricuspid regurgitation and aortic stenosis produce a systolic murmur. The murmur of aortic stenosis is heard with its maximum intensity at the third left sterno-costal articulation, and diminishes in intensity toward the apex of the heart. The murmur of *mitral regurgitation* is heard loudest at the apex-beat. The murmur of aortic stenosis is conveyed into the vessels of the neck; that of mitral regurgitation to the left, in the direction of the apex-beat, and is heard behind, between the fifth and eighth dorsal vertebræ, at the left of the spine, with very nearly the same intensity as at the apex. The pulse in aortic stenosis is normal in frequency, diminished in volume and fulness, and, as a rule, regular in rhythm, though it may be intermittent, compressible, and "jerky" in character; while in mitral regurgitation it is irregular in rhythm and force, and is easily increased in frequency. Gastric, intestinal, renal, hepatic, and bronchial symptoms are present in mitral regurgitation, while the subjective symptoms of aortic obstruction are cerebral in character. The pulmonic second sound is feeble in aortic stenosis, but in mitral regurgitation it is intensified. The murmur of aortic stenosis is *harsh,* that of mitral regurgitation *soft,* and often musical.

Tricuspid regurgitation is accompanied by a systolic murmur which is rarely heard *above* the third rib; while that of aortic stenosis has its point of maximum intensity at the right second sterno-costal articulation. Tricuspid regurgitation is accompanied by jugular pulsation; while the murmur of aortic obstruction is heard in the arteries of the neck.[1] The area of transmission of tricuspid regurgitant murmurs is not more than two inches from the point of their maximum intensity, while aortic stenotic murmurs are conveyed into the vessels of the neck. The pulse in tricuspid disease is normal; in aortic stenosis it is hard and wiry.

Anæmia produces a murmur heard loudest in the carotids and accompanied by a venous hum, which is continuous and best heard on the right side of the neck. Thus, in anæmia there are three murmurs: cardiac, venous, and arterial. In aortic disease the point of maximum intensity and the absence of a "venous hum" will aid in the diagnosis; besides, there will be cardiac hypertrophy and an increase in the force of the apex-beat, while the impulse is feeble in anæmia. The murmur is soft and blowing in anæmia, and harsh in aortic obstruction. The pulse is characteristic in aortic stenosis, in anæmia it may have a *thrill*, but is never hard and wiry. The etiology and subjective symptoms of these two are strikingly dissimilar.

In *thoracic aneurism* the dilating impulse on palpation, the normal force of the heart-beat, the single and double bruit, and the pain are all important signs, which are absent in aortic stenosis.

The prognosis and treatment of "valvular diseases of the heart" will be considered at the end of their history.

AORTIC INSUFFICIENCY, OR REGURGITATION.

This is an abnormal condition of the aortic valves, which prevents their complete closure, and allows a backward current of blood to flow from the aorta into the left ventricle during its diastole. It is usually associated with more or less aortic stenosis.

Morbid Anatomy.—In aortic insufficiency, the flaps of the valves may be thickened, puckered, or shortened, so that they do not meet. If the centre of a valve is indurated, it will curl up, either toward the orifice or back against the aortic wall. In the former case, there is insufficiency with great obstruction; in the latter, insufficiency with only slight obstruction. This valvular thickening or shortening may be due to endocarditis. In some cases, the flaps of the valves may become adherent to the walls of the aorta, or a diseased valve may be torn or ruptured, which will allow a free opening for the regurgitant blood. Following stenosis, little tunnels may form by the side of the valves and permit of a regurgitant current. The aortic valves are more liable to laceration than any other valves. In aortic regurgitation, during a cardiac diastole,

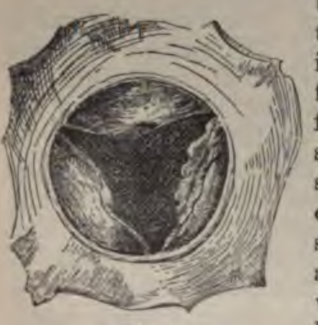

FIG. 100.

View of Aortic Semilunar Valves from above, showing insufficiency, the valves are thickened, shortened and curled upward, preventing the complete closure of the aortic orifice.

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[1] To distinguish between intrinsic pulsation of the jugular vein and throbbing of the carotids, press lightly on the vein above the clavicle; this arrests pulsation when due to tricuspid disease, while if due to aortic stenosis, the result is negative.

there is added to the blood, which normally flows from the auricle into the ventricle, a regurgitant current from the aorta, and so over-distention of the left ventricle results. Thus, after a time, the left ventricle becomes permanently dilated. To overcome this distention compensatory hypertrophy takes place. The left heart is often greatly enlarged. As a result, the arterial system is over-distended at each cardiac systole.

The extra ventricular power and the abnormal quantity of blood thrown against the arterial walls lead to endarteritis and subsequent atheroma, and the degeneration of the vessels predisposes to apoplexy and to aneurism. Since, normally, aortic recoil fills the coronary vessels, aortic regurgitation must be followed by imperfect blood-supply to the heart, and dilatation again commences at the expense of the walls of the heart, the hypertrophy ceasing to compensate for the increased dilatation. Atrophy of the papillary muscles may allow the mitral flaps to pass beyond their normal line at the auricles, when there is an increase in blood pressure, and then mitral regurgitation and impeded venous circulation will result. Passive pulmonary hyperæmia may be present *without* mitral lesions, when the left auricle cannot wholly empty itself.

Etiology.—This is similar to that of aortic stenosis. Rheumatic endocarditis is its chief source; but it may follow sudden and violent muscular effort, atheroma of the aorta or endarteritis. Congenital malformation, according to Virchow, is a frequent cause in chlorotic females. The atheroma which causes aortic insufficiency is often of gouty origin, especially when gouty kidneys coexist or when alcoholismus is associated with a gouty diathesis. Dilatation of the aorta at its origin may induce it. Fagge says only fifty per cent. of the cases of aortic insufficiency give a rheumatic history. The violence with which the valves are closed during prolonged and violent physical exertion may induce an interstitial endocarditis which will lead to it.

Symptoms.—So long as hypertrophy compensates for the regurgitation, there is little or no inconvenience experienced by the patient, even though the regurgitation is extensive. When the regurgitant stream is small, there is no disturbance of the general health, but in time the hypertrophy induces excessive heart-action during excitement or violent muscular effort. The heart-action then becomes labored and the patient is anxious, nervous and fretful, and knows well that exercise will augment his uncomfortable symptoms. The respirations are accelerated with the cardiac palpitation; as the disease advances attacks of headache and vertigo become more and more prolonged and severe; the patient complains of muscæ volitantes, dyspnœa, and giddiness, and is compelled to sleep with his head elevated. Palpitation and a visible carotid impulse are now constantly present. A comparatively frequent symptom is a distinctly paroxysmal shooting or stabbing pain over the heart, in the left shoulder, or extending down the left arm. This pain may be accompanied by numbness and a peculiar whiteness of the skin along the line of pain. In other cases, the pain passes from the middle of the sternum down the right arm. This pain is increased by excitement, physical exercise, and over-distention in the carotids, it is because preceded by a systolic murmur which has its maximum intensity in the "aortic area." Such a murmur indicates much more obstruction than regurgitation. When a diastolic murmur is heard distinctly in the carotids, and is also preceded by a systolic murmur in them, the combination indicates trifling obstruction with considerable incompetence.

Differential Diagnosis.—The diagnosis of aortic regurgitation is generally not difficult, as it rests almost exclusively upon the existence or non-existence of a diastolic murmur. It may be mistaken for *aortic stenosis*, *mitral obstruction*, *pericarditis localized* over the *aorta*, *aneurism* of that portion of the aorta immediately above the valves, *patency* of the *ductus arteriosus*, *insufficiency* of the *pulmonic valves*, and occasionally for a *rough* and *inelastic* condition of the *ascending aorta*.

Mitral obstruction gives a presystolic murmur, while aortic reflux produces a diastolic murmur. Mitral stenosis is accompanied by *no hypertrophy or dilatation of the left ventricle;* whereas these conditions are *always* present in aortic reflux. The quality of a presystolic mitral murmur is harsh and rough, and it has a churning, blubbering or grinding character; while aortic reflux has a murmur of low pitch, and a soft, blowing or musical character. Mitral stenosis is accompanied by a purring thrill, which is absent in aortic regurgitation. The murmur of mitral stenosis is the longest of all cardiac murmurs, and is never heard behind; whereas that of aortic regurgitation is heard at the sides of the chest and along the spinal column. Finally, mitral stenosis is attended by well-marked pulmonary symptoms during active physical exertion, which are rarely present in aortic insufficiency.

A pericardial friction sound over the aorta has its maximum intensity over the seat of its production, and is usually audible during both the cardiac systole and diastole. In aortic regurgitation, the character of the pulse, the existence of hypertrophy and dilatation of the left ventricle, and the carotid pulsation will establish the diagnosis.

An *aneurism* at the sinuses of Valsalva is diagnosticated by the history of the case, the presence of the murmur over the pulmonary artery, the evidence of arterial degeneration, the absence of left ventricular dilatation and hypertrophy, and by the peculiar jerking pulse. An aneurismal murmur is circumscribed, has a booming quality, is usually systolic in rhythm, and is never transmitted to the apex of the heart.

Patency of the ductus arteriosus is a rare condition; in a case where it was diagnosticated[1] the murmur was audible at the *left* of the sternum, was not everywhere continuous with the second sound, was only transmitted very feebly to the *left*, and had a wavy character sufficient of itself to distinguish it from aortic regurgitation.

Insufficiency of the pulmonic semilunar valves is the rarest of all valvular lesions; the murmur should be diastolic, having its maximum intensity in the second intercostal space of the left side, it would be transmitted only downward and toward the right apex, and would *not* be attended by arterial pulsation, a jerking pulse, or left ventricular dilatation and hypertrophy.

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[1] Guy's Hosp. Rep. Series 3, vol. xviii., 1872-3.

A *diastolic murmur in the ascending arch* due to roughening, rigidity, and dilatation of the artery is also rare, while the condition, which some say can produce it, is very common. Two cases are recorded in which the diagnosis rested upon the character of the pulse, throbbing of the arteries and the absence of left ventricular hypertrophy and dilatation.[1]

MITRAL STENOSIS.

Stenosis or obstruction of the auriculo-ventricular opening of the left heart, is due partly to constriction at the base of the mitral valves, and partly to adhesion of the valve tips or chordæ tendineæ. It usually occurs as a consequence of rheumatic endocarditis,—rarely of atheromatous degeneration,—and is most likely to occur in endocarditis affecting young persons. Usually, insufficiency and stenosis of the mitral orifice occur together, and stenosis probably never occurs without some insufficiency.

Morbid Anatomy.—As a result of acute exudative or interstitial endocarditis, the valves are rendered shorter and narrower, as well as thicker and more cartilaginous than normal. These rigid valvular projections not only obstruct the flow of blood from the auricle into the ventricle, but allow of its regurgitation from the ventricle into the auricle. In mitral stenosis, there is not only thickening and contraction of the valves, but the valve-tips or the chordæ tendineæ become adherent and sometimes each papillary muscle is changed into a corrugated, cylindrical mass, pierced with one or more slits, indicating the chordæ of which it was originally made up. The wall of the valve, especially toward its free edge, is greatly thickened, and these thickened portions are so dense that they have a distinct cartilaginous feel. On the valvular flaps that have undergone this sclerotic change calcareous masses are very frequently developed, and calcareous nodules are especially liable to form when a gouty diathesis exists. When the chordæ tendineæ and papillary muscles have become adherent, the edges of the valves are drawn down toward the apex of the heart; and since the flaps are adherent at a greater or less distance upward from their base, the valve presents a funnel-shaped appearance with its base looking toward the auricle, and its apex toward the ventricle, whose smaller opening, rarely circular, usually resembles a slit whose axis runs with the line which unites

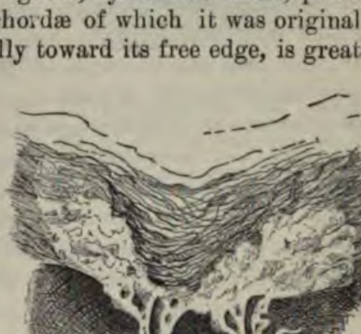

FIG. 102.

View of the Mitral Valve in a case of Mitral Stenosis. The chordæ tendineæ are thickened and shortened, and the edges of the valve are calcified and drawn downward, giving the funnel-shaped appearance.

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[1] Bellingham, Dis. of Heart, 1875, p. 152. Also Trans. Path. Society, vol. iii., Mar., 1868, p. 3. Article by Prof. Law.

the original segments of the valve. This "button-hole" slit may scarcely admit the tip of the little finger, while the *normal* mitral orifice permits the easy introduction of three fingers. Annular (ring-like) stenosis is far more common at the mitral than at the aortic orifice. Sometimes the funnel-shaped appearance is wanting, and the flaps are stretched horizontally across, with a small opening in the centre, like a diaphragm; looked at from the auricle, this slit often appears crescentic.

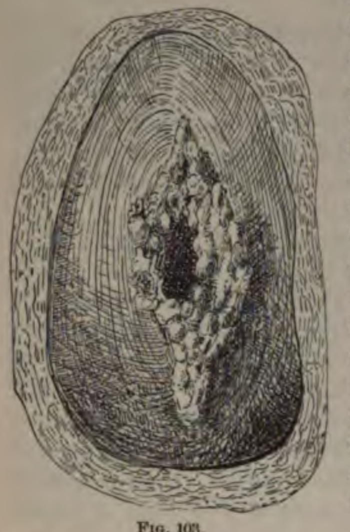

FIG. 103.

View of the Mitral orifice from the Auricle, with calcification of the valves and reduction of the opening. In the above case the point of the little finger was barely admitted through the "button-hole" slit. The valves are stretched horizontally across.

In cases of long standing the vegetations may become calcified. If the new tissue in the diseased valves undergoes fatty change and softens, ulcerative processes are set up and the chordæ tendineæ may rupture. On the floor of such ulcers calcareous masses and débris are frequently found. Dr Hayden thinks that "all funnel-shaped mitral stenosis is the result of primary acute inflammation of the valve segments with cohesions of their adjacent edges." Out of sixty-two cases of mitral stenosis, fifty-nine assumed the "button-hole" form and *three* only the funnel shaped.[1] In rare instances the tendons will adhere to the wall of the heart. Adjacent to the valves, the endocardium will usually be found slightly thickened. The valves presenting the roughest and most irregular surfaces do not give rise to the harshest or loudest murmurs.

The following changes are developed in the heart and vessels as a result of mitral stenosis. The left ventricle becomes smaller, sometimes its walls are thinner than normal. The aorta is also small and thin-walled. An almost necessary result of mitral stenosis is dilatation with subsequent hypertrophy of the left auricle. Sometimes the auricular cavity is enormously dilated, and its appendix is elongated and curved. Not infrequently the left auricular walls are from one eighth to one-seventh of an inch in thickness As soon as the auricular hypertrophy ceases to be compensatory, the pulmonary circulation becomes obstructed, causing tension in, and distention of, the pulmonary vessels. The walls of the pulmonary vessels, especially those of the main trunk, are thickened and hypertrophied; they have been found *twice the thickness of those of the aorta.* Although mitral stenosis is a disease of youth, and atheroma one of old age, yet it not infrequently happens that, even before the age of puberty, atheromatous degeneration occurs in the pulmonary vessels, especially in the small branches, as a result of the increased blood tension in the pulmonary system.[2]

The passive pulmonary hyperæmia which results from the obstructed pulmonary circulation may lead to changes which collectively constitute *brown induration* of the lung. Another occasional occurrence directly due to extensive mitral stenosis is nodular hemorrhagic infarctions. In some instances an extensively dilated left auricle may, by pressing on a bronchus, reduce its calibre one-half, and thus interfere with the functional activity of the left lung. When the pulmonary hyperæmia is extensive, violent physical exertion or violent coughing may cause a rupture of one of the larger pulmonary vessels, and true *pulmonary apoplexy* results. Bronchorrhœa is a frequent result of the intense hyperæmia of the mucous membrane of the bronchial tubes which may be produced in mitral stenosis. The lungs are always so liable to congestion and œdema that any sudden or violent exercise may cause sudden death. Again, when the above conditions have existed for some time, mitral stenosis may lead to dilatation and hypertrophy of the right heart. In some rare cases, the tricuspid orifice has become slightly insufficient.

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[1] Fagge and Hayden.

[2] Trans. Path. Society, xvii., p. 90.

Etiology.—Mitral stenosis is most frequent in the young; it rarely occurs after fifty. Statistics show it to be twice as frequent in females as in males. It is not infrequently of congenital origin. *Acute rheumatic endocarditis* is its most frequent cause. In some few instances stenosis results from extension of the inflammatory process from the aortic valves. It is a question if endocarditis in scarlatina or diphtheria in children ever causes mitral stenosis.

Symptoms.—The subjective symptoms of mitral stenosis are few. Usually after violent exercise there is more or less cardiac palpitation, and this will cease as soon as the auricle can empty itself, which is accomplished by the patient assuming a recumbent position on the right side, with the head slightly elevated. This class of patients are usually pale and anæmic, and frequently experience a sharp pain in the region of the apex-beat. The pulse is regular and normal in character, so long as the auricular hypertrophy compensates for the auricular dilatation. When the ventricle does not receive and discharge its normal quantity of blood with normal regularity, the pulse becomes small in volume, feeble in force, rapid and irregular in rhythm. The sphygmograph exhibits a tracing, sometimes called the "mitral pulse," the nature of which is the same as when the ventricle throws a greatly diminished blood current into the aorta.[1] The auricular systole commences earlier than normal on account of the hypertrophy of the auricle. This premature contraction of the auricle stimulating ventricular contraction, is indicated by a second ventricular systole, which is much less forcible than the first.

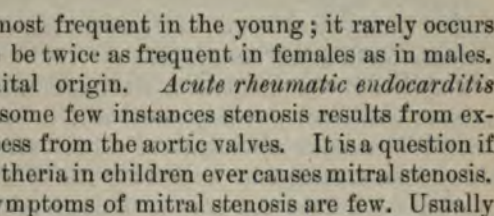

FIG. 104.

Sphygmographic tracing in a case of Mitral Stenosis. The line of descent is broken by pulsations from premature contraction of the over-filled auricle.

The passive pulmonary hyperæmia which attends the advanced stages of this form of cardiac disease causes habitual dyspnœa, which is exaggerated by physical exertion and by a dry, hacking, "teasing" cough, which resembles the so-called "nervous" cough. After violent or prolonged exertion there may be bronchorrhœa, a pint of glairy, watery mucus often being expectorated in a few moments. Severe exercise sometimes induces attacks of profuse, watery, blood-stained expectoration, indicative of pulmonary congestion and œdema. The exertion of walking rapidly against a strong wind will often cause such intense congestion and œdema of the lungs in one with extensive mitral stenosis as to induce sudden death. Hæmoptysis is not infrequent, small quantities of pure, florid blood being expectorated. Orthopnœa is a rare symptom, for even in extensive and long-standing cases the pulmonary congestion is *not* constant, for the auricle is ordinarily able to empty itself, and only becomes engorged during active physical exertion or great excitement.

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[1] Balfour differs from other authorities in the statement that among the most remarkable subsidiary phenomena of mitral stenosis is *irregularity* of cardiac rhythm which is always present in a greater or less degree.

Physical Signs—*Inspection.* As the left ventricle does not receive its normal quantity of blood, the cardiac impulse is feeble. Sometimes it has a visible undulating movement.

Palpation.—On palpation, although the apex-beat is less forcible than normal, a distinct *purring thrill* will be communicated to the hand; this thrill is a constant attendant of mitral stenosis. While mitral stenosis is always accompanied by a purring thrill, it should be remembered that a purring thrill does not *always* indicate mitral stenosis. It is most distinct at the apex-beat, although it may be diffused over the whole precordial space. It either continues through the entire diastole, or is only present just before the systole. It is sometimes called a "presystolic" thrill. It ceases at the apex-beat.

Percussion.—The increased size of the left auricle may cause an increase in the area of cardiac dulness, upward and to the left, at the inner part of the second left intercostal space. This increased area of dulness will only be recognized on careful percussion during expiration.

Auscultation.—Mitral stenosis is characterized by a loud "churning," "grinding," or "blubbering" presystolic murmur; this murmur is of longer duration than any other cardiac murmur, on account of the time required for the blood to pass through the narrowed and obstructed orifice. It ends with the commencement of the first sound and the apex-beat, being synchronous with the purring thrill. The murmur is heard with its maximum intensity a little above the apex-beat. It is louder when the patient is erect than when in a recumbent posture. When there is great debility or just before death, the murmur becomes indistinct. A presystolic murmur is never present unless there is narrowing of the auriculo-ventricular orifice, and then it is seldom, if ever, absent. A prolonged murmur and a sharp first sound indicate a "funnel-shaped" stenosis. The pulmonic second sound is intensified. When mitral reflux and mitral obstruction coexist, the two murmurs run into each other, constituting a single murmur. A mitral obstructive murmur is never soft or musical; it is usually separated from the first sound by a short interval. In about one-third of all cases, the second sound is reduplicated. Pulmonary congestion sufficiently accounts for the reduplication.[1] Some regard the length of the pause between the murmur and the first sound as a measure of the stenosis: the shorter the pause the greater the stenosis.

Differential Diagnosis.—The diagnosis of mitral stenosis is not difficult: it depends upon the existence of two physical signs, the "purring thrill" and a loud, long, *blubbering* presystolic murmur. It may be mistaken for *pericardial friction*, for a prolongated *systolic murmur* replacing the first sound at the apex, and for a *pre-diastolic basic murmur* transmitted to the apex.

To diagnosticate between *local pericarditis* and mitral stenosis the same methods are employed and the same rules are to be observed as in the diagnosis between aortic murmurs and local pericarditis (*q. v.*).

A prolonged *systolic apical* murmur reaching to the second sound is distinguished from a presystolic murmur by its soft and blowing character, and its synchronism with the systolic impulse and carotid pulsation.

A *pre-diastolic* murmur is distinguished from a mitral stenotic murmur by its progressively diminishing intensity, from the base to the apex, by its not being accompanied by hypertrophy of the left ventricle, and by a jerking, irregular pulse.

MITRAL REGURGITATION.

Regurgitation at the mitral orifice is due to a condition of the mitral valves which allows the blood to flow back from the left ventricle into the left auricle.

Morbid Anatomy.—The most common lesions are thickening, induration and shortening of the mitral valves. In rare instances, regurgitation may occur independently of valvular disease, from displacement of one or more of the segments of the valve, the result of changes in the papillary muscles, chordæ tendineæ, or the ventricular walls. It may also occur in extensive anæmia or from relaxation of the papillary muscles and dilatation of the left ventricle without a corresponding elongation of the papillary muscles, and from rupture of the chordæ tendineæ. In most instances, however, the valves are shortened, thickened, and indurated. In some cases, lime salts and large masses of chalky matter are found embedded in the indurated valves. In such cases the surface and edges of the valves are so rough and jagged that more or less obstruction accompanies the regurgitation. All these changes, except calcification, may also occur in the chordæ tendineæ and columnæ carneæ. The valves may also become adherent to the walls of the ventricles, or, as a result of the shrinking and shortening of the chordæ tendineæ, the valve-flaps will not pass back to the plane of the orifice. Again, the chordæ tendineæ may be ruptured so that the valves are pressed back into the auricle during the cardiac systole. If the chordæ tendineæ which are inserted nearest the centre of the valve become lengthened, that part of the flap will be bent upon itself, having evidently yielded to the blood pressure, and this allows of regurgitation. Sometimes when the valves appear perfectly healthy they will be found by the application of the "*water test*" to be insufficient.

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[1] Geigel ascribes it to "non-coincidence in the closure of the valves." Guttman regards it as originating at the stenotic orifice itself. Balfour thinks that thrill and reduplication of the second sound are sufficient to make a diagnosis in the absence of murmur.

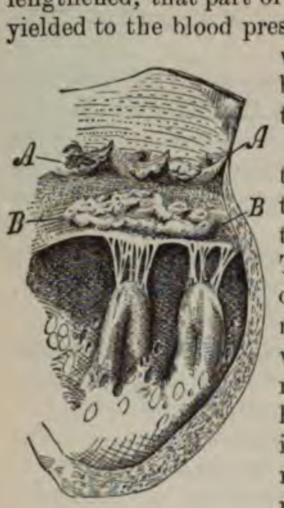

FIG. 105.
View of the Left Heart in Mitral Regurgitation.

The auriculo-ventricular valves are thickened with calcareous deposits, as shown at B, B. The aortic valves A, A, were in this case the seat of like deposits.

The first effect of mitral regurgitation is dilatation of the left auricle, due to the pressure of the two blood currents during its diastole, one from the lungs, and the other from the left ventricle. The dilatation leads to thickening and hypertrophy of the left auricular walls; as a result, the pulmonary circulation is impeded. The pulmonary vessels enlarge and may undergo degeneration, as a result of the continued regurgitant pressure. Passive hyperæmia of the lungs, with brown or pigment induration, is an early pathological sequel of mitral regurgitation. The constant interference with the return circulation from the lungs, more or less obstructs the outward current of blood to the lungs from the right ventricle. As the obstruction is a gradual one, the right ventricle becomes sufficiently hypertrophied to overcome it, consequently the hypertrophied right ventricle compensates for the mitral regurgitation.

So long as the hypertrophied right ventricle is able to fully overcome the abnormal pressure of the blood in the lungs from the mitral regurgitation, the patient is comfortable. Sooner or later, however, the compensatory hypertrophy of the right ventricle ceases, and a secondary dilatation occurs which admits of no compensation. This final dilatation of the right ventricle is favored by the myocardial degeneration which occurs as a result of defective nutrition of the heart walls; when this condition is reached, the veins throughout the body are placed in a similar condition to those in the lungs. This general venous congestion is indicated by passive hyperæmia of the abdominal viscera and by cyanosis of the surface during active physical exercise. The liver is the organ first affected on account of its great vascularity, and from the fact that the hepatic veins do not collapse readily and possess no valves. Thus, the liver becomes enlarged and has a stony hardness; as a result of the obstruction to the emptying of the hepatic vein the portal vein is obstructed, and this leads to passive hyperæmia of the intestines and stomach, and enlargement of the spleen. The impediment to the return of venous blood to the heart causes cerebral congestion, renal congestion, and, in fact, general systemic venous congestion. In addition to these changes, the dilated and hypertrophied left auricle throws an abnormal quantity of blood with abnormal force into the left ventricle during the diastole, which leads to dilatation of its cavity, and necessitates a compensatory hypertrophy of the left ventricular walls; this hypertrophy increases the force of the reflux current, so that during excitement and active physical exertion, pulmonary congestion and œdema are liable to occur.

Etiology.—Mitral regurgitation may occur at any age; it is especially liable in the young to follow rheumatic endocarditis, which causes extensive valvular retractions and thickenings. It is not infrequently secondary to changes at the aortic orifice produced either by an extension of endocarditis from the aortic to the mitral valves and their appendages, or by the secondary mitral valvulitis excited by the regurgitant blood current from the aorta. Mitral insufficiency may also be the result of that enlargement of the left auriculo-ventricular orifice which accompanies excessive dilatation of the left ventricle. Diseases of the columnæ carneæ and chordæ tendineæ, when their structures are so weakened as to allow the flaps of the valves to pass back of the plane of the orifice, will also cause mitral insufficiency. Ulcerative endocarditis may also cause it, either by perforation and rupture of the valves or by rupture of the chordæ tendineæ.

Symptoms.—During the early stage, when the hypertrophy of the right ventricle compensates for the regurgitation, there are no rational symptoms which would lead one to suspect its existence; but when the right ventricle is unable to overcome the obstruction to the pulmonary circulation caused by the regurgitant blood current, there will be more or less dyspnœa accompanied by a short, hacking cough with an abundant expectoration of frothy serum. Sometimes the watery expectoration is blood-stained. Active physical exertion increases the dyspnœa and causes cardiac palpitation.

In advanced cases, the extremities, face, and lips become blue, the result of the interference with the capillary return circulation. The liver becomes enlarged and hardened, a condition easily recognized by palpation and percussion. The patient will complain of a sense of weight and fulness in the right hypochondrium, and there will be anorexia, nausea, and a sense of oppression in the epigastrium, and sometimes the hepatic circulation becomes so obstructed that the biliary secretion is interfered with and a jaundiced hue of the surface will be added to the cyanotic discoloration, which will give to the skin a greenish tint. Headache, dizziness, vertigo, stupor, somnolence, and sometimes a peculiar form of delirium of short duration, result from the passive cerebral hyperæmia induced by obstruction in the superior vena cava. Following the hepatic derangement, are frequent attacks of gastric and intestinal catarrh, and evidences of embarrassed renal circulation. The *urine* is diminished in quantity, high colored, and loaded with lithates. Sometimes albumen and blood casts are found in it. Frequently the blood-stained expectoration is accompanied by free hæmoptysis; a cough and watery expectoration with occasional dark blood stains are usually present as advanced symptoms of mitral regurgitation. Another late symptom of mitral regurgitation is *dropsy;* it first appears in the lower extremities, and gradually extends over the whole body. With the general anasarca there is more or less dyspnœa. Late in the disease, pulmonary hemorrhagic infarctions may occur.

The *pulse* of mitral regurgitation is, at first, regular in force and rhythm;

later it becomes diminished in volume, irregular in rhythm, and diminished in force; it is never jerking in character. When the heart's action is excited, it becomes feeble and compressible and has a certain tremulousness. The sphygmographic tracing shows great depth and amplitude of the diastolic notch.

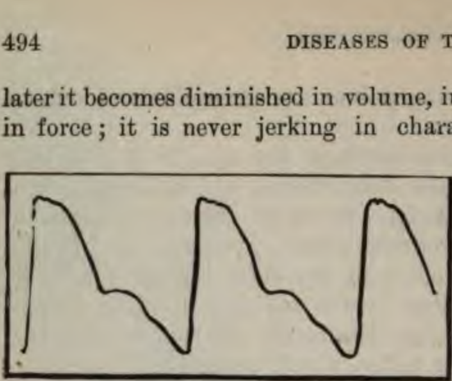

FIG. 106.

Sphygmographic tracing in Mitral Regurgitation showing great depth and amplitude of the diastolic notch.

Physical Signs. — *Inspection.* The area of the visible cardiac impulse is increased, and not infrequently there is a slight pulsation, corresponding in rhythm with the heart beats.[1] The epigastric pulsation is due to right ventricular hypertrophy, which is a condition always found with extensive mitral regurgitation. The jugular veins appear swollen, especially when the patient is lying down.

Palpation.—The apex-beat is displaced to the left and is felt *lower* than normal. When the dilatation exceeds the hypertrophy, the apex-beat is carried outward and often slightly upward. The impulse is diffused and more or less forcible, according as right or left ventricular hypertrophy predominates. Palpation sometimes reveals a systolic thrill, which is confined to the region of the second left intercostal space near the sternum. This systolic *fremissement* is not noticeable when the base of the heart lies close to the chest-wall because of retraction of the margin of the left lung. A purring tremor, systolic in rhythm, felt most intensely at the apex, and becoming feebler the farther the hand is removed from that part, either to the right or upward, is invariably due to mitral regurgitation.[2]

Percussion.—Percussion reveals an increase in the area of cardiac dulness, especially laterally; it extends both to the left and right of the normal line, as well as downward. The area of the superficial as well as of the deep-seated dulness will be increased laterally and downward.

Auscultation.—Mitral insufficiency is attended by a systolic murmur which either completely or partially replaces the first sound of the heart. The quality of the murmur is variable, and not in itself distinctive. It is usually soft and blowing; sometimes, toward its end, the murmur will assume a distinctly musical character. The first sound of the heart may be heard distinctly in the early stages, but later the murmur nearly always takes the place of the heart sound. Hence many English writers rightly call this murmur "post-systolic" rather than "systolic" in its nascent stages. It is heard with its maximum intensity at the apex-beat. Its area of diffusion is to the left, on a line corresponding to the apex-beat. It is audible at, or near, the inferior angle of the left scapula. It can he heard between the lower border of the fifth and the upper border of the eighth vertebra, at the left of the spine, with nearly the same intensity as at the apex. The second sound of the heart, over the pulmonary valves, is accentuated, while at the junction of the third rib with the sternum on the left side, both heart sounds are feeble. Skoda first drew attention to exaggeration of the second pulmonary arterial sound as a "positive and unerring" indication of mitral regurgitation. It is not always present. Whatever may be its character, the murmur is generally loudest at its commencement.

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[1] Skoda, Bamberger and Leyden record instances in which inspection showed a *double impulse*, accompanying, with more or less regularity, each cardiac systole. This only occurs in aggravated cases, and arises from non-coincidence of contraction of the ventricles.

[2] Hayden states that "*it is exceptional* to have a purring thrill with simple mitral reflux." I have never found it, except in those cases where left ventricular dilatation greatly exceeded the hypertrophy.

A loud systolic murmur at the *apex*, and *not* heard at the back, is not indicative of mitral reflux. If stenosis and regurgitation occur in the same individual, they give rise to a combined presystolic and systolic murmur, which begins shortly after the second sound of the heart, and continues until the second sound commences. The two sounds, although mingling to form one murmur, can, in the majority of cases, be readily distinguished from each other, for the point of maximum intensity and the very limited area of diffusion of a presystolic murmur readily distinguish it from a mitral systolic, which is audible in the left scapular region. It is important to recognize the existence of both these murmurs in estimating the prognosis in any case.

Differential Diagnosis.—It is usually not difficult to recognize mitral regurgitation. The seat and rhythm of the murmur and its area of diffusion are sufficient to distinguish it from other cardiac murmurs. The character of the pulse, the symptoms referable to the right heart, and the pulmonary complications will also assist in its diagnosis. It may, however, be mistaken for *aortic obstruction*, since each gives rise to a systolic murmur, for *tricuspid regurgitation*, and *for roughening of the ventricular surface of the mitral valve*, or of the *ventricular wall near the aortic orifice*. The diagnosis between mitral regurgitation and aortic stenosis has already been considered.

Mitral and *tricuspid insufficiency* both produce a systolic murmur; a mitral regurgitant murmur has its maximum intensity at the apex, and is conveyed toward the left axillary and scapular regions, while the maximum intensity of a tricuspid regurgitant murmur is to the left of the base of the xiphoid cartilage, and it is transmitted upward and to the right,—the area of transmission establishes the diagnosis. Pulmonary symptoms are prominent in mitral reflux and absent in tricuspid regurgitation. The pulmonary second sound is markedly *enfeebled* in tricuspid regurgitation, and markedly *intensified* in mitral regurgitation.

Roughening of the ventricular wall gives rise to a murmur which has its maximum intensity at the base of the heart, and is transmitted along the aortic arch and into the vessels which spring from it in the thorax. The *vibration* of an irregular *chorda tendinea* stretched across the aortic orifice, its extremities being inserted into opposite walls of the ventricle, may produce a systolic musical murmur, but the line of its transmission will correspond to that of an aortic obstruction. A systolic mitral murmur, due to a sudden rupture of one or a number of the valve-flaps, of the papillary muscles, or tendons, is a loud, blowing murmur, usually appearing suddenly, which is immediately accompanied by all the urgent symptoms of acute pulmonary congestion.

TRICUSPID STENOSIS.

This lesion is so rare that there are no rules for its diagnosis. Its *morbid appearances* and *etiology* are similar to those of pulmonic stenosis. Its symptoms would be those due to obstruction to the entire systemic venous circulation. The right auricle would be dilated, and there would be visceral enlargements in the abdomen, cyanosis of the face and extremities, scanty and albuminous urine, hemorrhoidal tumors, headache, dizziness and vertigo (due to passive cerebral hyperæmia), and general anasarca. The few cases recorded are associated with mitral stenosis, with one exception, a case of Bertin's.[1] In a case exhibited by Quain, the tricuspid flaps, thick and opaque, were united for one-third of their extent. In the other cases, the flaps of the valve formed a diaphragm whose central opening admitted only the point of one finger. In every recorded case of tricuspid stenosis the heart was enlarged. Tricuspid stenosis (as also pulmonic stenosis) may be the result of the pressure of a tumor. In all well-authenticated cases, the chief symptoms seem to have been extreme lividity, palpitation, and dyspnœa.

Physical Signs.—*Inspection* reveals general cyanosis. The jugulars are turgescent and exhibit presystolic *pulsation*. This pulsation is sometimes the only inconvenience the patient suffers.

Palpation may discover a venous thrill at the base of the neck.

Percussion may show the right auricle to be greatly enlarged, and cardiac dulness will be increased laterally and toward the right.

Auscultation.—Tricuspid stenosis should be attended by a presystolic murmur whose maximum intensity would be at the lower portion of the sternum just above the xiphoid cartilage. This murmur may be propagated faintly toward the base, but never toward the apex of the heart. It is sometimes accompanied by fremitus. Hayden offers the following "diagnostic point:"—the murmur of mitral stenosis (without which tricuspid stenosis never occurs) is limited to the apex region; a murmur of the same *rhythm* is produced at the sternum by tricuspid stenosis, "*and between these two localities there is a point where no murmur can be heard.*"

It is unnecessary to consider its *differential diagnosis*. The lesion would be diagnosticated (if at all) by exclusion, and *prognosis* and *treatment* would depend upon the gravity of the accompanying condition. As tricuspid stenosis never occurs unless there is extensive mitral obstruction, the latter condition is always the predominant one.

TRICUSPID REGURGITATION.

This lesion is usually secondary, yet it may be primary. Mitral disease is, in nearly every instance, the antecedent condition.

Morbid Anatomy.—The lesions are similar to those occurring in mitral insufficiency. The valves are thickened, shrunken and opaque; the papillary muscles are shortened and thickened, and the chordæ tendineæ undergo like changes and are sometimes adherent. The valves, or the columns or cords, may rupture; in either case acute and extensive insufficiency results. Acute endocarditis of the right heart is rare in adults, but when it occurs the tricuspid valves are its principal and primary seat, on account of their anatomical structure and the tension to which they are subject in mitral disease. They are rarely the seat of rheumatic or calcareous degeneration. Ulcerative endocarditis in the right heart is seldom met with.[1] Any infection from emboli from the tricuspid flaps will produce their secondary effects in the lungs. The first effect of tricuspid regurgitation is dilatation of the right auricle; following this, there will be hypertrophy of its walls. The auricular hypertrophy soon ceases to compensate, and then venous engorgement occurs.

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[1] Traité. des Mal. du Cœur, Obs. 17.

As soon as the valves in the subclavian and jugular veins are no longer able to resist the regurgitant current, jugular pulsation follows. But, before this occurs, the tributaries of the inferior cava and the organs to which they are distributed will become greatly engorged, for they have no valves to resist the regurgitant current. The inferior cava and the hepatic veins sometimes become enormously distended under these circumstances, the liver showing the peculiar appearance on section that has gained for it the name of *nutmeg liver*. Following the hepatic changes, the skin assumes a dingy yellow hue. When this is combined with cyanosis it has a peculiar greenish tint, only met with in heart disease. The spleen enlarges and hardens; the mucous membrane of the stomach is congested and ecchymotic, and often presents numerous hemorrhagic erosions. Intestinal catarrh is subsequently developed, and the general venous congestion within the abdominal cavity is exhibited by hemorrhoids and ascites. The kidneys become congested and stony, and thrombi may form in the femoral vein and induce subsequent pulmonary infarctions.

The stasis in the veins below the diaphragm is accompanied by transudation of serum, first in the ankles, and thence the dropsy progresses upward until the patient may finally reach a condition of general anasarca. The resulting obstruction to the general systemic circulation may cause hypertrophy of the left ventricle, and then we have the rare occurrence of disease of the left heart following that of right. Since tricuspid reflux has mitral disease for its principal cause, the heart becomes greatly enlarged, and a condition of extreme cardiac dilatation and hypertrophy is reached.

Etiology.—The most frequent cause of tricuspid regurgitation is mitral stenosis and regurgitation. Tricuspid reflux from primary endocarditis is *very* rare. Any condition of the lungs which will produce hypertrophy and dilatation of the right ventricle will lead to it; it is met with in extreme pulmonary emphysema and in cirrhosis of the lung with extensive chronic bronchitis. Balfour regards chronic bronchitis as its most frequent cause after mitral stenosis. Any valvular disease in the left heart of long duration may lead to it. In all these causes the *rationale* is the same: the abnormal amount of blood in the right ventricle presses with undue force against a valve which physiologists regard as ***normally slightly insufficient***, and the stress upon the valve flaps and the valvular attachments is such that endocardial inflammation is excited at the part subject to the greatest strain, and valvular insufficiency results.

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[1] Charcot and Vulpian record a case where one of the tricuspid valves was softened and perforated, presenting numerous vegetations. Scattered abscesses in the lungs were found in this case.

Symptoms.—As tricuspid reflux is usually secondary to some other form of valvular disease, or to some chronic pulmonary affection, the symptoms during its early stages are vague and masked by those of the primary disease. But directly the venous return is markedly impeded, a train of symptoms is developed which has its origin in the visceral derangements. In addition to these symptoms, there may be, in extensive tricuspid reflux, cardiac palpitation, cardiac dyspnœa, and marked irregularity in the force and rhythm of the heart. The liver is enlarged, the skin becomes dingy, and there is obstinate constipation and hemorrhoids. The liver is rendered liable in such cases to interstitial hepatitis. The spleen is enlarged. Venous stasis in the stomach is evinced by dyspepsia, nausea, vomiting, and hæmatemesis. The secretion of the kidneys is scanty, dark colored, of high specific gravity, often containing albumen and casts. Passive cerebral hyperæmia is marked by headache, dizziness, vertigo, and muscæ volitantes; there is a peculiar mental disturbance which is not met with in any other form of heart disease. Placing the patient in a horizontal position, after the disease has existed for some time, causes the face to become turgid and blue, and if the position be retained, stupor and coma may supervene. Jugular and epigastric pulsation are its characteristic physical signs. A very late symptom is dropsy, which begins at the ankles, extending upward until there is general anasarca. It is a noticeable point, that in the dropsy from tricuspid reflux the genital organs suffer slightly, if at all.

Physical Signs.—*Inspection.* In extensive tricuspid disease, the area of cardiac impulse is increased more than in any other valvular lesion. This area sometimes extends from the nipple to the xiphoid cartilage, and it may reach as high as the second right intercostal space. There is a visible impulse in the jugular veins, more apparent in the right than in the left. Sometimes the veins in the face, arms, and hands are seen to pulsate, and even the thyroid and mammary veins.

Palpation.—The apex-beat is indistinct, except in cases where there is marked hypertrophy of the left ventricle. Pulsation occurs in the epigastrium, which may be due to reflux into the enlarged hepatic veins, or to the fact that the dilated and hypertrophied right ventricle so presses on the liver, that the impulse is conveyed through the diaphragm with each cardiac pulsation. Early in the disease, the impulse in the jugular is confined to the lower part of the vessel. Beyond this point, the vein rarely undulates. Later, a systolic pulsation is felt as high as the angle of the jaw, and may be accompanied by distinct, though feeble, presystolic pulsation. The liver may simply undergo systolic depression, chiefly at the left lobe; or the whole liver may pulsate from an impulse coming from an enormously dilated vena cava; or the systolic pulsation of the veins within the organ may give rise to a palpable expanso-pulsatory movement. The hepatic pulsation is synchronous with the cardiac impulse. In rare cases it precedes jugular pulsation. Sometimes pulsation is felt in the femoral veins.[1] Sphygmographic tracings of the jugular pulse show it to be dicrotic.

Percussion shows an increase in the area of cardiac dulness to the right and upward, sometimes as far as the second intercostal space.

Auscultation.—The murmur of tricuspid reflux is heard with, or takes the place of, the first sound of the heart; it is superficial, of low pitch, blowing, soft, and faint, and is heard with greatest intensity over the lower part of the sternum, at its left border between the fourth and sixth ribs. It is rarely audible above the third rib, or to the left of the apex-beat. This murmur is transmitted from the region at the base of the xiphoid cartilage, upward and to the right, from one to two inches. Sometimes it is heard over a very limited area, and then it may be overlooked.

Differential Diagnosis.—A tricuspid regurgitant murmur may be confounded with an *aortic obstructive, pulmonic obstructive,* and *mitral regurgitant.* A tricuspid regurgitant murmur is never audible above the third rib, is accompanied by an accentuation of the second sound over the pulmonary artery and by jugular and epigastric pulsation, and is heard with maximum intensity near the base of the ensiform cartilage. These points are sufficient to differentiate it from an *aortic obstructive* murmur.

The differential diagnosis between it and a mitral regurgitant murmur has been given.

PULMONIC OBSTRUCTION.

Very little is known of diseases at the pulmonary orifice. Their diagnosis is arrived at by exclusion, and they cannot be recognized, except by their physical signs. Endocarditis in the right heart is rare, except in intra-uterine life. Valvular diseases of the right heart are usually the sequelæ of valvular disease in the left. The pulmonary artery may become atheromatous, but, even then, disease of the pulmonary valves is rare. Balfour believes that constriction of the pulmonary artery may occur at various periods of intra-uterine life; as a rule, the pulmonary valves are subject to no lesions except congenital malformations.

Morbid Anatomy.—Bertin records an instance of pulmonary obstruction where the distorted and adherent valves formed a horizontal septum across the orifice, which was only one-fourth of an inch wide. A rigid tricuspid valve has been found to be the cause of obstruction at the pulmonary orifice, the pulmonary valves themselves being normal. A few autopsies have revealed obstructions at the pulmonary artery caused by aneurisms, tumors of the pericardium or of the anterior mediastinum, enlarged bronchial glands, or pressure of a solidified lung. The pulmonary artery may be occluded just beyond the valves by a cancerous tumor, and there are examples where a phthisical process in the left lung has induced it. A murmur indicative of pulmonary obstruction may be produced by a cardiac thrombosis.

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[1] Guttman thinks epigastric pulsation is due wholly to reflux into the veins of the liver, and not to right ventricular pulsation.

The above statements I place under the head of morbid anatomy of the lesion, as they cannot be appreciated nor their pathological significance realized during life. Reasoning from analogy, obstruction at the pulmonary orifice ought to be followed by compensatory hypertrophy of the right ventricle, and accompanied by tricuspid regurgitation and dilatation of the right auricle.[1] I have met with only two cases of pulmonic obstructive murmurs in which autopsies were obtained. In both cases it was found that the murmur had been produced by mediastinal tumors pressing upon the pulmonary artery so as to diminish its calibre.

Etiology.—Pulmonary stenosis is rarely the result of endocarditis or of degenerative changes in the pulmonary artery. Bertin states that when abnormal communication between the two sides of the heart has existed, the arterial blood may excite endocarditis in the right heart. Syphilis has been advanced as a possible cause of degenerations at the pulmonary orifice.

Symptoms.—The only rational symptoms that have been noted in the few recorded cases of pulmonic disease admit of manifold explanations, and no one is either constant or diagnostic. In some cases anæmia existed; in others there was cardiac palpitation, dyspnœa, cyanosis, and dropsy, but none of these belong exclusively to a pulmonic lesion, nor do they necessarily depend upon it.

Physical Signs.—*Inspection, palpation,* and *percussion* give negative results. Palpation *may* give a systolic thrill, confined to the second left intercostal articulation. Such a *fremissement* results both from roughness and contraction of the pulmonic orifice.

Auscultation.—A systolic murmur is heard with its maximum intensity directly over the pulmonic valves; it is very superficial and consequently very distinct, and is limited in its diffusion. It is never heard at the xiphoid cartilage, nor along the course of the aorta. If it has an area of diffusion, it is toward the left shoulder. The murmur is loud and soft in character, sometimes "bellows;" it is not audible in the vessels of the neck, nor is it attended by arterial pulsation. When phthisical consolidation partially occludes the pulmonary artery, a loud but soft systolic murmur is heard, which is sometimes high-pitched and musical, and which is often entirely suspended during a full inspiration. In some few instances, there is a *bruit de diable* in the jugular veins.

Differential Diagnosis.—It is possible to confound a pulmonic obstructive murmur with a *mitral regurgitation* which is propagated upward into the left auricular appendix. But the area of a mitral regurgitant is also backward, and by this it could be distinguished from a pulmonic obstruction. Beside, in mitral disease the pulse is very different from the pulse of pulmonary stenosis.

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[1] Dr. Ormerod records three cases in which pulmonary obstruction was diagnosticated during life, and where the post-mortem proved the accuracy of the diagnosis. Two of these occurred in men under twenty-eight, and the other in a woman twenty-one. In two of these cases all the cardiac valves were healthy, except the pulmonic. The pulmonic orifice would barely admit a goose quill. Warburton Begbie mentions a case (man: aet. 18) in whom reflux and stenosis at the pulmonary orifice coexisted. There were four valves, and these were incompetent. All the other valves were normal. Congenital stenosis of the infundibulum of the right ventricle is the probable result of fœtal myocarditis or syphilis.

Aortic stenosis can hardly be mistaken for pulmonary obstruction, for the arterial pulsation, the peculiar pulse, and the transmission of the murmur into the arteries of the neck will suffice to discriminate between them. An *aneurism* at the *sinus of Valsalva* may produce a murmur in the pulmonary artery by the pressure which is exerted upon that vessel. It would be impossible to distinguish this murmur from that of a pulmonic stenosis.

The diagnosis of pulmonary obstruction is usually reached only by exclusion.

PULMONIC REGURGITATION.

Many doubt the occurrence of this form of valvular lesion. There are only a few well authenticated cases,[1] and in them the lesion has been the result of injury or congenital defect. The statement[2] that the pulmonary valves exhibit a cribriform condition nearly as often as the aortic, is not sustained by post-mortem examinations. In one of the cases to which I have referred as an example of pulmonary stenosis, the valves were also insufficient. In Dr. Begbie's case, where there were four flaps to the valves (producing obstruction), marked insufficiency coexisted. The *morbid anatomy*, *etiology*, and *rational symptoms* do not require a separate consideration. The anatomical conditions are the same as those found in similar conditions of the aortic valves; and the etiology and rational symptoms are those of pulmonic stenosis.

Physical Signs.—Theoretically pulmonic regurgitation should be accompanied by a diastolic murmur having its maximum intensity over the pulmonic valves; and its area of diffusion should be downward and toward the xiphoid cartilage. It should be soft and blowing in character. This murmur is rarely heard alone; it is usually associated with obstruction at the same orifice, or with some murmur whose origin is on the left side of the heart. Niemeyer states that dyspnœa, hemorrhagic infarction, and consumption of the lungs have followed insufficiency at the pulmonary orifice. No other authority mentions such symptoms, while the assignment of valvular disease as a cause of phthisis is absurd. With a pulmonic regurgitant murmur there should be, on palpation and percussion, physical evidences of hypertrophy and dilatation of the right heart, the *rationale* of whose production should be identical with that which was considered in aortic regurgitation. I have never heard a regurgitant pulmonic murmur.

Differential Diagnosis.—The murmur of pulmonary regurgitation may be mistaken for that of *aortic regurgitation*. The points in connection with their differentiation have already been given. The prognosis and treatment are the same as those of the former lesion.

Prognosis in Valvular Disease of the Heart.—The duration of life in valvular disease of the heart varies greatly.

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[1] Path. Trans., vol. xvi., p. 74.

[2] Dis. of Heart. Bellingham.

To establish a basis of comparison, I shall give a *résumé* of eighty-one cases, in all of which the diagnosis of valvular disease was confirmed by a post-mortem examination.[1] In fourteen cases of different valvular diseases, each of which was complicated by cardiac hypertrophy and dilatation, fifty per cent. of deaths were directly due to the valvular lesion. In one of these, where there was stenosis at both the mitral and tricuspid orifices, death was sudden. In fifteen cases in which there was only cardiac hypertrophy, eleven deaths occurred from the heart-lesion, five of which were sudden and directly due to the valvular lesion. In six cases in which dilatation alone existed, four deaths directly resulted from the heart-lesion, and two of these were sudden. In *not one* of fifteen cases of aortic disease did death occur directly from the heart-lesion. Of these fifteen cases, sudden death occurred in only two. In twelve cases of calcified mitral valve, *no death* occurred directly from the heart-lesion; there were but two sudden deaths, both from cerebral apoplexy. The aortic and mitral valves were diseased in fourteen cases; two deaths were due to the heart-lesion, and there were but three sudden deaths (uræmia, apoplexy, and croupous laryngitis). The aortic and pulmonary valves were involved in three cases, all of which died suddenly, and none directly from the heart-lesion. In two instances, the aortic, mitral and tricuspid were involved, in neither of which sudden death occurred. Thus, of eighty-one cases, twenty-four deaths only were directly due to the heart-lesion, and of these only eight were sudden.

From the above cases it seems evident that the prognosis is not bad in valvular disease, except when hypertrophy and dilatation coexist, and then many complications are liable to occur. In 1870, I had a patient sixty years old with extensive aortic reflux, who had had three attacks of pneumonia during the eight years he was under my observation. There was only slight cardiac dilatation in this case.[2]

In *aortic stenosis*, life may be prolonged many years. So long as the left ventricular hypertrophy compensates for the stenosis, the prognosis is good; but when it fails, and dilatation begins, cerebral anæmia soon results. If violent or prolonged efforts are followed by irregular heart-action, sudden death may occur. Hypertrophy and dilatation, syncope, cerebral anæmia, vertigo, muscular debility, a very pale face, and an irregular pulse render the prognosis unfavorable. Should vegetations be suspected, there is danger of cerebral embolism. Complicating (secondary) mitral disease renders the prognosis unfavorable. Death results from complications, degenerations of the heart, and pulmonary œdema.

Aortic regurgitation is a graver form of disease than aortic stenosis. Its duration is indefinite, as it may give rise to no symptoms until it is far advanced. Twenty-one days and five years are the extreme limits recorded. In *no other valvular disease is sudden death so liable to occur.* Reference to the above cases shows that mitral stenosis ranks nearly equal to it in this respect. The shorter and more gushing the murmur, the more extensive the regurgitation, the *effects* of which must always be carefully estimated before a prognosis can be given. Aortic regurgitation is, however, more serious in the young than in adults, because in children the changes are less atrophic and more inflammatory. In middle life and in those who are subjected to great physical or mental strain, the prognosis is unfavorable; if the vessels in these patients show evidences of degeneration, apoplexy and cerebral thrombosis are liable to occur. In the very old, I have known extreme aortic regurgitation to exist a long time and cause little inconvenience. Cyanosis and dropsy and signs of heart failure, dilatation, or degeneration of the walls of the heart, render the prognosis unfavorable; if mitral regurgitation is developed, visceral derangements occur and hasten the fatal issue. *Sudden* valvular incompetence is far more dangerous than that which has developed slowly. The prognosis is determined more by the condition of the heart walls and the general nutrition of the patient than by any other elements. When aortic regurgitation is complicated by aortic stenosis and mitral regurgitation with marked derangement of the general circulation, the prognosis is bad. Death may result from embolism, apoplexy, dropsy, pulmonary œdema, sudden cardiac insufficiency, or from visceral complications. When the radial impulse is felt a little after the apex-beat, it is always important to determine whether the heart's action remains regular under mental excitement or violent physical exertion; if it does, the prognosis is *good.*

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[1] Med. Rec., N. Y., Apr. 1, 1870, p. 66, etc.

[2] Dr. Walshe states that the order of relative gravity of valvular lesion is: Tricuspid reflux, Mitral reflux and stenosis, Aortic reflux, Pulmonary stenosis and Aortic stenosis.

Mitral stenosis admits of no compensation. If extensive, it is always a grave disease. The prognosis is estimated by the severity of the thoracic symptoms; if these are greatly increased by physical exertion, the prognosis is bad, for pulmonary congestion and œdema, infarctions and diffused pulmonary apoplexy with large extravasations are liable to occur. Statistics show that sudden death occurs nearly as often in mitral stenosis as in aortic regurgitation. Congenital mitral stenosis is not dangerous, nor does it occasion inconvenience, for it is always associated with hyperplasia of the arterial system. The later in life mitral stenosis occurs, the worse the prognosis.

Mitral regurgitation, when uncomplicated, gives rise to very little disturbance of the circulation, because it is generally most fully compensated for, and the changes which lead to it are of slow growth and their tendency is to remain stationary. Patients with moderate regurgitation suffer little, even on exercise. As long as right ventricular hypertrophy compensates, there is no dyspnœa. As regards the duration of life, the prognosis in mitral reflux is good. When, however, stenosis and regurgitation coexist, sudden pulmonary complications are very liable to occur, and the prognosis is bad. When signs of right heart failure occur, the prognosis is bad. Œdema of the extremities, fluid in serous cavities, cyanosis, dyspnœa, and hæmoptysis are indications of such failure. Death may result from general anasarca, serous effusions into the pleuræ, peritoneum, or pericardium, pulmonary œdema and congestion, or from sudden cardiac insufficiency.

Extensive obstruction or regurgitation at the *pulmonic orifice* would lead to serious results, but we have no statistics upon which to base a prognosis.

Tricuspid stenosis and obstruction, when associated with mitral disease, are very grave lesions, but not so bad as when resulting from chronic bronchitis or pulmonary emphysema. When jugular and epigastric pulsation are marked, the changes in the viscera already referred to quickly ensue. Walshe says: "Tricuspid regurgitation is the worst of all valvular lesions." Patients with tricuspid regurgitation are in constant danger from intercurrent attacks of acute pulmonary hyperæmia. Tricuspid disease leads more rapidly than any other valvular lesion to cyanosis and dropsy.

Treatment of Valvular Diseases of the Heart.—The treatment of *aortic* valvular disease can be summed up in, *rest, diet* and *regimen.* Rest must be mental as well as physical. The appetite, emotions and passions must be under perfect control, hence a sedentary country life is best. Straining, especially when the hands are above the head, is to be avoided. The nutrition must be kept as perfect as possible to guard against cardiac degenerative processes. Sugar, sweet vegetables, and animal fat must be sparingly used. The food should consist of nitrogenized material taken in quantities that do not interfere with the heart's action. In aortic regurgitation, patients while sleeping should assume, as nearly as possible, a horizontal position, as they thus lower the height of the distending column of blood, and relieve both the cardiac circulation and the tendency to pulmonary congestion. When defective aortic pressure reacts injuriously on the gastric and hepatic secretions, moderate alcoholic stimulation may be cautiously employed. The bowels should be daily *gently* moved. That the skin may be active, the body must be warmly clothed. Prolonged exposure to cold is to be avoided. Warm baths, especially warm sea-baths, are beneficial. Medicine is not to be given until the hypertrophy ceases to compensate. In aortic reflux with feeble heart-power, tr. digitalis and tr. ferri perchlor. are to be given in ten-drop doses, three times a day. The iron is especially called for when anæmia is present. Digitalis is given as a cardiac tonic, hence small doses only are required. As long as it increases the urinary secretion it is safe to continue it. When vertigo and syncope are prominent symptoms, quinine and strychnia may be given with the digitalis. Should the heart act with violence and rapidity, or if there is evidence of high arterial tension, *aconite* is serviceable.

In *aortic incompetence* small doses of arsenic have a stimulating effect, when given with digitalis and iron. Iron may disturb the stomach; arsenic seldom does. Quassia or calumba should always be given *with* iron. When the hepatic or gastric vessels are engorged, three or four leeches over the epigastrium or liver, followed by warm anodyne poultices, will often afford relief. Large quantities of fluid should never be taken into the stomach at one time. Symptoms of angina pectoris with dyspnœa and local pain are signs of *aortitis,* which demands leeches over the sternum and small doses of mercury. The treatment of dyspnœa, dropsy, etc., etc., will be considered in the treatment of mitral disease. The pain of aortic disease may be so severe as to demand an anodyne; opium by the mouth cannot be given, but the sulphate or hydrochlorate of morphia can be given hypodermatically. Nitrite of amyl often relieves the angina promptly.[1]

The first thing in the *treatment of mitral stenosis* is to have the patient fully understand his exact condition, that he may follow your advice implicitly, for the treatment is for the most part in his own hands. As to nutrition, the same rules hold as in aortic disease. There must be at least one gentle daily evacuation of the bowels. Straining at stool is to be avoided. The use of alcohol, strong tea or coffee or tobacco is to be prohibited. If anæmia exists, give iron one-half hour after meals, gr. x—xx of Vallette's mass, two or three times daily for a long period. The prolonged use of the voice is dangerous. Small doses of quinine and strychnia alternating with the iron are advantageous. If there is anorexia, the vegetable bitters are to be given. The triple phosphates of iron, quinine and strychnine, or small doses of dilute sulphuric acid will improve these patients when they show signs of extreme debility. In every case of mitral disease there comes a time when pulmonary hyperæmia shows failure of right cardiac compensation. An adjustment of the heart to the circulation is now effected by administering *digitalis,* which should only be given when heart failure is marked and is accompanied by pulmonary congestion. Half an ounce of the infusion, every two hours for twenty-four or forty-eight hours, is often required to overcome the cardiac failure. The time will come when digitalis ceases to sustain the heart, hence it should be used sparingly and carefully—*never* continuously. When the pulse is rapid, feeble, and irregular, more time and greater force for the ejection of blood from the ventricle are demanded. Digitalis meets both indications. The pulse becomes regular, full, and forceful. The urine becomes abundant and normal. Pulmonary engorgement diminishes and commencing dropsy slowly disappears. Hayden advises 10 drops of chloroform, 15 drops of tincture of digitalis, and 15 drops of tincture ferri perchloridi, in one ounce of water every three hours. When asystolism is present, or suppression of urine is threatened, digitalis must be given in large doses. In most cases of mitral stenosis digitalis is contraindicated. The dropsy of advanced mitral reflux may be promptly relieved by *pulvis jalapæ co.* combined with calomel in sufficient quantity to produce prompt and free catharsis. Squills, juniper, broom, and cream of tartar act as diuretics in such cases.

In *mitral regurgitation* a compound of digitalis and nitrous ether acts well as a diuretic. Whenever a diuretic is given in heart disease, the loins should be cupped or warm poultices applied and the bowels freely purged. In copious hæmoptysis in cardiac disease, ergotin in full doses hypodermically may be given. The hæmoptysis that accompanies pulmonary apoplexy of heart disease may relieve the dyspnœa ; hence Drs. Dickenson, Fagge and other English authorities recommend venesection for relief of pulmonary engorgement. Precordial pain accompanying valvular disease may be relieved by the application of leeches over the precordial space.

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[1] Barlowe and Fagge advise senega and carbonate of ammonia for the less severe effects of aortic regurgitation, which they regard as least amenable to treatment of all cardiac diseases.

Hyoscyamus, hydrochlorate of morphia, nitrite of amyl, chloroform, and a belladonna plaster over the precordium may be employed for the same purpose. Such pain is the cry of a heart-muscle for higher nutrition. Bleeding favors dropsy by thinning the blood and by diminishing the heart-power; it should never be practised except in emergencies.[1] When digitalis fails to regulate the circulatory disturbances, its use does harm; but in all cases of mitral disease where this drug has not been used, it is safe to say that its administration will give prompt relief. Morphia is the best anodyne and hypnotic to be used in mitral disease.

Hygiene, diet, and exercise are to be the same in *pulmonary*, as in mitral disease, further treatment is solely symptomatic.

The treatment of *tricuspid obstruction* depends on the gravity and sequelæ of the accompanying mitral disease. For tricuspid stenosis never occurs till mitral stenosis is excessive, and the latter condition is the predominant one. The general treatment is the same as in aortic and mitral diseases. The patient should lead a perfectly quiet life in a warm, equable climate. When this lesion occurs with mitral disease, digitalis should not be omitted, for the drug promotes ventricular contraction, and thus relieves the tricuspid pressure. In tricuspid regurgitation with emphysema, this drug should be very cautiously given, and its use or omission must depend upon the effects produced in each case. If cerebral symptoms are exaggerated it must be stopped. Tonics should be given on the same principles as in mitral disease and the same drugs used. A drastic purge or taking a few ounces of blood from the arm temporarily relieves the venous engorgement. Dropsy and local œdema are treated as in mitral disease. For the relief of the gastric, hepatic and intestinal symptoms, which are often the most troublesome occurrences in tricuspid regurgitation, I have found one or two purgative doses of calomel to act promptly and satisfactorily—in fact, in all cases of heart disease in which there is evidence of hepatic hyperæmia, an occasional calomel purge will be followed by marked relief and improvement.

Several new drugs have recently been employed in the treatment of cardiac diseases. Strophanthus and nitroglycerine are the more important. In many cases where a simple cardiac tonic is desired, or when digitalis causes gastro-intestinal disturbances, strophanthus will be found valuable. It is more prompt in action than digitalis, and thus may be used to initiate an action which is to be continued by digitalis. Nitroglycerine, in patients who are not affected unpleasantly by its action, may be employed to lower arterial tension, and thus relieve a laboring heart. Convallaria is at times of value, but its action is not constant or permanent, and, like strophanthus, it has little diuretic action. Adonidine relieves many of the distressing symptoms, but appears to me to act only as a sedative. Caffeine is a valuable cardiac stimulant for temporary effects. When digitalis fails or cannot be used in cases of cardiac dilatation, spartein sometimes gives relief. Its action is quickly obtained, as it is absorbed rapidly and eliminated with equal rapidity.

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[1] Niemeyer advises arsenic and antimony in mitral valvular disease; when and why he does not say.

No drug, however, has yet been introduced which supersedes digitalis as a cardiac tonic in the early stages of cardiac disease.

CARDIAC HYPERTROPHY.

By the term *cardiac hypertrophy* is meant thickening of the walls of the heart by an increase in their muscular tissue. This muscular increase may be confined to one portion of the heart, or it may involve the walls of both auricles and ventricles. There are three recognized forms of cardiac hypertrophy.

I. *Simple Hypertrophy.*—In this form there is an increase in the thickness of the cardiac walls, the capacity of the cavities remaining normal. Simple hypertrophy is usually confined to the left ventricle, and is most frequently met with in connection with chronic Bright's disease and chronic alcoholismus.

II. *Eccentric Hypertrophy.*—In this form there is thickening of the walls of the heart, with increase in the capacity of its cavities. It is most commonly met in connection with, or occurs as the result of, some valvular lesion.

III. *Concentric Hypertrophy.*—In this form there is thickening of the walls of the heart, with diminution in the size of the cavities. Some observers deny its occurrence, and claim that the diminution in the capacity of the cavities is only apparent—that it is the result of violent ventricular contraction just prior to death. I have never seen any example of this form of hypertrophy.

Morbid Anatomy.—The anatomical changes in cardiac hypertrophy vary according to its seat, and sometimes according to the character of the hypertrophy. In eccentric hypertrophy there will always be an increase in the size of the papillary muscles, and the septum will be thickened, which does not necessarily occur in connection with simple hypertrophy. The ventricular septum is far less liable to hypertrophy than the rest of the ventricular parietes. It is often difficult, even after death, to determine the existence of a moderate degree of cardiac hypertrophy, while extensive hypertrophy is very readily recognized.

When cardiac hypertrophy exists, the first thing noticed is a change in the shape of the organ, and this change will correspond to the seat of the hypertrophy. If the hypertrophy is confined to the left ventricle, either simple or eccentric, the heart will assume a more than usual pyriform shape, and will become elongated—the right ventricle seems to be a mere appendage to the left. On the other hand, hypertrophy of the right ventricle increases the horizontal measurement of the organ and gives it a more oval shape, the apex not being as pointed as in health, since extremities of both ventricles are on the same level. If all the cavities the heart are increased in capacity, and their walls hypertrophied, whole heart will be increased in size, but the change will be most mark in its horizontal direction, and the organ will assume a globular sha Sometimes the shape of the organ is not notably changed in gene hypertrophy.

Left *ventricular* hypertrophy occurs oftener than right, and hypertrop of the *right* auricle much oftener than that of the left. The ventric are hypertrophied oftener than the auricles. In all varieties of hypert phy the cardiac walls are stiff, so that when the cavities are opened a the blood has been removed from them, they do not collapse. The st stance of an hypertrophied left ventricle can generally be torn with ea while an hypertrophied right ventricle is tough and leathery. The co of the muscular tissue is redder than normal; there is an increase in number of the muscular fibres, which differ in no way in their anatomi structure from those of normal heart muscle. Occasionally there is increase in the size of the cardiac muscular fibres.[1] There may be m or less increase of connective-tissue between the muscular bundles; a Dr. Quain stated that this may be so excessive as to be a "false hyp trophy." Sometimes there are accumulations of fusiform involunt fibres which have not as yet developed into the higher state of stri fibres.

There is no limit to cardiac hypertrophy. The heart may reach suc degree of enlargement as to weigh forty ounces more than in its nor state ("*cor bovinum*"). After the hypertrophy reaches a certain po there is dilatation, preceding and accompanying which is fatty degene tion, which first occurs in the more recently formed muscular fibres. increase in the number or size of the muscular fibres of the heart w causes a corresponding increase in the heart power. The walls of the pertrophied heart vary in thickness according to the cause of the hyp trophy. The walls of the left ventricle may become an inch and a or even two inches thick, while those of the right ventricle rarely re an inch in thickness. The auricles are seldom more than double th normal thickness. The columnæ carneæ of the right ventricle are m liable to hypertrophy than the walls. Sometimes the walls of a cavity thinned at one point while they are hypertrophied at another. The hea a heart becomes, the deeper does it lie in the thoracic cavity; the phragm is pushed down, and the heart inclines more to the left of thorax.

Etiology.—In general terms cardiac hypertrophy is caused by o work; for some reason the cardiac walls are called upon to perfo re than their normal amount of labor, and an increase in the number their muscular fibres necessarily follows. Whenever the function the heart is permanently or repeatedly overtaxed, or when the resistce which it should normally encounter is increased, hypertrophy of walls is the result. The modes by which it is directly induced are as llows :

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[1] Cornil and Ranvier state that "it is not yet known whether hypertrophy is entirely due to increa size of the muscle-fibres, or to a new formation of these fibres. The phenomena of development of muscle-fibres have never been observed, so that the former hypothesis seems the more probable."

(1) *Dilatation of the Cavities of the Heart.*—Under certain circumstances ilatation of one or all of the cavities of the heart takes place during its diatole, and they receive more than their normal quantity of blood. A certain legree of force is required to discharge the normal quantity of blood ; if here is more than the usual amount, an abnormal degree of force is required o expel it. This demand for increased heart power is supplied by an inrease of muscular fibres in the heart walls,—the hypertrophy is developed n proportion to the increase of force required. This is the cause of those orms of cardiac hypertrophy which occur in connection with valvular nsufficiency. Under these circumstances the hypertrophy is always eccenric, and is not due so much to the valvular lesions as to the dilatation of he heart cavities which occurs as a result of these lesions. The order is, irst, dilatation, then hypertrophy to compensate for the dilatation. Dilaation is developed during cardiac diastole ; hypertrophy during cardiac ystole.

(2) *Mechanical Obstruction.*—Of those causes which originate in the heart, aortic stenosis gives rise to hypertrophy of the left ventricle ; mitral stenosis to hypertrophy of the left auricle ; pulmonic disease to hypertrophy of the right ventricle ; and tricuspid stenosis to hypertrophy of the right auricle. In the list of mechanical causes are included all those diseases of the arteries which diminish their elasticity. The walls of the large arteries may lose their elasticity from atheromatous degeneration, or they may be constricted or dilated, and thus offer obstruction to the blood current. An aneurismal tumor may have developed sufficiently to obstruct the current of blood,[1] or some tumor may press upon and diminish the calibre of the aorta ; under such circumstances a more than normal amount of work will be imposed upon the left ventricle, and simple cardiac hypertrophy will be developed as the result. Twisting of the thorax and deformities of the spine, thorax, etc., may act in the same way. Again, bstruction to the pulmonary circulation will give rise to hypertrophy of the walls of the right ventricle ; in many instances dilatation will occur rior to the hypertrophy, but in quite a large number of cases direct ypertrophy of the right ventricular walls will occur as the result of bstruction to the pulmonary circulation. Such obstruction may be develped in connection with pulmonary emphysema, fibroid and compressed ung, chronic pleurisy, asthma, hydrothorax, and other chronic diseases hich interfere with the circulation of blood through the lungs. It does

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[1] But this is rare : Axel Key has shown that aneurism of the aorta *alone* is not productive of left vencular hypertrophy, since it does not lead to increase in the arterial tension.

not occur in the early stage of pulmonary phthisis, for the pulmonary circulation is not obstructed until the advanced stage of the disease. Hypertrophy of the left ventricle may also result from interference with the general capillary circulation.

Simple hypertrophy of the cardiac walls is one of the most constant attendants of Bright's disease.

The relations between cardiac hypertrophy, arterial and renal disease are exceedingly complex. It is known that the changes of general arterial capillary fibrosis induce an increased arterial tension which causes hypertrophy of the heart, and that a similar cardiac condition is a constant attendant of all forms of chronic Bright's disease. The theory that the renal obstruction alone is responsible for the hypertrophy is now abandoned, and two distinct conditions are recognized. In one the renal change is cirrhotic and part of a systemic condition, developed in a patient of fibroid diathesis. In such cases the vascular changes causing increased arterial tension and cardiac hypertrophy are organic and permanent. In the other cases the renal changes are parenchymatous, and the vascular contraction is arterial, and due primarily to contraction of the muscular coat, through a sympathetic nervous stimulus, that is intended to produce increase of arterial tension for the sake of its diuretic action. It is claimed that this reflex vasomotor contraction may result in hypertrophy of the muscular coats of the arteries. In chronic alcoholismus, rheumatic hyperinosis, or any other condition which interferes with the systemic capillary circulation, more or less extensive simple cardiac hypertrophy of the left ventricle is developed.

Anything which increases for any length of time the rapidity and force of the heart's contraction may produce cardiac hypertrophy. Among this class of causes may be included excessive and prolonged muscular exercise, *especially in young subjects*, and in soldiers who are on the march. Emotional conditions that produce cardiac palpitation, prolonged mental excitement, the immoderate use of strong coffee or alcohol, are causes of cardiac hypertrophy. These are styled "nervous" causes (Quain); and to this class probably belong those cases occurring in Graves's or Basedow's disease. Pericarditis is not infrequently a cause of cardiac hypertrophy, either by inducing softening and dilatation of the ventricles, or by the obstruction which is offered to the heart's action by the adhesions between its two surfaces. The heart becomes hypertrophied in pregnancy, but returns again to normal after delivery. Sometimes no cause can be found for cardiac hypertrophy.

Symptoms.—The valvular lesions, arterial changes, or capillary obstructions which are associated with cardiac hypertrophy modify, or to a greater or less extent obscure, the phenomena which attend the hypertrophy. Total eccentric hypertrophy usually cannot be detected except by a physical exploration of the chest. There are, however, certain subjective symptoms which are important and which will aid in its diagnosis. The direct effect of general hypertrophy of the heart is to cause an abnormal fulness of the arteries and a lack of blood in the veins. The pulse is full and strong, and is bounding in character; the face is easily flushed, the eyes somewhat prominent and brilliant, and there is carotid pulsation. The respiration is not usually disturbed until the heart becomes so increased in size as to give rise to pressure upon the adjacent lung-tissue and upon the diaphragm; then the patient will have a sense of fulness about the chest, and with that sense of fulness there will be more or less uneasiness in the epigastrium, and the stomach digestion may be more or less interfered with. If dyspnœa is present it is due to the pressure of the enlarged heart rather than to any change in the lung-tissue. In eccentric hypertrophy with dilatation, more especially when the right cavities are affected, pulmonary œdema and congestion are usually present, and then there is marked dyspnœa.

In simple hypertrophy there is often a dry irritating cough; and in young fleshy females it has a wheezing character. In right heart enlargements the cough is often distressing. This class of patients when excited are very apt to complain of cardiac palpitation. The heart's action is often irregular and intermittent. In almost all cases there is some cerebral hyperæmia, consequently it will be found that in those who are the subjects of eccentric cardiac hypertrophy alcoholic stimulants, nervous excitement, and active physical exercise will cause headache, vertigo, ringing in the ears and bright spots or flashes before the eyes. In left hypertrophy, hæmoptysis is common and comes on suddenly. Rupture of the bronchial arteries may occur.[1] Cerebral apoplexy may at any time occur, when the arteries are predisposed to, or have already developed *small aneurisms*. In fact, the majority of cerebral apoplexies which occur in young subjects are associated with cardiac hypertrophy. It is now well established that there is a close connection between atheroma of the arteries and cardiac hypertrophy. Some observers claim that the cardiac hypertrophy is secondary to the arterial changes; but it is a fact of every-day observation that hypertrophy from valvular changes will give rise to atheromatous changes in the arteries for reasons which have already been fully considered in connection with the history of valvular diseases. The steps of the change are, first, cardiac hypertrophy; second, endarteritis; and, lastly, atheroma. The general symptoms considered in connection with its physical signs render its diagnosis easy.

Physical Signs.—The physical signs of cardiac hypertrophy will vary with the seat and extent of the hypertrophy. When it is general, upon *inspection* it will be noticed that although the heart's action is regular there is an increased area of impulse, and that there is a motion with each cardiac pulsation over and even beyond the precordial space. In children there is often bulging of the precordial space.

In *right ventricular hypertrophy*, *inspection* may reveal a rounded smoothness of the epigastrium, with, perhaps, some bulging of the ensiform and lower left costal cartilages. The apex-beat may be diffused, extending toward the ensiform cartilage.

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[1] Niemeyer states that epistaxis is not infrequent. When the right heart is hypertrophied melæna or hæmatemesis may be produced from obstructed hepatic circulation.

On *palpation* the cardiac area is abnormally increased, and the impulse has a heaving, lifting character. The shock of an hypertrophied heart may be perceptible over the whole precordial space, and in cases of extensive hypertrophy the head of the listener is often lifted by the shock. When the right ventricle is the seat of the hypertrophy, it may cause a strong epigastric impulse. When the left ventricle is the seat of the hypertrophy the apex-beat is felt farther to the left than normal, sometimes three inches below, and three or four inches to the left of the normal position.

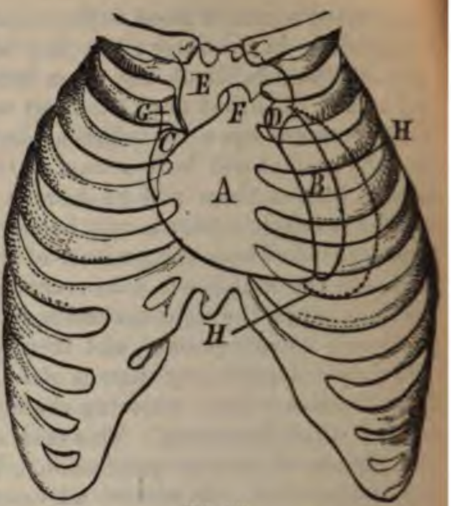

FIG. 107.
Diagram illustrating the Physical Signs in Hypertrophy of the Left Ventricle.

A. Right ventricle; B. Left ventricle; C. Right auricle; D. Left auricle; E. Aorta; F. Pulmonary artery; G. Second cartilage; H. H. Dotted line showing limit of hypertrophy of the ventricle.

On *percussion*, in general cardiac hypertrophy, the normal area of cardiac dulness, both deep-seated and superficial, will be increased to the right, left and downward. The dulness is increased upward only when the auricles are not only hypertrophied but also dilated. If the hypertrophy is confined to the right ventricle the area of dulness may extend considerably to the right of the sternum, sometimes reaching an inch or more beyond the right sternal edge, and extends *lower down* than normal. While if the hypertrophy is confined to the left side of the heart, the area of dulness may extend considerably beyond the left nipple. The area of superficial dulness will also be increased. In eccentric hypertrophy of the left ventricle, the superficial area of dulness will be increased to the left; when the same condition of hypertrophy is present in the right ventricle, the superficial area of dulness will be increased to the right and downward.

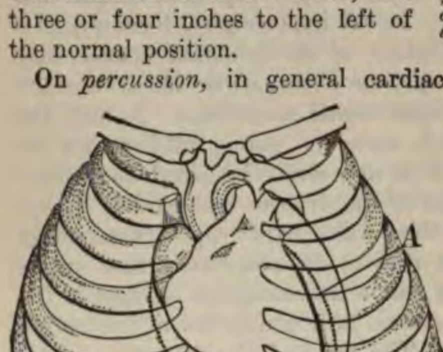

FIG. 108.
Diagram Illustrating the Physical Signs in Hypertrophy of the Right Venticle.
A, A. Limit of hypertrophy.

On *auscultation*, the first sound of the heart, if not accompanied by a murmur, is dull, muffled, and prolonged, and in some cases greatly increased in intensity. The post-systolic silence is shortened. If the hypertrophy is confined to the left ventricle, the second sound heard over the aortic orifice is increased in intensity; if the right ventricle is hypertrophied the second sound over the pulmonic orifice will be increased in intensity. In hypertrophy of the *right* ventricle the first sound is more distinct and more *superficial* than normal, and the second sound is not infrequently reduplicated. In extensive hypertrophy both sounds of the heart often have a metallic ring. There is a diminution or an entire absence of the respiratory murmur over the normal precordial region. When extensive pulmonary emphysema exists, although the heart may be very much increased in size, the increase in the volume of the lungs will prevent appreciation of the increased force in the apex-beat, and the heart-sounds will be diminished rather than increased in intensity. It may, however, be assumed that when extensive pulmonary emphysema is present and is attended by venous pulsation in the neck, there is hypertrophy and dilatation of the right ventricle.

Differential Diagnosis.—Cardiac hypertrophy may be confounded with *cardiac dilatation, thoracic aneurism, mediastinal tumors, consolidation of lung-tissue* surrounding the heart, and *displacement of the heart.* Under certain circumstances pleuritic effusion may be confounded with cardiac hypertrophy. The differential diagnosis of the first four conditions can be better considered in connection with cardiac dilatation.

Displacement of the (normal) heart may be distinguished from hypertrophy by there being no increase in the area of dulness, no change in character or intensity of heart-sounds, and no "heaving" impulse. Besides, certain subjective symptoms, especially those of cerebral hyperæmia, are marked in cardiac hypertrophy and absent in displacement.

Prognosis.—Cardiac hypertrophy admits of a more favorable prognosis than any other cardiac affection. In almost all instances it is compensatory; the urgent symptoms of some other cardiac affection are relieved by it and life is prolonged. Simple cardiac hypertrophy, unless the result of aortic stenosis, may exist for years without the occurrence of any dangerous or very troublesome symptoms. Slight hypertrophy of the left ventricle is very common in those who have led an active life, and have been compelled to perform active and prolonged physical labor; the hypertrophy is no more than is required to maintain an equilibrium in the circulation, and in no way interferes with duration of life. In the young and in athletes, if the cause be removed, the prognosis is very favorable. The patient should not be made aware of the presence of such hypertrophy, for although there is no danger attending it, a knowledge of the fact may greatly alarm him. When there is not only hypertrophy but also degeneration of the hypertrophied walls, the result of imperfect nutrition, the prognosis is very unfavorable.

The prognosis in hypertrophy of the right ventricle is not as favorable as in hypertrophy of the left, because it must inevitably be accompanied by considerable pulmonary obstruction, and consequently is rapidly progressive. In Bright's disease, or when there is disease of the arterial coats, the prognosis is unfavorable. The prognosis in any case of cardiac hypertrophy depends upon the cause of the hypertrophy, and upon the kind of valvular or other cardiac lesion coexisting.

Treatment.—Although cardiac hypertrophy cannot be removed, still, much can be done to arrest its development by removing the causes which produce it, or by rendering them inoperative. Patients with cardiac hypertrophy must especially avoid alcoholic stimulants, immoderate eating, active and prolonged physical exercise and mental excitement. All those conditions which interfere with the general circulation must, if possible, be removed. This embraces interference with the abdominal circulation, as well as with the pulmonary and systemic. Straining at stool and constipation should be avoided by daily keeping the bowels freely moved. This condition of the bowels should be maintained chiefly by habits of life and regulation of diet, cathartics being resorted to only in exceptional cases. As little liquid as possible should be taken into the stomach. Any symptoms of cerebral hyperæmia must be immediately relieved by those means which diminish the force of the heart's action. When the pulse is full and strong and there are evidences of cerebral hyperæmia, it has been the practice of some to bleed, but this treatment is contraindicated, for the presence of anæmia greatly aggravates the dangers arising from cardiac hypertrophy, since it increases irritability and excitability of the heart. The symptoms must be very urgent to warrant venesection.

Of all the remedial agents which diminish the force of the heart's action, I have found aconite the best. When given in full doses it is more reliable than any other means. From two to three drops of Fleming's tincture of the root may be administered every three or four hours. No drug that I have used so fully and promptly relieves the vertigo and other painful sensations that attend cardiac hypertrophy. Hydrocyanic acid, belladonna, and conium are used, but are inferior to aconite. Whenever the dilatation of the cavities exceeds the hypertrophy of the cardiac walls, aconite does harm.

Digitalis is contraindicated, unless there is evidence of heart insufficiency. When digitalis is administered in chronic Bright's disease, although hypertrophy of the left ventricle is one of its constant attendants, failure in the renal secretion indicates the advent of degenerative changes in the cardiac muscle and heart failure. In such cases, the heart, although hypertrophied, is not able to overcome the obstruction to the circulation in the small arteries and capillaries, and the tonic effect of the digitalis raises the heart-power to the point where the obstruction is overcome and the equilibrium of the circulation established. Acetate of lead and veratrum viride are much thought of by many American authorities. For painful palpitation, wild cherry bark is the best drug. Morphine is seldom of service.

CARDIAC DILATATION.

By the term *cardiac dilatation* is understood a condition of the heart in which there is an increase in the capacity of its cavities, with relative diminution of its contractile power. There are three forms :—

I. *Simple Cardiac Dilatation,* in which the capacity of the heart-cavities is increased without any marked change in the cardiac walls. Such a condition is apt to occur during convalescence from any disease in which there has been great impairment of nutrition, such as typhoid fever.

II. *Hypertrophic Cardiac Dilatation.*—In this form there is increase in the capacity of the heart-cavities and increase in the thickness of the heart-walls ; but the relative contractile power of the heart may be diminished as the result of a degeneration following eccentric hypertrophy, or independent of any hypertrophy of the cardiac walls.

III. *Atrophic Cardiac Dilatation.*—In this form the capacity of the heart-cavities is markedly increased, and the cardiac walls are thinner than normal. Sometimes the ventricular walls are not more than two or three lines thick, and the auricular walls may become so thinned that they will present the appearance of a simple membrane. Under these circumstances the contractile power of the heart is almost lost. Anatomically, as well as clinically, the significance of cardiac dilatation is in proportion to the excess of the capacity of the cavities over the thickness of the cardiac walls. A cardiac cavity may be very much increased in capacity, but so long as there is an increase in the muscular power of its walls sufficient to meet the demand for the increased work they are called upon to perform, there will be little or no disturbance of the general circulation. Eccentric hypertrophy and hypertrophic dilatation approach each other very closely, and it is often very difficult to draw the line between them.

Morbid Anatomy.—One or all of the heart cavities may be the seat of dilatation. The shape of the heart is changed according to the cavity which is the seat of the dilatation. If the dilatation is confined to the right ventricle, the heart will be increased in breadth and the apex may appear bifid ; while if the dilatation affects mainly, or only, the left ventricle the heart will be elongated. Dilatation occurs most frequently in the auricles, and thinning of the cardiac walls is most commonly met with here ; next the right ventricle and last of all the left ventricle is the seat of dilatation. When all the cavities are dilated the entire organ is increased in size and assumes a globular shape. When the ventricles are excessively dilated, the trabeculæ are sometimes reduced to the condition of fleshy tendinous cords. When the walls of the left ventricle are very much thinned they collapse when the ventricle is cut into. It is a question whether dilatation ever exists without some hypertrophy. The hypertrophy is apt to be overlooked, for the walls of the dilated cavities seem to be of normal thickness.

The structural changes which take place in the muscular tissue of the walls of the dilated cavities vary with the morbid process which precedes and attends the dilatation. When it results from pericarditis or myocarditis there are serous infiltration and granular degeneration of the muscular fibres; when it is the result of fatty metamorphosis the muscular fibres undergo fatty degeneration. In hypertrophic dilatation it is often impossible, even by a microscopic examination, to determine the exact changes which the muscular fibres undergo; the abnormal state of the muscular fibres can only be determined by the other evidences of feeble heart power.

A heart distended with blood and relaxed by putrefaction may, on first view, be mistaken for a dilated heart. The distinctive marks of a heart softened by the putrefaction processes are its extreme softness, its saturation with the coloring-matter of the blood, and the evidences of decomposition in other parts of the body.

Etiology.—The causes of cardiac dilatation may act by disturbing either one or more of the elements determining cardiac action. They are:

I. *The muscular power of the heart.*
II. *The amount of work required, or the cardiac vascular tension.*
III. *The cardiac innervation.*

I. The contractile power of the heart may be weakened by any of the causes of defective nutrition, as anæmia, deficient food, disturbances in the digestive organs, etc. The physiological decline of the nutritive processes in old age results in a similar condition. In these cases the heart muscle is not necessarily degenerated, but either atrophies or simply loses its normal power without obvious change in its elements. When thus weakened, the normal cardiac tension during diastole as well as systole may stretch the muscle and result in dilatation. Degenerations of the cardiac muscle are prominent causes of decrease in its power; so that all causes inducing fatty, fibroid, or other degenerations are secondarily causes of dilatation.

Prominent among these are the myocardial inflammatory changes associated with pericarditis and endocarditis. Retention and accumulation of excrementitious matter in all forms of Bright's disease is peculiarly liable to cause cardiac degeneration and dilatation, while the perversion of the nutrient elements of the blood, and the presence of poisonous compounds developed in the course of the specific diseases, as typhoid and the other eruptive fevers, diphtheria, septicæmia, and phthisis, are direct causes of those degenerative changes which precede dilatation. That form of dilatation which follows atheroma of the aorta or coronary arteries is also preceded by degeneration.

II. Any increase in the amount of force demanded in the heart to maintain the circulation results directly in dilatation only when that demand is sudden and extreme. Rupture of a valvular leaf or the immediate increase of vascular tension attendant upon some supreme muscular effort may induce acute dilatation in a heart possessing but little reserve power.

The more common and gradually developed obstructions to the circulation found in valvular stenosis, atheroma, capillary fibrosis, the arterial contraction of parenchymatous nephritis, or the compression of large vessels by new growths, induce primarily hypertrophy, which passes into dilatation only when the nutritive processes are no longer able to keep pace with the demand for new tissue and the repair of the old. In these conditions dilatation may supervene either with or without precedent degeneration of the muscle. Emphysema, pulmonary compression, fibroid disease, and like conditions of the lung, are to be included under this head as affecting the right heart.

Increase of intracardiac tension during diastole, caused by valvular insufficiency, produces cardiac dilatation or increase in the capacity of the cardiac cavities; but when slowly developed, the dilatation is so immediately followed by compensatory hypertrophy, unless the muscle is weakened, that the direct result of aortic and other regurgitant lesions were better regarded pathologically as what it is clinically—eccentric hypertrophy rather than hypertrophic dilatation.

It becomes dilatation when cardiac failure supervenes even in the smallest degree.

III. Dilatation is less frequently to be attributed to disturbances in the nerve supply. All forms of excess in the use of narcotics, as tea, coffee, tobacco, opium, chloral, etc., or excesses in venery, and even prolonged and intense mental strain and anxiety, may so disturb the nervous mechanism of the heart as to allow of dilatation under even the normal vascular tension. The nervous condition known as Graves's disease, and some other lesions of the vasomotor system, may also be the cause of cardiac dilatation. It is evident that a single cause of those enumerated is seldom responsible for the cardiac condition. In most cases some form of degeneration is associated with overstrain or a valvular lesion.

Symptoms.—The symptoms that attend the development of cardiac dilatation will depend upon the character and seat of the dilatation. In simple cardiac dilatation the heart walls are of normal power, but the capacity of the cavities is increased, and the amount of blood to be expelled with each cardiac pulsation is greater than normal; consequently there is labored action of the heart (often so great as to be mistaken for the action of an hypertrophied heart), yet the *force* of the heart's action does not increase, and therefore we have a feebleness of the radial pulse. The rhythm of the heart's action will not be disturbed. In that form termed atrophic dilatation there is a very different state of affairs. The heart cavities are not only dilated, but the walls of the cavities are thinner than normal; the heart power is insufficient for the expulsion of the blood from its cavities, and as a result there is a labored action, and the heart, on account of the increased amount of labor, staggers in its action, the arteries are imperfectly filled with blood, the veins become over-distended, the rhythm of the heart's action is disturbed, and the radial pulse becomes markedly feeble and intermitting. These latter points are of special importance as affecting the question of prognosis, for if a patient has all the symptoms of cardiac dilatation without an irregular and intermitting pulse, the prognosis is comparatively good. The same disturbance of the circulation occurs in that form of dilatation which is developed from the degeneration of eccentric hypertrophy.

The first and perhaps the most constant symptom which is common to all varieties of cardiac dilatation, is cardiac palpitation. At times this palpitation is very distressing. There is almost constantly a sense of painful pulsation in the region of the heart. The patient complains of weight, oppression, or uneasiness in the cardiac region, with a sense of fluttering and a tendency to sighing respiration. Very soon after the palpitation has manifested itself, the patient will begin to suffer from dyspnœa on slight exertion; when he is perfectly quiet he suffers very little. As the irregularity of the heart's action and the palpitation increase, the patient's countenance assumes a pale, languid, anxious expression, with more or less lividity of the lips. The extremities are habitually cold. On excitement, or active physical exertion, the entire face and neck become livid; the pulse, which is usually regular, for a time becomes irregular and intermittent. In this condition patients often live some time in comparative comfort; but they are conscious not only of a loss of physical, but also of mental power, and they are troubled with dyspeptic symptoms and a sense of fulness about the epigastrium. Vomiting is not infrequently a troublesome symptom.

As the cardiac dilatation reaches a point at which there is constant cardiac insufficiency, the patient suffers constant dyspnœa, which becomes severe on slight exertion; the cardiac palpitation is always present, and often accompanied by attacks of syncope. The countenance assumes a still more anxious expression, and the lips are always livid; the pulse is constantly irregular and intermitting. With these symptoms there will be scantiness of urine, which will contain albumen and perhaps blood; the feet and ankles become œdematous, the œdema generally extending upward until the patient is in a state of general anasarca. The breathing becomes very difficult, so much so that the patient is unable to lie down, but is obliged to sit with his head inclined forward and resting on some firm support; he is unable to utter more than a single word at a time. The respirations may be thirty or forty per minute, and panting and noisy in character. Cough and expectoration are not uncommon; hæmoptysis may occur, and in some cases pulmonary infarctions form. Petechial extravasations not infrequently occur, especially in dilatation of the right heart. The extremities become cold and blue; the mind wanders, the skin assumes a yellow tinge, and the patient dies from general anasarca with pulmonary œdema or from urinary suppression. During the advanced stage of this affection violent paroxysms of dyspnœa sometimes occur, in some cases of which it seems as though the patient must die, yet they are rarely immediately fatal, but the patient passes from them into a state of coma and, later, dies unconscious. There is always danger from sudden syncope, which may prove immediately fatal.

Although the general symptoms vary greatly in different cases, the physical signs are very distinctive.

Physical Signs.—Upon *inspection* it will be noticed that the area of the cardiac impulse is increased; but it is so indistinct that it will be difficult to determine (by inspection) the exact point where the apex of the heart strikes the walls of the chest. This is especially the case if the chest walls are covered with adipose tissue, or are at all œdematous. Epigastric pulsation occurs in dilatation of the right ventricle. In persons with thin chest-walls, there will sometimes be noticed an undulating motion over the whole of the precordial space. Successive beats strike the chest-wall at different points, and cause the undulatory motion.

Upon *palpation*, dilatation can readily be distinguished from hypertrophy by the feebleness of the cardiac impulse. Although it can sometimes be felt as far to the left as the axillary line, yet there is an absence of the lifting, forcible impulse which attends cardiac hypertrophy. It is often difficult to determine the exact point of its maximum intensity, but it will be accompanied by an undulating motion, wanting in power. Sometimes a purring thrill may be obtained.

Percussion shows a greatly increased area of lateral dulness. The area will be increased to the right if the right side of the heart is dilated, and it may extend to the right nipple. If the left side of the heart is the seat of the dilatation, the area of dulness will be increased to the left, and it may extend well into the axillary space. In general dilatation the shape of the increased precordial area will be oval. This point is of importance in the differential diagnosis between cardiac dilatation and pericardial effusion. The area of the superficial cardiac dulness is not increased in the same proportion as the deep-seated, as is the case in cardiac hypertrophy. Dilated auricles are recognized by an upward increase in the area of dulness, even to the first rib. When the jugular veins are permanently dilated and knotted, the existence of dilatation of the right auricle will not be difficult to determine.

Auscultation.—The sounds of a dilated heart are short, abrupt, and feeble; the second sound is often inaudible at the apex, and the two sounds are of very nearly equal duration and character, so that it is very often difficult to distinguish them. Reduplication of the first sound sometimes occurs. A systolic murmur generally accompanies dilatation ; many authorities regard its production as possible without attendant valvular lesion, from tardy and incomplete contraction of the ventricle. Whenever a cardiac murmur has existed prior to the development of the dilatation, the rhythm of the murmur is lost as the dilatation develops, and it becomes simply a confused murmuring sound. This condition has been denominated *asystolism*. It is a condition in which it is impossible to determine whether the murmur is synchronous with the first or second heart-sound ; pauses or intermissions occur at irregular intervals, which are of more frequent occurrence during exercise than when the patient is quiet. When the asystolic condition is present, the prognosis is very unfavorable, independent of the general condition of the patient ; under such conditions the patient is liable to die suddenly. Asystolism is generally accompanied by a diffused cardiac impulse, which is peculiar, and readily appreciated by the ear as it rests over the precordial space. The respiratory murmur is diminished in intensity over the whole of the upper portion of the left lung.

Differential Diagnosis.—The diagnosis of dilatation of the heart rests mainly on the following conditions :—feeble heart action, undulating impulse, indistinctness of apex-beat, lateral increase in the area of percussion dulness, very nearly square in its outline ; short, abrupt, and feeble heart sounds that strikingly resemble each other, and a feeble, irregular and intermitting pulse, accompanied by the general symptoms of systemic and pulmonary obstruction and congestion.

The differential diagnosis between *cardiac hypertrophy* and cardiac dilatation is never difficult. The heart sounds are intensified in hypertrophy and feeble in dilatation. In both cases there is an increased area of apex-beat, but in hypertrophy it is distinct and forcible, in dilatation it is feeble, diffused and indistinct. The fact that an individual has had cardiac hypertrophy with all its attendant symptoms, but now has a tired expression of countenance, livid lips, and loss of physical vigor, daily becoming more and more marked, and accompanied, it may be, by œdema of the feet, shows that cardiac hypertrophy is giving place to cardiac dilatation. The pulse is full, strong and bounding in hypertrophy, and weak and feeble in dilatation. The first sound is dull, muffled, prolonged, and intensified in hypertrophy ; while it is indistinct and resembles the second sound in dilatation. The face is flushed in hypertrophy : pale, livid and anxious in dilatation. The presence of distended, irregular, turgid jugular veins tells very positively of dilatation of the right auricle ; and pulsation in the jugulars, with feeble heart action and increase in the area of cardiac dulness to the right, indicates dilatation of the right ventricle associated with tricuspid regurgitation. At the same time there will be hepatic, renal, and cerebral disturbance.

The differential diagnosis between *enlargement* of the heart (whether from dilatation of its cavities or hypertrophy of its walls) and *thoracic* tumors is sometimes difficult. One very reliable differential sign is the direction of the increased area of percussion dulness ; thoracic aneurisms and mediastinal tumors always enlarge upward and to the right or left, while in cardiac enlargement the area of dulness is increased laterally and downward. In *aneurism* there is a dilating impulse, vibratory thrill, dysphagia, pain in the dorsal spine and the peculiar aneurismal "bruit."

Consolidation of lung-tissue in the region of the heart may give rise to some of the signs of cardiac enlargement, but the other attending physical signs of pulmonary consolidation will distinguish between the dulness on percussion thus produced and the increased area of dulness due to cardiac enlargements. The character of the first sound of the heart, the pulse, the shape of the dulness and the presence or absence of pulmonary or bronchial symptoms will aid in the diagnosis.

Prognosis.—The prognosis in cardiac dilatation is always bad, and the danger to life is increased in proportion to the excess of the capacity of the cavities over the thickness of their walls. Feebleness of the general muscular system and impoverishment of the blood increase the danger. The presence of disease of the kidney, or other disease of the heart, renders the prognosis in dilatation very grave. If patients have been subject to paroxysms of dyspnœa and attacks of syncope, the prognosis is especially bad, for then there is danger of sudden death. Whenever dropsy exists, the prognosis is immediately unfavorable ; under such conditions few patients, even with the best of care, live more than eighteen months; the majority die within a year. In those cases in which the pulse is regular or only becomes irregular after violent physical exertion, the prognosis is comparatively good; much can be done to relieve symptoms and prolong life. When general anasarca exists and the patient is no longer able to assume the recumbent posture, relief may be given, but it will only be temporary.

Treatment.—Cardiac dilatation is incurable. Even the good effects of palliative measures are temporary. There are, however, two important things to be accomplished. *First*, the nutrition of the body must be maintained at its highest point. *Second*, all irregular or violent action of the heart must be prevented.

To accomplish the first result, the diet must be most nutritious and taken in small quantities and at short intervals. An exclusive milk diet will often be found most advantageous; stimulants must only be taken in small quantities and with the food. When symptoms of anæmia are present, iron may be administered with the food; as a rule it is always safe to administer iron daily to a patient with dilated heart. Strychnia and arsenic may be administered alternately with iron. The greatest amount of fresh air and the best hygienic surroundings should be secured.

To accomplish the second result, this class of patients must be placed under strict rules in regard to exercise. They should never allow themselves to be placed in such circumstances as to render sudden and violent exertion necessary, for a single violent physical strain may jeopardize life. Flannel should be worn next the skin. A dry, bracing air generally best agrees with this class of patients. As regards the medicinal agents to be employed, each case must be studied by itself. All discharges that are exhausting must be arrested. If hyperæmia of the liver and of other abdominal viscera exists, it must be relieved by the administration of small doses of mercury combined with rhubarb and soda; excessive purgation is not admissible, but a daily movement of the bowels without exhausting is important. When there is loss of appetite and impaired digestion, vegetable tonics and mineral acids are indicated.

Those remedial agents which have a direct effect upon the heart itself are important. The most serviceable of this class of remedies is digitalis. It may always be administered in full doses, so long as the amount of urine passed is increased. Often when the feet become œdematous and the patient cyanotic, it has a wonderful effect, entirely removing for a time all unpleasant symptoms. When the heart's action becomes regular, the digitalis may be given in smaller doses, but the small doses must be continued for a long time. If, after a time, the heart's action cannot be controlled by the digitalis, strophanthus, convallaria, or spartein may sometimes be employed with advantage. Belladonna and opium may be given to tranquillize the excited heart, but they should only be resorted to when the digitalis has been thoroughly tested and has failed. In the use of digitalis the same restriction is to be observed which was described in connection with the treatment of other cardiac diseases—that is, it should never be used indiscriminately. It is always desirable to postpone its use as long as possible. Should the heart become nervously excited during the administration of the digitalis, as it often does, the various antispasmodic remedies may be employed. Should cough be persistent, morphine may be given. Paroxysms of dyspnœa may be temporarily relieved by ether, nitrite of amyl, and dry cupping along the spine.

During the slow progress of a chronic case of cardiac dilatation, a great variety of measures may be indicated and afford temporary relief; still, our chief reliance will always be upon digitalis and iron, combined with the most nutritious diet and absolute rest. Ammonia and the diffusible stimulants are rarely of service.

MYOCARDITIS.

(*Carditis.*)

Myocarditis, or carditis, is an inflammation of the muscular structure of the heart; it may be acute or chronic. The chronic form is attended by fibroid changes, general or local. It is met with most frequently in connection with peri- or endocarditis.

Morbid Anatomy.—The diseased process consists in changes which take place either in the primitive bundles of muscular fibre or in the connective-tissue. Both are usually involved, but when the muscular structure alone is attacked, it is called *parenchymatous* myocarditis. When the change primarily affects the connective-tissue it is called *interstitial* myocarditis.[1] Although these two varieties may not be determined during life, they are very readily recognized after death. As a rule, the layers of myocardium just beneath the peri- and endocardium are primarily and chiefly affected. The change may have its seat in any portion of the muscular tissue of the heart; the portion most frequently affected is the left ventricle. The first change noticed is one in color; at first, the muscle is a dark red, later it assumes a grayish, mottled, opaque, buff color, and finally it changes to a dark green.

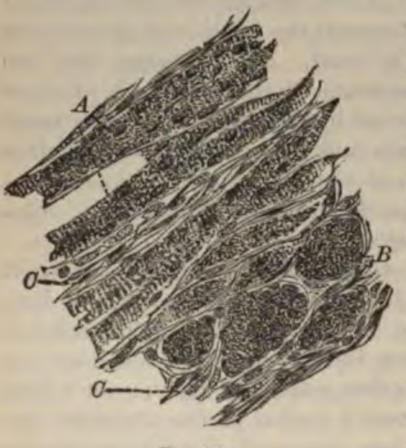

FIG. 109.
Section of Acute Myocarditis.

A. Longitudinal section showing granular muscle-fibres, obliteration of striæ, and prominence of nuclei.
At B the section includes some fasiculi cut obliquely and transversely. The connective-tissue infiltration may be noted at C, C. × 500.

The *microscopical* appearances will vary with the stage of the inflammation. At first the primitive bundles are large, opaque, and swollen from infiltration of serum; their striæ become indistinct, and there is nuclear proliferation. Later the fibrillæ rupture and break down into a finely granular detritus; then the muscular fibre is replaced by connective-tissue, or the degenerative process goes on until there is a breaking down of tissue and the formation of abscesses. When a large amount of the muscular tissue of the ventricular wall is replaced by new connective-tissue formations, the power of resistance of the ventricular wall is diminished, so that during the ventricular diastole the new connective-tissue is liable to become gradually and slowly stretched, and finally it gives rise to aneurism of the heart. This is the manner in which aneurisms of the heart are most commonly formed. Calcareous matter may also be deposited in the newly formed connective-tissue, and then calcareous developments will take place in the walls of the aneurism.

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[1] Cornil and Ranvier deny the existence of Virchow's parenchymatous myocarditis, p. 296.

When the inflammatory process terminates in the formation of an abscess the molecular degeneration replaces more and more the muscular fibres, until finally there are formed swollen yellow-white masses (abscesses) surrounded by red ecchymotic and boggy embryonic tissue, which gives, on section, a small quantity of various colored puriform material consisting of pus and muscular débris. Sometimes the whole cardiac tissue is infiltrated with pus. This form is not met with except in pyæmia and low forms of fever. Abscesses from acute local myocarditis are small; they may burst externally into the pericardium or one of the heart cavities. As a result of this gradual destruction of muscular tissue, rupture of the heart may take place with or without abscesses, and at the post-mortem the pericardium will be distended with blood. *Pyæmic abscesses* are very small and multiple, they may project from either surface of the heart, and the surrounding muscular tissue may be either fatty or granular; bacteria are often present. There may be emboli in the coronary arteries that serve as foci for the pyæmic abscesses.

Etiology.—The causes of myocarditis, endocarditis, and pericarditis are almost identical. Rheumatism, the most frequent cause of pericardial and endocardial inflammation, is a frequent cause of myocarditis. It is maintained by some that endocarditis and pericarditis never occur unless they are associated with *some* myocarditis; but in most cases, the myocarditis is so slight that it little affects the diagnosis or prognosis. Myocarditis may be the result of embolism or degeneration of the coronary arteries. It occurs in connection with all septic dieases, such as pyæmia, septicæmia, typhus, typhoid fevers, and acute ulcerative endocarditis. When it occurs with pyæmia it generally terminates in abscess; when it occurs with rheumatism, it usually terminates in connective-tissue formation, especially at the apex of the left ventricle.

Rheumatic myocarditis may be independent of either peri- or endo-carditis. It most frequently occurs in males before the twenty-fifth year. Sometimes no cause can be discovered. Occasionally it has its starting-point in syphilitic connective-tissue changes. Prolonged high temperature and exposure to cold are mentioned as *possible* exciting causes.

Symptoms.—There are no distinctive symptoms of myocarditis. In a large majority of instances it is impossible to positively determine its existence during life. A rapid, feeble, compressible, and irregular pulse, coming on suddenly in the course of an acute endocarditis or pericarditis, is its most reliable symptom. Restlessness and urgent dyspnœa are common. The face is anxious and cyanotic, there is great restlessness, anxiety, and sometimes delirium. The principal symptoms which should lead one to suspect its existence, are attacks of cardiac palpitation, a feeble, irregular, intermitting pulse, syncope on slight exertion, and all the phenomena of heart failure ; if these come on suddenly in one who is suffering from some severe septic disease there is reason to suspect myocarditis.

There are no *physical signs* except those common to all conditions of heart failure, though at first the heart action is violent. The heart sounds are at first short and sharp, and then feeble. If, however, the myocarditis has terminated in connective-tissue formations, and aneurism of the ventricular wall has occurred it may be recognized by a change in the shape of the heart.

The area of precordial dulness will be increased upward and toward the left shoulder, rather more than when there is cardiac hypertrophy or dilatation. The diagnosis of myocarditis can only be conjectural. When abscess of the heart occurs as a termination of myocarditis, it will probably go unrecognized until the post-mortem. But the sudden occurrence of a murmur indicative of rupture of a portion of the wall or of a valve, along with restlessness, delirium, and rigors, may cause one to suspect it.

Differential Diagnosis.—The existence or non-existence of a murmur alone enables us to distinguish endo- and peri- from myo-carditis. In children it may be mistaken for *acute meningitis.*

Prognosis.—General myocarditis must of necessity prove fatal ; circumscribed myocarditis may be recovered from. The present state of our clinical knowledge of the disease admits only of a speculative prognosis, based rather on our knowledge of its pathological lesions than on any symptoms to which these changes may give rise. Extensive connective-tissue formations, frequently found in the cardiac walls, give evidence that circumscribed myocarditis is frequently recovered from. But the extent and stage at which recovery is possible and the symptoms which indicate fatal termination are still undetermined. It lasts from a few hours to a few days, death occurring from asthenia, heart failure, rupture, aneurism, hæmopericardium, embolism, and secondary septicæmia.

Treatment.—If myocarditis is suspected in the course of an endocarditis or pericarditis, the plan of treatment will not be materially changed. It is essentially the same as that already indicated for the management of those affections. Great care should be exercised not to overtax the heart. This class of patients should never be allowed to go up-stairs or take active exercise until some time after convalescence. Warmth to the extremities is of service, as it tends to equalize the circulation, and thus relieve and prevent cardiac strain. It is probable that many cases of fatty heart are the sequelæ of myocarditis.

Palpitation is an indication for the moderate use of alcoholic stimulants. Digitalis and ammonia should be very cautiously given. Not infrequently septic and fever patients, after violent physical exertion during convalescence, die suddenly ; death under such circumstances may be the result of overtaxing a heart weakened by myocarditis. Besides absolute rest and sustaining measures, all that can be done for these patients is to relieve unpleasant symptoms.

FIBROID DISEASE OF THE HEART.

(*Chronic Myocarditis.*)

As has been stated, acute inflammation of the myocardium ends in abscess or in connective-tissue formation. When fibroid tissue replaces part of the muscular structure of the heart, we have a fibroid heart, or what some call "connective-tissue hypertrophy," a condition analogous to what Gull and Sutton call arterio-capillary fibrosis.

Morbid Anatomy.—The walls of the ventricle are oftenest involved; there may be distinct patches of fibroid tissue or there may be patches just under the endo- or peri-cardium, radiating from which are bands of fibrous tissue which insinuate themselves into the deeper muscular structure. A "fibroid patch" is most frequently found near the apex of the left ventricle. When it is a continuation of endo- or pericarditis the new tissue blends imperceptibly from the lining or covering membranes into the muscular structure of the heart. The tissue is dense, firm, inelastic, and gray-white in color. Sometimes it has a glistening blue or even green appearance; the form of the masses is variable: sometimes they are little spherical projections into some one of the cardiac cavities, and, again, they bulge out into the pericardial sac. They may be dots, streaks, bundles, or islands. The hard tissue interferes materially with the movements of the heart. Aneurism, dilatation, and annular constriction within one of the cavities not infrequently result from interstitial myocarditis. The aneurismal dilatations sometimes contain thrombi.

In "connective-tissue" hypertrophy the heart is enlarged, the weight increased, and the walls are firm, tough, and leathery. The color varies from a pale pink to a deep purple. Gummy tumors are not infrequently found along with fibroid (syphilitic) patches. Under the microscope the muscular tissue is seen atrophied, granular, or fatty; in some places it has entirely disappeared. The apices of the papillary muscles are not infrequently involved in the same process.

Etiology.—All the causes of acute myocarditis are causes of chronic myocarditis. Rheumatism and syphilis are its most frequent causes. In the latter case the fibroid mass is called a "syphilitic patch," but histologically it is identical with non-specific developments. Arterial fibrosis and cirrhotic kidney seem to be associated with its development. It occurs oftenest in males who are past middle life. In many instances no cause can be made out. It has been regarded by some as part of a "senile" change.[1]

Symptoms.—In limited or in slight general fibrosis there are no symptoms. Slight precordial pain, palpitation on exertion or excitement, dyspnœa on active exercise, small and sometimes irregular pulse, and later dropsy and visceral complications are frequent accompaniments of fibroid disease of the heart.[1] Should fibrosis of the columnæ carneæ induce insufficiency on account of shortening of the papillary muscles, then there will be a systolic murmur. But a murmur is usually evidence of the non-existence of fibrosis. The heart sounds may be sharp and short, resembling the sounds "tick tack." The patients emaciate and are very feeble. In connective-tissue hypertrophy, the physical signs are in nowise different from those of ordinary hypertrophy.[2]

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[1] Long continued congestion of the heart, Jenner states, may lead to its induration.

Differential Diagnosis.—The subjective symptoms of chronic endocarditis simulate *fatty degeneration* of the heart. The etiology will aid in establishing a diagnosis, which can never be positive.

Prognosis.—The disease is not immediately fatal, though it is incurable and sooner or later causes death. Its course is chronic, but sudden death is possible at any moment, and a very common ending, being more common than in any other form of cardiac degeneration. Dropsy and congestion and œdema of the lungs are common complications.

Treatment.—To relieve symptoms and aid nutrition is all that can be done unless syphilis be a cause, and then anti-syphilitics often cause marked improvement. They should be administered tentatively in all cases and the results carefully watched. Digitalis is of doubtful efficacy; a restricted diet is an important part of the treatment.

FATTY DEGENERATION OF THE HEART.

This is a common form of cardiac degeneration. It may be circumscribed or diffused. When circumscribed it has a local cause. There are two distinct morbid processes connected with fatty degeneration of the heart.

I. Fatty degeneration of the primitive muscular fibre, termed "Quain's fatty degeneration."

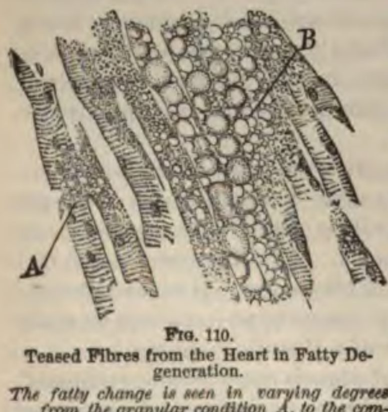

FIG. 110.
Teased Fibres from the Heart in Fatty Degeneration.
The fatty change is seen in varying degrees from the granular condition A, to the complete obliteration of the muscle fibres by fat globules, B. × 400.

II. Fatty accumulation on the surface and in the substance of the heart so as to interfere with its functions.

Morbid Anatomy.—In true fatty degeneration the first change noticed is that the primitive muscular fibres lose their nuclei, their striæ disappear, and they become completely granular. This granular material at first presents the appearance of albuminous matter; soon, however, the sarcous substance gives place to fat granules and to oil globules, which are arranged in rows, and eventually entirely obliterate the muscle fibres. The degenerated fibres are of the same size as the normal fibres. All e fibres are not involved. The muscular tissue assumes a yellow, buff, or rty brown color, and loses its power of resistance, sometimes tearing like aper and readily breaking down under pressure. The heart in most astances is dilated; it may be hypertrophied. When a fatty heart is ypertrophied it is friable, not flabby. The coronary arteries may be theromatous, calcified, obliterated, or normal; when the degeneration occurs secondarily to muscular hypertrophy, the coronary circulation is more or less obstructed and the fatty changes are local. Its most frequent seat is in the left ventricle, and is most marked in the columnæ carneæ and in the inner wall of the heart.

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[1] Ruhle, who has made a careful study of this disease (and considers it as far more frequent and less often diagnosticated than acute myocarditis) regards the irregularity and variability of the pulse, occurring at any moment during the day, as being most characteristic.

[2] Hyde Salter's case was marked by epistaxis and hæmoptysis.

In *fatty infiltration* there is simply an increase of fat in the areolar tissue of the heart; this fat does not interfere with the function of the muscular fibres except by its pressure.[1] If the fatty accumulation is extensive, it may cause atrophy of the muscular fibres. Fatty degeneration may cause death by so weakening the walls of the heart that rupture will take place, or by so weakening the contractile power of the heart as to render it incapable of performing its function.[2] Fatty infiltration may diminish the heart-power, but it rarely, if ever, either directly or indirectly causes death.

Etiology.—All the causes of fatty degeneration of the muscular fibres of the heart are as yet undetermined. It is evident, however, that anything which interferes with the nutrition of the heart tends to fatty degeneration of its walls; it is essentially a disease of middle and advanced life; it comes on with senile decay.[3] It is often a prominent sign of the marasmus which comes on in Bright's disease, chronic alcoholismus (especially when combined with syphilis), gout, phthisis, cancer, etc.; when developed in this connection it never reaches a point where it seriously interferes with the action of the heart. In quite a large proportion of cases, fatty degeneration of the heart is the result of mal-nutrition from some interference with the supply of blood through the coronary arteries. Such interference may arise from atheroma or calcification of the coronary vessels, embolic obstruction, external compression from pericardial thickenings, or impairment of the aortic recoil; it is met with in connection with phosphorus poisoning, changes in the heart in specific evers, acute yellow atrophy, etc. The same degenerative tendency which anifests itself in other tissues of the body, due to constitutional condiions, either hereditary or acquired, predisposes to it.

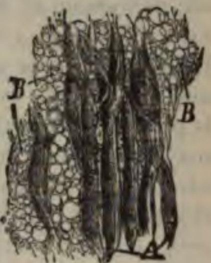

FIG. 111.
Fatty Infiltration.
A portion of cardiac wall from left ventricle, teased and showing the infiltration of fat, B, in the areolar tissue with atrophy of the heart fibres, A. × 400.

Fatty infiltration of the heart occurs as a part of general obesity, which frequently develops after persons have passed middle life. It is quite equently met with in connection with chronic alcoholismus. Sedentary abits increase the liability to its development.

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[1] Normally there is more or less fat in the auriculo-ventricular grooves.

[2] One-half the cases of true fatty degeneration end in rupture of the heart. Partial rupture called ardiac apoplexy" is common; aneurism is not uncommon, and the weakened columnæ carneæ per t of valvular insufficiency.

[3] Hence the French call it "senile cardiac softening."

Symptoms.—Moderate fatty degeneration of the heart will go unrecognized; sudden death has occurred from this cause when there was no suspicion of its existence. As a rule, the progress of the disease is very gradual and insidious, and most of the symptoms which attend its development are due to heart-insufficiency. Persons who are subjects of fatty heart cannot undergo active physical exertion for any length of time, without complete exhaustion; their skin is of a pale, "pasty" yellow color, at times more or less livid. Their extremities are cold and œdema is not uncommon, especially in old age; digestion is feeble; they perspire profusely on slight exertion; they suffer from paroxysms of dyspnœa after physical exercise. "Uneasy" feelings and pain about the heart, and sudden suffocating palpitation of the heart are not infrequent. During these paroxysms the liver enlarges, the respiration is feeble and irregular, often sighing in its character; "Cheyne-Stokes' breathing" is present in some cases, and is regarded as an important symptom. The cardiac insufficiency is progressive. The tissues become flabby; there are evidences of arterial degeneration; the *arcus senilis* is often present. The temper is irritable; there is habitual depression of spirits, disturbance of vision, failure of memory, giddiness and vertigo; sudden cerebral anæmia may occur during excitement, inducing syncope or epileptiform attacks. Frequent attacks of fainting in one who has the symptoms of fatty heart are always alarming. The *pulse* is peculiar: it is always feeble, although it apparently varies in force; it may be perfectly regular in rhythm while the patient is quiet, yet on slight exertion it becomes greatly accelerated and irregular both in force and rhythm. It may be very rapid for some minutes, then suddenly it becomes irregular, not beating more than thirty or forty times in a minute;—this is very characteristic.

In an advanced stage of the disease, in addition to cerebral symptoms already referred to, patients sometimes get into a condition which bears a striking resemblance to a state of anæsthesia. Attacks of angina pectoris sometimes occur in connection with fatty heart. Fatty infiltration of the heart gives rise to no functional disturbance of the organ, and is not attended by any unpleasant or dangerous phenomena. Should atrophy of the muscular substance of the heart, from pressure of the fatty accumulation, occur (which seldom happens), the attending symptoms and results differ in no respect from those already detailed as attendants of fatty degeneration of its muscular fibres.

Physical Signs.—The physical signs of fatty heart are few and not diagnostic.

On *inspection*, the apex-beat will be indistinct.

On *palpation*, no impulse will be detected over the precordial space, or it will only be perceptible when the body is bent forward. If the fatty metamorphosis has occurred in an hypertrophied heart, there will be an undulating motion similar to that which accompanies excessive cardiac dilatation.

On *percussion*, the area of precordial dulness, both superficial and deep, is usually increased. If hypertrophy and dilatation exist it may be very greatly increased.

Upon *auscultation*, the muscular element of the first sound will be indistinct or absent. The valvular element is "toneless," and is followed by an unusually long period of silence.

Differential Diagnosis.—Fatty heart may be confounded with other cardiac degenerations. The differential diagnosis between *cardiac dilatation* and fatty heart is always difficult. In both there may be a feeble, irregular pulse, vertigo, ringing in the ears, and attacks of syncope. A dilated heart occupies an abnormal space in the thoracic cavity, and consequently gives rise to an abnormal area of cardiac dulness; the area of a fatty heart does not exceed the normal area. The muscular element of the first sound may be feeble in dilatation, but it is *never* absent, as in fatty heart. Cerebral symptoms, and Cheyne-Stokes' breathing are marked symptoms in fatty heart and absent in dilatation. If fatty degeneration accompanies cardiac dilatation, there will be a greater disturbance of the heart's action than in fatty degeneration *without* dilatation.

Prognosis.—The prognosis is always unfavorable; its tendency is steadily to advance.[1] Individuals with fatty heart may live for years, but when the disease reaches an advanced stage, life is very uncertain; a fatal termination may occur suddenly from syncope,[2] from rupture of the heart, coma, or as the result of cerebral anæmia; it may also terminate slowly by asthenia, which is usually attended by dropsy.

Treatment.—There is no plan of treatment that can restore the degenerated muscular fibres. The principal thing is, to improve or rather increase the tissue-making power of the blood; to this end, iron, cod-liver oil, and strychnine may be given in connection with a good nutritious diet, fresh air, and physical exercise which must never be sufficient to cause dyspnœa or any irregularity in the heart's action. All active or violent physical exercise and excitement must be avoided; the life of the patient must be that of an invalid. If alcoholic stimulants have been used habitually or to excess, they must be stopped. By avoiding everything that may stimulate the heart's action, and by strict observance of all the laws of hygiene, life may be prolonged. Digitalis does harm.

In fatty infiltration the only treatment which seems to be of any service is to restrict the diet to animal food and place the patient under a systematic physical training, so as to diminish or remove fatty accumulations in other parts of the body. It is upon this principle that Oertel's treatment by mountain-climbing is based. All the excreting organs must be active, so as to relieve the heart as much as possible. Quain says that galvanism applied from the back of the neck to the precordium, by the interrupted current, has been found useful.

AMYLOID DEGENERATION.

Amyloid or *waxy degeneration* of the heart is rare.

Morbid Anatomy.—This form of cardiac degeneration is never met with except in connection with similar changes in other organs of the body, and s due to a constitutional cause; it consists in the formation of a shining homogeneous substance in the primitive muscular fibres, which gives the reaction of amyloid material. It is most frequently found in the walls of the right ventricle, causing its cut surface to present the characteristic appearance of waxy metamorphosis. The primary changes take place in the connective-tissue surrounding the muscle-bundles; it is often associated with syphilitic gummata.

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[1] Rindfleisch states that "new fibrillæ can be formed from cell-elements remaining within the sarcolemma."

[2] Quain says death is sudden in fatty heart in the proportion of five to one to any other mode of death.

Etiology.—Waxy degeneration of the walls of the heart is due to those causes which produce similar degeneration in the other organs and tissues of the body; among these causes syphilis stands first.

Symptoms.—There are no special symptoms attending it, except those which are indicative of cardiac failure. Its existence can only be suspected, never positively determined. If the signs of cardiac failure, with waxy degeneration of other organs, as the spleen and liver, are present in an individual who has never been the subject of rheumatism or any valvular disease, but who has a syphilitic history, there is good reason to suspect waxy degeneration of the heart.

Treatment.—There are no special indications different from the treatment of waxy degenerations in other organs.

PARENCHYMATOUS DEGENERATION OF THE HEART.

Parenchymatous or granular degeneration, or "*cloudy swelling,*" is that variety usually met with in acute (specific) diseases attended by high temperature.

Morbid Anatomy.—The whole heart is soft, flabby, friable, and of a dirty red-yellow, *clouded* appearance. It may be slightly enlarged. The pericardium is dull, clouded, ecchymotic and somewhat œdematous. *Under the microscope* the muscle-fibres are swollen, some of them rupture, and they all have a granular appearance, which disappears on the addition of acetic acid. The striations are very indistinct.

Etiology.—Parenchymatous degeneration of the heart is caused by extensive blood-poisoning with or without high temperature.

Symptoms.—Its symptoms are obscured by those that attend the causative disease. The heart impulse is feeble, the apex-beat is indistinct. The first sound gradually disappears and the second sound becomes indistinct. Violent palpitations are often present.

The *Diagnosis* is made by the character of the pulse and the indistinct apex-beat, common in the course of any acute febrile disease.

The *Prognosis* depends on the conditions under which it occurs. If in the course of any acute specific fever, signs of heart-failure come on, the prognosis is very bad.

Its *Treatment* consists in the prompt and judicious administration of stimulants.

PIGMENTARY DEGENERATION OF THE HEART.

Pigment granules are found in the cardiac muscle-fibres in nearly every case of chronic valvular disease. In atrophy of the heart pigmentation is especially marked, and the particles lie near the axis of the fibres. Pigmentation also occurs in cases of long-standing jaundice. In melanosis we find a pigmentary infiltration of the heart differing from the above by the black color of the granules, by their seat being in the connective-tissue and in the muscular-tissue at the same time, and by their localization in points and circumscribed spots.

This condition has no clinical importance.

ATROPHY OF THE HEART.

Atrophy is a diminution in the size and weight of the heart. When the term eccentric atrophy is used a condition of simple dilatation is indicated. Atrophy may be confined to the walls of one cavity, or it may involve the walls of all the cavities of the heart.

Morbid Anatomy.—Some writers describe atrophy of the heart under the head of simple, concentric, and eccentric; but these terms are hardly necessary, as all cases of true cardiac atrophy are concentric; that is, are accompanied by diminution in the capacity of its cavities. In some cases, wasting of the cardiac muscles is attended by inter-muscular connective-tissue increase; in such cases there will be no decrease in the size of the heart, but a marked diminution in its contractile power. There may be a decrease in size and number of the muscular fibres. The pericardium is puckered and opaque. The coronary vessels are tortuous and prominent. When fatty or fibroid changes have induced by (pressure) atrophy of the heart-muscles, the term "yellow atrophy" has been given to it. Senile ("brown") atrophy is due to extensive pigmentation. There may be no histological change in the muscular fibres, or they may undergo fatty degeneration.

Etiology.—Any chronic exhausting disease, as phthisis, syphilis, cancer, or any disease that is accompanied by wasting of the general muscular system, may produce atrophy of the heart. It is frequently met with in the very aged. Atrophy of the heart may result from the pressure of extensive chronic pericardial effusion. Mediastinal growths may also cause it, by their pressure. Fibrous thickening of the pericardium, causing constriction of the coronary arteries, as well as atheroma and thrombosis of these vessels, may cause partial or complete cardiac atrophy. Abnormally small hearts are not infrequently congenital, and are associated with imperfect vascular and sexual development.

Symptoms.—Cardiac atrophy is usually attended by no special symptoms, as it is rarely met with except in connection with wasting of the muscles of the general system. It is difficult to decide whether the symptoms indicating enfeebled circulation depend upon loss of heart-power or upon general muscular feebleness. The existence of that form of cardiac atrophy which is met with in the aged cannot be positively determined during life. That form which results from local interference with the nutrition of the heart is attended by symptoms similar to those of fatty heart. In both forms, the heart's impulse is feeble and its sounds indistinct, and the apex-beat is to the right of and above its normal position.

Prognosis.—The prognosis depends upon the cause and extent of the atrophy. In extensive atrophy attended by fatty degeneration, and in atrophy depending upon the pressure of a pericardial effusion, the prognosis is unfavorable; the atrophy of old age is not attended by any special danger to life.

Treatment.—All that can be done in this disease is to avoid excessive physical exertion and mental excitement. The food must be nutritious and wine may be indulged in rather freely. Iron, which is so serviceable in other cardiac affections attended by enfeebled nutrition and failure of heart-power, will be found of service in this condition.

RUPTURE OF THE HEART.

Rupture of the heart rarely if ever occurs, unless preceded by degenerative changes in the heart walls. The seat of the rupture is usually in the left ventricle, and it may be single or multiple. The fissure generally runs parallel to the fasciculi of the heart fibres—it may be partial at first, and complete some time after. Complete rupture may vary in size from two inches to an opening only large enough to admit a probe; ecchymoses are usually found around the rent; fluid blood and large coagula distend the pericardium; the rupture usually takes place from within outward, and occurs or commences during the cardiac systole.

Etiology.—Rupture of the heart may follow atrophy, cardiac aneurism, abscess, hemorrhagic softening, fatty and other degenerations of the cardiac walls; its immediate cause is usually some violent physical effort or mental excitement. If it occurs during sleep, or when the individual is quiet, there is reason to believe that it commenced some time before it became complete, and that this apparently sudden rupture is only its completion. It is rare before forty and occurs usually after the sixtieth year.

Symptoms.—If the rupture is complete the patient's hand is suddenly carried to the chest, a few convulsive twitches occur, and unconsciousness and death immediately follow. If the rupture is partial, the symptoms are those of collapse:—rapid, feeble pulse, restlessness, faintness, pallor, cold skin, vomiting, dyspnœa, and perhaps convulsions; death may not occur for several hours. Rupture of the heart sometimes occurs in connection with a paroxysm of precordial pain resembling angina pectoris.

Prognosis.—Death is certain; nothing can avert it. In seventy-five per cent. it is sudden.

Treatment.—Necessarily this can only be palliative. Stimulants and narcotics may be given to afford temporary relief.

CARDIAC THROMBOSIS.

At nearly every autopsy there will be found a dark red clot of blood in the right heart, or in the auricles. This clot will be most firm in those who die of chronic disease ; it will be more or less adherent to the cardiac walls and the trabeculæ and may extend like a cord into the vessels. In phthisis they are usually *very* firm ; in anæmia they are jelly-like and pale ; in leukæmia they are soft, creamy and puriform. In the exanthemata they are very soft, and when an acute disease runs a *very* short sharp course there is often *no clot.* At one time these clots are entirely composed of fibrin, and are of a pale straw-color ; at another time they contain red globules, and are of a dark red color. The coagulum is not infrequently whitish at its upper portion, and deep red at its lower, according to the position of the body. These clots are formed during the last hours of life, and immediately after death. They have no pathological significance. They are often called *passive* coagula.

Morbid Anatomy.—In true cardiac thrombosis coagula are formed in the heart-cavities, either a short time before death, or they may have existed for years. They vary in size from a pin's head to a walnut, and may fill the greater part of one of the heart-cavities. If they are of small size and firmly adherent to the valves or chordæ tendineæ, they are called vegetations. If they are of large size, they are called thrombi, and form in any of the heart-cavities ; they are more or less firmly adherent to the endocardium. Their projecting portion is smooth and globular. In those diseased conditions which interfere with the free circulation of the blood through the heart, thrombi usually form in such portions of the heart-cavities as are farthest removed from the active blood-currents. The constitution of these thrombi varies ; sometimes they are firm, dry and of a whitish color, composed of exsanguinated fibrin ; at other times they have a globular outline, are firmly attached to the endocardium, and have the constitution of cysts.

Cardiac thrombi may remain permanently attached to the endocardium, or they may become separated from it in masses of considerable size, or in minute particles, giving rise either to embolism or septic infection ; they may be detached, and, as "fibrinous balls," lie free in the auricular cavities.[1]

Etiology.—All cardiac thrombi originate in coagulation of the blood. In some instances the coagulation is rapid and the coagula are of large size ; in others, the coagulation is slow, and the coagula are of small size. The conditions which favor these coagulations are, first, obstruction to the passage of blood through the heart ; second, abnormal changes in the composition of the blood ; and third, inflammatory changes in the interior of the heart. Obstruction to the passage of blood through the heart may be due to valvular lesions, cardiac dilatation, or feebleness of the contractile power of the heart, inherent, or from degenerations. The thrombi in the latter case are called *marantic thrombi.* The condition of the blood which favors its coagulation, is that which we find in acute inflammation, rheumatism, Bright's disease, and certain acute infectious diseases, as hemorrhagic variola and puerperal fever. Phosphorus poisoning causes it. Coagulation in endocarditis is due to the roughening of the endocardial surface produced by the inflammation.

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[1] According to the theory of Schmidt, the formation of true or cardiac thrombi is due to condensation of the fibrogenic substance of the blood in contact with an inflamed wall. Hence results a slow coagulation and one that does *not* include the red corpuscles.

Symptoms.—The symptoms of cardiac thrombosis in its gravest form are urgent. At the moment of coagulation, the heart's action becomes frequent and irregular, the pulse is small, weak, and irregular in force and rhythm. Partial syncope, with restlessness and jactitation are combined with symptoms of more or less complete pulmonary obstruction. Dyspnœa is intense, there is active delirium, convulsions, and finally a fatal coma. Pulmonary congestion, infarction and œdema occur. Life is rarely prolonged beyond the third day.

In less grave forms, the symptoms are not so urgent. The dyspnœa is slight, the cyanosis is not extreme, the jugular veins are but slightly distended, the respiration is somewhat hurried, and the pulse is increased in frequency, is intermittent and irregular; the symptoms are those of advanced heart disease. Where the coagula are of small size, and the coagulation takes place slowly, there will be few, if any, subjective symptoms to indicate their presence, and life may not be seriously endangered; these latter cases, however, are rather cases of vegetations forming on the valves and chordæ tendineæ, than true cardiac thrombosis. The dislodgment of a large piece of a thrombus *en masse* may block up a valvular orifice completely, and thus cause sudden death. Arterial embolism results from breaking off of small pieces, and there may be subsequent well-marked pyæmic symptoms.

Physical Signs.—*Inspection* and *palpation* show irregularity in the cardiac impulse. The area of cardiac *percussion* dulness is increased to the right of the sternum.

On *auscultation*, there is marked irregularity in the heart-sounds. New murmurs are developed, or, if murmurs existed prior to the occurrence of the thrombosis, they are increased in intensity. The most common murmur is that indicative of obstruction at the right auriculo-ventricular or at the pulmonic orifice, having its maximum intensity at the xiphoid cartilage and being conveyed to the left of the sternum. Occasionally there will be a murmur indicating obstruction in the left ventricle. If the coagula are of small size, the murmurs are similar to those which accompany endocarditis.

Differential Diagnosis.—The symptoms of sudden shock to the heart, and the systemic effects of sudden intra-cardiac obstruction, taken in connection with the sudden development of a loud cardiac murmur evidently originating on the right side of the heart are sufficient to lead one to suspect the existence of cardiac thrombosis. The only condition which is liable to be mistaken for it is the *rupturing of a valve*, or of one of the *chordæ tendineæ* from ulcerative endocarditis. I know of no means by which a differential diagnosis can be made between them until some time after the occurrence.

Prognosis.—It is unfavorable in all cases of extensive cardiac thrombosis. If the coagula are small, it is possible for them to disappear after a time, or to become changed into vegetations; but large cardiac thrombi destroy life, sometimes in twelve hours, and at other times life may be prolonged for two or three days.

Treatment.—Theoretically, the alkaline carbonates have the power of arresting or preventing the formation of cardiac thrombi, hence some give sesquicarbonate of ammonia in endocarditis and pneumonia, to prevent the formation of heart-clots, which they believe to be very frequently the cause of sudden death in these diseases. There is no positive evidence in favor of, or against this theory. Bleeding, and every agent which has a tendency to enfeeble the heart-power must be avoided. Absolute quiet must be insisted upon and digitalis and opium may be administered in small doses. Alcoholic stimulants must be given with great care, and only to prevent collapse. Formerly many described cardiac thrombosis as "polypi" and polypoid growths in the heart.

ANEURISM OF THE HEART.

Aneurisms of the heart may be fusiform, sacculated, or globular, and they are usually situated in the wall of the left ventricle near its apex.[1] They may be single or multiple, and if multiple, open separately or in common. Sometimes cardiac aneurism looks like an elongated sac winding around the aorta.

Morbid Anatomy.—In most instances, cardiac aneurisms form slowly, and are the result of inflammatory processes in the endocardium and myocardium. These processes (as I have already stated) may convert a small or large portion of the muscular wall of the ventricle into fibrous tissue. The portion so charged yields to the internal blood pressure, and a circumscribed pouch or sac is formed which communicates with the heart-cavity by an opening which may be very narrow, or may be the largest part of the sac. The neck is hard, often cartilaginous, and may be smooth or jagged. As these pouches increase in size, their walls become thinner and sometimes rupture; they may undergo calcification. The wall consists mainly of fibrous tissue with endocardium internally and pericardium externally.[2] Adherent pericardium usually strengthens the sac, which varies in thickness from that of a sheet of paper to a quarter of an inch. These sacs may be partially or completely filled with fibrin, fluid blood, or blood-clots. Aneurisms of the inter-ventricular septum, and at the base, usually esult from the extension of a "valvular aneurism." The heart is usually nlarged.

Etiology.—Among the causes of aneurism of the heart may be included ndocardial, pericardial, and myocardial inflammations, the different forms f degeneration, fibroid changes, and tuberculous and syphilitic new rowths. Rare before twenty, it seems to become more frequent as age dvances. Males suffer twice as frequently as females.

Symptoms.—The symptoms of this affection are obscure. There is nothing in its clinical history which distinguishes it from other diseases of the ventricular walls. In some instances every known symptom of cardiac disease is present.

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[1] In Quain's 56 cases, 52 were in the left ventricle.

[2] The cells are flat and arranged parallel to the surface of the aneurism on account of pressure.

The *physical signs* are equally unsatisfactory and unintelligible.[1] The physical signs of chronic pericarditis, endocarditis, hypertrophy, and dilatation are sometimes all present. In twenty per cent. of cases murmurs exist that replace the heart sounds.

Prognosis.—Sudden death may occur from rupture of the heart into the pericardium, or the patient may be worn out by the attendants of cardiac dilatation.

Treatment.—It has no special treatment. Those means advised for the relief of cardiac dilatation will be found most serviceable.

NEW FORMATIONS IN THE HEART.

Morbid growths, or new formations in the walls of the heart have no clinical importance, and I shall only enumerate them.

Cancer of the heart, as a primary affection, is exceedingly rare; while cancerous nodules in the walls or on the surface of the heart, in connection with general cancerous infection, occasionally occur. It is apt to be associated with cancer of the lungs, or mediastinum. Under these circumstances, the disease usually manifests itself in the form of small circumscribed medullary or melanotic tumors, which are developed either in the heart walls or under the pericardium or endocardium. The surfaces of the heart rather than the substance of the myocardium are affected, and the right heart suffers oftener than the left, although the cancer nodules are nearly always multiple. Encephaloid is the form most frequently met with, and epithelioma is the rarest. When cancer of the heart is the result of extension of cancer from the neighboring parts, large portions of the heart may become transformed into cancerous tissue. Its existence cannot be recognized during life; it is of interest only pathologically. In a few cases local pain, anginal symptoms, murmurs and symptoms generally indicative of heart disease have led to suspicion of cancer of the heart when evidences of cancer existed elsewhere.

Tubercle is found in the heart only in connection with acute general tuberculosis; then it develops in the connective-tissue. Its existence cannot be recognized during life. Both gray miliary and yellow cheesy masses are found at the post-mortem. They are usually situated near the pericardium.

Fibroma, lymphoma, lipoma, sarcoma, and *myoma* are rare forms of circumscribed tumors found in the cardiac walls, or under the endocardium or pericardium. Their existence cannot be determined during life.

Parasites.—The heart may be the seat of parasites. The echinococcus, the cysticercus, and entozoa have all been found in the heart-walls, and have been known to lead to their rupture, causing death. Three and one-half per cent. of the cases of hydatid disease occur in the heart.[2] They project into the pericardium or into the heart-cavities as cystic tumors. The sac may rupture in either direction, giving rise to embolism or to pericarditis, usually with hæmopericardium.

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[1] Extensive dulness down and to the left accompanied by a feeble impulse may cause one to suspect it.

[2] Cobbold states that hydatid cysts in the heart are commonly multiple.

True *cysts*, containing serum or grumous fluid, are very rarely found in the heart-walls. All of these developments have the effect of depressing or interfering with the heart's action, but their diagnosis in most cases cannot be made.

TUBERCULOSIS OF THE PERICARDIUM.

Tuberculosis of the pericardium is only met with in connection with acute general miliary tuberculosis. Unless the tubercular development takes place only a short time previous to death, it will give rise to pericarditis. Its presence may be suspected from the existence of the pericarditis in connection with the symptoms of general tuberculosis. In these cases tubercles may develop in the layer of fibrinous exudation or be in the visceral membrane itself. Hemorrhage is common when the neoplasm is accompanied by pericarditis.

CANCER OF THE PERICARDIUM.

The pericardium may be the seat of *cancer*, but the cancerous development is nearly always secondary to cancerous developments in other parts of the body. It may comport itself (as to pseudo-membrane and exudation) precisely like tubercle in the pericardium. More frequent than either is the formation of tuberculous or cancerous masses in the lung or mediastinum, which by pressure and nearness to the pericardium excite fatal pericarditis, by some called cancerous or tuberculous pericarditis.

CARDIAC NEUROSES.

The two prominent neuroses of the heart are *nervous palpitation* and *angina pectoris*. Both are functional disorders.

Nervous Cardiac Palpitation.—As has already been stated, cardiac palpitation is a very common symptom of organic disease of the heart. A purely nervous cardiac palpitation may occur independently of organic heart disease. It comes on suddenly, and is generally intermittent. Indeed, all cardiac neuroses have a paroxysmal character.

Morbid Anatomy.—There are no known anatomical changes either in the heart, or in its nerve-supply, which can be regarded as the constant causes or concomitants of cardiac palpitation.

Etiology.—The direct cause of this affection is over-stimulation of the cardiac muscle, or the excitability from functional derangement of the vagus or cardiac ganglia, which is either induced by direct or reflex causes. Violent physical exercise, or indulgence in intoxicating liquors will accelerate the circulation and give rise to a form of cardiac palpitation, which ceases as soon as the cause is removed. Blows on the epigastrium cause it. Adults with contracted chests, and young persons about the time of puberty, whose growth has been rapid, often complain of palpitation. In these cases it seems to be caused by the narrowness of the chest, which interferes with the free play of the heart. Palpitation is a very frequent symptom in states of debility or anæmia. Under this head are included sexual excesses, chlorosis, enervating habits, diabetes, and all acute infectious diseases that are attended by extensive nutritive disturbances, as typhoid fever, scurvy, etc.

Cardiac palpitation is of frequent occurrence in persons with what is called a nervous temperament, induced by late hours, the habitual use of strong tea and coffee, the inordinate use of tobacco, derangements of the digestive organs, sudden shock or fright, chorea, etc. The excessive habitual use of aconite and digitalis is known to have caused it. Cardiac palpitation is frequently met with in those with a gouty diathesis and chronic diease of the liver, accompanied by dyspeptic symptoms which are attended by flatulence. It is more common in women than in men, and often seems distinctly allied to hysteria.

Symptoms.—In a perfectly healthy subject with a well-formed chest, the cardiac impulse is so slight that the motion is not perceptible, unless the hand be applied to the precordial space. Whenever a person becomes sensible of the beating of his own heart, he may be said to have cardiac palpitation; by the term is understood an unnaturally strong cardiac impulse accompanied by an unnaturally rapid action of the heart, which may be irregular or intermitting. Sometimes there is a loss of three or four beats which causes a sense of oppression or even of impending death. It may be accompanied by a choking, paroxysmal, "fluttering" sensation. In some cases the impulse communicates a quick shock to the chest walls; in other cases the impulse is prolonged and heaving in character, and in others is weaker and. almost imperceptible. The heart-sounds may be so increased in intensity as to be audible to the patient when he lies on his left side. There may be precordial pain, but usually it only amounts to precordial "anxiety." The carotids throb; the heart may beat from thirty to one hundred beats in a minute; the impulse and sounds increase and diminish at the same time; the fits of palpitation may come on suddenly, and be of short duration, or they may come on gradually, and be protracted and severe. Murmurs are usually due to the accompanying anæmia. Reduplication of the second sound is quite characteristic. Sometimes there is extreme dyspnœa and headache, vertigo and ringing in the ears, and photophobia. The mind may be bewildered and the patient may stagger, yet no paralysis or vertigo exists. The respirations are irregular or oppressed, with dyspnœa and a short, dry cough.

Differential Diagnosis.—To distinguish between cardiac palpitation independent of organic disease of the heart, and cardiac palpitation depending upon *organic cardiac disease*, is of the greatest importance. Cardiac palpitation independent of cardiac disease comes on suddenly, and is not constant, whereas organic cardiac palpitation comes on slowly and is persistent. In functional palpitation, all the physical signs of organic cardiac disease are absent. Persons free from organic heart disease complain more frequently of palpitation than those who are the subjects of organic disease; palpitation of organic heart disease is increased by exercise.

Prognosis.—In cardiac palpitation independent of organic heart disease, the prognosis is always good; although it may cause the patient great uneasiness, it never destroys life.

Treatment.—In each case of cardiac palpitation it is important to find out and, if possible, remove its cause. Anæmic subjects should take iron in large doses for a long period. In hysterical palpitation all uterine derangements must be relieved. If the excessive use of alcoholic stimulants, tobacco, strong tea or coffee, is the cause, it must be stopped. Occurring in a gouty subject, those means which have been found to relieve gouty manifestations must be employed. Those in whom no special cause can be found, should be directed to sponge the surface of the body night and morning in cold water, exercise moderately in the open air, and live on a nutritious diet.

During the attacks, relief will usually be obtained by the administration of some of the more reliable nervines and diffusible stimulants. Narcotics generally do harm. Digitalis should never be given in purely nervous cardiac palpitation. Ether, ammonia, chloral hydrate, and the bromides are occasionally useful; sometimes camphor, assafœtida, musk and valerian are serviceable as anti-spasmodics. A very important element in the successful management of an attack of nervous cardiac palpitation, is the positive assurance of the medical attendant that there is no danger attending the paroxysm, and that there is no disease of the heart.

ANGINA PECTORIS.

Angina pectoris is a neurosis of the heart due to organic changes in its structure or to diseases involving its nerve supply; strictly speaking, it is a symptom or a collection of symptoms of organic cardiac disease. It has no special morbid anatomy.

Etiology.—There is no form of cardiac or aortic disease with which angina pectoris has not been found associated, and there is no form with which it is invariably or even generally present. Inherited, nervous or "neuralgic" tendencies predispose to it; eighty per cent. of cases occur after the fortieth year. Gout, albuminuria, diabetes, and certain hepatic diseases are often associated with it. Trousseau dwells on the relationship between angina pectoris and epilepsy. There are, however, two forms of heart disease with which it is especially liable to occur:—obstruction to the coronary circulation, and fatty degeneration of the heart.

The other diseased states with which it is liable to occur are, insufficiency of the aortic valves, with a rigid dilated state of the ascending portion of the arch of the aorta, combined with dilatation of the left ventricle. When these conditions exist, angina pectoris will not occur unless the heart's action is suddenly disturbed, or its movements impeded by some mechanical cause.

Symptoms.—The symptoms which attend an attack of angina pectoris are quite characteristic. The patient is suddenly seized with an intense agonizing pain in the precordial region (usually commencing on a level with the xiphoid cartilage) extending through the back and along the left arm. This pain is of a stabbing or lancinating character and produces a sensation of impending suffocation—a feeling as though death was near at hand. There may be true laryngeal pain. At the commencement of this pain the countenance becomes deadly pale and is expressive of extreme anxiety and suffering; the surface is covered with a cold perspiration, the pulse falters, and may be almost imperceptible, the respiration is short and hurried, the face livid, and the patient is unable to lie down or even to move, for the least motion aggravates his sufferings. His consciousness is undisturbed, and his spinal as well as his cerebral functions are unaffected, but there may be slight wandering as the attack passes off. Not infrequently the rhythm of the heart's action is undisturbed and the patient does not even experience palpitation. Sometimes the action of the heart is so much deranged that syncope or even sudden death occurs. The pulse may be slow and feeble or markedly irregular. Usually after the paroxysm has continued for a few moments, or at the longest an hour, it gradually subsides. The attack may come on during sleep.

At first, there are long intervals between these attacks, but after a time they become frequent. Between the attacks the general health may be unimpaired.

Differential Diagnosis.—Angina pectoris may be confounded with *spasmodic asthma, hysteria, intercostal neuralgia, myalgia*, and the first stage of *acute pleurisy*.

Although the phenomena attending a paroxysm of angina pectoris may bear a striking resemblance to those of *spasmodic asthma*, a physical examination of the chest will detect the presence or absence of the characteristic physical signs of the asthma, and thus lead to a correct diagnosis.

The intermitting and irregular character of the pulse in angina pectoris will distinguish it from an *hysterical* paroxysm.

In *intercostal neuralgia*, the duration of the attack, the points of tenderness, the direction of the pain, and the absence of cardiac disturbance, will distinguish it from angina pectoris.

Myalgia and *acute pleurisy* may simulate angina pectoris. In each, acute pain and catching breath are present; but the condition of the circulation, taken in connection with the locality of the pain and the physical signs of pleurisy, will generally decide the question.

Prognosis.—The prognosis in angina pectoris is necessarily unfavorable. Sometimes the first attack proves fatal; in more instances the second or third, while in many more, perhaps in the majority of instances, the patient at irregular intervals experiences a succession of attacks, each paroxysm being more severe than the previous one, until finally, after a period extending from one to six or eight years, an attack occurs in which the heart's action is arrested and death ensues. The later attacks are excited by trivial causes, or apparently come on spontaneously. The tendency of angina pectoris associated with organic disease of the heart is to grow steadily worse, and terminate in death within a year.

Treatment.—During an attack, means should be employed to alleviate or arrest the paroxysm ; during the interval the exciting cause should be removed or its predisposing power diminished. It is doubtful whether there are any remedial agents that have the power to arrest or very greatly relieve a paroxysm. Diffusible stimulants, sedatives, and anti-spasmodics have all been employed, but so far as my experience goes they have no power to alleviate or arrest the paroxysm. Rest, and the free administration of digitalis, are of the greatest service. Chloroform should not be used. An emetic for an overloaded stomach, or hot foot baths, etc., when cold causes a paroxysm, are often advantageous. Quain and many others advocate the nitrite of amyl, ♏ v—vi, inhaled from the handkerchief ; nitro-glycerine (1-100 ♏ a dose) is very useful, and hypodermatics of morphine may be given in conjunction with it. It is well for those who suffer from angina to carry constantly either the nitrite of amyl pearls or the nitro-glycerine tablets.

During the interval all violent emotions and all active physical exercise must be avoided. Indigestion, or flatulence, when present, should be relieved by careful attention to the diet. The only medicinal remedies which I have found of service in delaying and rendering less severe the paroxysm of angina pectoris are iron, strychnine, and arsenic ; these should be administered daily in small doses. Phosphorus and zinc are useful in "nervous temperaments." When angina pectoris is associated with fatty heart, the rules given for the management of the latter disease should be observed. Quain states that a continuous current, the + pole on the sternum and the – pole on the lower vertebræ, has often produced marked amelioration of anginal paroxysms. Trousseau strongly advises belladonna given continuously in small doses, on the ground of the analogy of the affection to epilepsy.

HYDROPERICARDIUM.

(*Dropsy of the Pericardium.*)

Hydropericardium is a sero-albuminous effusion into the pericardial sac, non-inflammatory in character, and when absorbed leaves no trace behind it. It is often very abundant and a source of great discomfort to the patient, but rarely directly causes death. The effect of such fluid effusions is to embarrass the action of the heart, while the heart-fibre becomes pale and is easily torn, the result of the serous infiltration. Six, seven, or more ounces of fluid are usually found, of a yellow, green, red, or red-brown color. Thirty-three per cent. of albumen is usually present, and a small amount of fibrinous matter that coagulates on exposure to the air.

Etiology.—Non-inflammatory effusions into the pericardium occur most frequently in connection with renal and cardiac diseases. In that form of renal disease which complicates scarlatina, it is especially liable to occur, and under such circumstances it is passive in character and is soon reabsorbed on the restoration of the renal function. When it occurs in chronic forms of Bright's disease, it is more serious and obstinate. When it accompanies chronic cardiac disease it is the result of the general venous congestion, and its pressure greatly embarrasses the already enfeebled heart. It may result from any disease where there is, from physical causes, a tendency to serous transudation into the cavities of the body.

Symptoms.—The *symptoms* and the *physical signs* which attend such effusions do not materially differ from those already detailed as marking the stage of fluid effusion in pericarditis, except that there is entire absence of any febrile disturbance. There is no friction sound present at any time during the progress of the effusion. It is an early symptom when due to heart or lung disease; and occurs late when due to splenic, hepatic, or renal disease. It occurs *very* late in the tuberculous and cancerous cachexiæ.

Prognosis.—In chronic Bright's disease and in advanced cardiac disease, it is usually the precursor, although it can scarcely be called the cause of death. In other conditions the prognosis will depend upon the circumstances which attend its development.

Treatment.—In the treatment we must be guided by the peculiarities of each case. All the measures recommended for the treatment of hydrothorax may be employed in the treatment of hydropericardium. To find out and remove its cause is of the greatest importance; in other words, treat the diseased condition which gives rise to, or permits the effusion. Only in scarlatinal albuminuria is the accumulation so sudden that paracentesis may be demanded.

PNEUMOPERICARDIUM.

Pneumopericardium, or air in the pericardial sac, is the result either of a perforating wound of the thorax, or the perforation of the pericardial sac by an ulcerative process and the admission of air from some organ naturally containing it—stomach, intestine, lung, or œsophagus; or to the putrefaction of an exudation.

The *diagnosis* of this accident rests on the tympanitic percussion sound over the pericardial space, and the tinkling, splashing, or metallic sound heard directly over the heart. With the exception of those cases which are of traumatic origin, this accident rapidly proves fatal; 80 per cent. die in non-traumatic and 50 per cent. in traumatic cases. Its treatment is altogether symptomatic.

HÆMOPERICARDIUM.

Hæmopericardium,[1] or blood in the pericardial sac, may be of traumatic origin, or may result from rupture of the heart, or, far more frequently, the pericardium becomes distended with blood from the rupture of one of those small aortic aneurisms which develop on that portion of the aorta included within the pericardial sac. Unless of traumatic origin, it rapidly proves fatal, and will be found at the autopsy of many cases of sudden death. When of traumatic origin, the effused blood is not often absorbed.

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[1] Hæmopericardium is non-inflammatory. Blood may fill the sac when inflammation exists; then the name hemorrhagic pericarditis is applied.

SYPHILITIC DISEASE OF THE HEART.

There may be two manifestations of syphilis in the heart,—the *fibroid patch* and the gummy tumor or *gumma.*

Morbid Anatomy.—Pale, yellow, gummy masses are found, usually intimately blended with the cardiac substance, but often projecting as nodules from its surface. At first they are elastic, firm, homogeneous, often *very* hard ; later they soften and become cheesy. They may become fluid and open inward and give rise to cardiac aneurism. As a rule the cheesy products are absorbed and a puckered, fibrous scar remains at their site. Sometimes the gummata—which are nearly always multiple—appear as " infiltrations " or " deposits." They may occur in *any* portion of the heart. When the outer zone of a gumma undergoes development into fibroid tissue, the caseous portion remains as a compact mass. Bruce regards this as an intermediate form between the fibroid patch and the true gumma or "syphiloma." The myocardial vessels are not infrequently the seat of (syphilitic) *endarteritis obliterans,* giving rise to infarctions in the wall of the heart ; and the pericardium is commonly found adherent.

Etiology.—Fibroid patches and gummata arise both from congenital and acquired syphilis.

Symptoms.—Symptoms of cerebral or visceral syphilis may and often do mask those of the cardiac affection. Should the puckered fibroid tissue narrow or distort any part of the heart, or involve the valves to such an extent as to cause obstruction or allow of regurgitation, then a murmur—differing in no respect from other murmurs—will be the chief symptom. Syncope, infrequent pulse, palpitation, dyspnœa, choking, and many other symptoms of heart disease have occurred in the few recorded cases of syphilis of the heart.

The *diagnosis* rests mainly on the exclusion of all other forms of heart disease, and the evidences of syphilis, hereditary or acquired, in the individual.

The *prognosis* would be more favorable than, probably, with any other similar condition, on account of its amenability to *treatment,* which, of course, is purely anti-syphilitic.

BASEDOW'S DISEASE.

(*Exophthalmic Goître.*)

Basedow's or Graves' disease is an affection in which there is enlargement and hyperæmia of the thyroid body, protrusion of the eyeballs, cardiac palpitation, and anæmia. It is closely allied to functional cardiac diseases.

Morbid Anatomy.—It is attended by no constant morbid lesions. The enlargement of the thyroid body is due to a dilatation of its vessels. The protrusion of the eyeball is caused by dilatation of the vessels behind the globe; both of these changes appear simultaneously with derangement of the circulation, and *cardiac palpitation.* There are many circumstances which render it probable that the enlargement of the vessels is due to some vasa-motor disturbance which allows of their passive dilatation in the neck, the thyroid body, and the orbit; at the same time it causes an excited action of the heart. The thyroid body may be filled with cysts or be the seat of hyperplasia. Atheroma of the ophthalmic arteries has been found.

Etiology.—It rarely occurs in males. It is met with in women between twenty and thirty years of age. A "neuropathic tendency" is usually strongly marked. Menstrual derangements attended by violent mental emotions of various kinds often precede its development.

Symptoms.—This disease may come on suddenly or slowly; if it develops slowly, the patient will at times for a long period complain of severe attacks of cardiac palpitation, and pulsation in the arteries, gradually these attacks of palpitation will become more frequent and severe, the eyes will become slightly prominent and staring, and after a time they may become so prominent that the lids will not cover them. Occasionally the insertion of the recti muscles can be seen. The protrusion is often greatly increased under excitement. The attacks of cardiac palpitation grow more severe, the thyroid gland visibly enlarges, and the eyes become lustrous and projecting.[1] On casting the eye down, the eyelids follow but slowly;—this gives a peculiar look to the patient. Vision is not usually disturbed, but there may be slight loss of co-ordination. In proportion as the eyes bulge the eyelashes and eyebrows fall out. Diplopia, traceable to paresis of the right trochlearis has been noted. Profuse lachrymation is not uncommon. Exophthalmus is often more marked on one side than the other, and is then apt to be attended by enlargement of the thyroid body on the *opposite* side.

The thyroid gland usually enlarges slowly; the patient's attention is first attracted to it on account of a continued pulsation of the lower part of the neck. It is usually unequally enlarged, is soft, elastic, and at first pulsates, due to the dilatation of the vessels in the gland; after a time there is increase of tissue, and blowing sounds are audible over the enlargement.

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[1] Eulenberg regards increased development of fat in the cellular tissue of the orbit, as, in part, the cause of the bulging of the eyeballs.

There may be a change in the pitch of the voice, perhaps from pressure of the enlarged gland on the recurrent laryngeal nerve. Sometimes the voice is hoarse or *entirely lost.* There is always danger from pressure of the enlarged thyroid gland upon the trachea. The *cardiac* palpitations are rapid and irregular, the pulse-rate varying from one hundred to one hundred and forty per minute. The heart-sounds are loud, and a soft, systolic bellows-murmur may be heard at the base and in the large arteries. There may be a distinct thrill. The carotids may be dilated. The circulation is rapid, the veins filling rapidly, and the pulsation of the small arteries is felt by the patient. Mental emotion and violent physical exertion bring on attacks of palpitation, which may be so violent as to produce a visible enlargement of the precordia with every beat. Stimulation of the *accelerator nerves* of the heart probably causes the palpitation.[1]

Debility, anæmia, indigestion, anorexia, and diarrhœa may be present during the whole course of the disease. Insomnia, amenorrhœa, and hysterical symptoms are very frequently observed in nervous females. In a few instances the temperature is often elevated to 103° F., and followed by profuse sweats.

Differential Diagnosis.—When the three classical symptoms are present in a female, viz., bulging of the eyeballs, cardiac palpitation, and enlargement of the thyroid, a mistake in the diagnosis will scarcely occur. Von Graefe makes a diagnosis on the "want of harmony between the movement of the eye and its lid."

Cystic goître is not accompanied by exophthalmus, nor by paroxysmal enlargements. The thyroid in Basedow's is far more elastic than in cystic goître.

The lustrous appearance of the eye suffices to diagnosticate it from prominence due to *heart disease* (*e. g.*, hypertrophy), which latter would give evidence of organic changes.

Local orbital or cranial causes of exophthalmus are excluded by the absence of squint and other cerebral symptoms.

Prognosis.—This must always be guarded. The younger the subject, the more favorable the prognosis. It may increase for months, remain stationary for a year or two, and then gradually decline, but not wholly disappear. In some instances its course has been acute and rapid. *Recovery* occurs in from four to five per cent. of cases. Great improvement has occurred in from thirty to forty-five per cent. of cases. It does not directly cause death, but intercurrent affections are generally ill-borne and fatal. Any heart disease (organic), great anæmia, or evidence of the "neuropathic disposition," renders the prognosis unfavorable. Pregnancy is said to have a favorable influence.[2] Death may occur from valvular disease of the heart, pulmonary tuberculosis, gangrene of the extremities, pulmonary apoplexy, or œdema.

Treatment.—The first remedies proposed in the treatment of this affection were quinine and iron, and their use is still followed by the best re-

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[1] Friedrich's ingenious theory is that, the vaso-motor nerves being paralyzed, dilatation of the coronary artery follows, and hence there is increased excitement in the ganglia of the heart.

[2] Trousseau and Corliea.

sults. Traube gives them alternately, five grains of quinine one da
ten grains of iron, in the form of Vallett's mass, the following day.
senic does harm. Iodine is condemned by some and recommenc
others. It has been claimed that belladonna, hydrocyanic acid, anc
tranquillize the heart. Galvanization of the cervical sympathetic
ishes the exophthalmus and lowers the pulse-rate : it is to-day the
ite plan of treatment with many. Hydropathic treatment is
praised by some French authorities. It seems to me important that
cases, and especially when chlorotic conditions are present, hepatic
lation form an important part of the treatment, and that the diet
stricted to meat and milk as far as possible.

DISEASES OF THE BLOOD-VESSELS.

Under this head will be considered the following diseases of the *arteries* and *veins*.

DISEASES OF THE ARTERIES.

- I. *Acute Endarteritis.*
- II. *Chronic Endarteritis, or "Atheroma."*
- III. *Periarteritis.*
- IV. *Degenerations: Fatty, Waxy, and Calcareous.*
- V. *Arterio-capillary Fibrosis.*
- VI. *Syphilis of Arteries.*
- VII. *Atrophy, Hypertrophy, Dilatation, Narrowing.*

DISEASES OF THE VEINS.

- I. *Acute and Chronic Phlebitis.*
- II. *Dilatation of the Veins.*
- III. *Embolism and Thrombosis.*

ACUTE ENDARTERITIS.

Acute endarteritis is an inflammation of the tunica intima, which is formed of endothelium lying on longitudinally arranged elastic tissue. As an isolated lesion it is rare.

Morbid Anatomy.—Along some vessel, chiefly the aorta, numerous elevated round patches are seen projecting from the internal layer. They are red, opalescent, soft, and elastic (" gelatinous patches of the aorta "). The elevated patches are made up of embryonic cells arranged in parallel lines. These patches may undergo ulceration. Fibrin may form on their surface and inclose either the white blood corpuscles or the proliferated and free elements of the diseased surface. Pus formations and gangrene may result. The middle coat is not extensively involved, but a periarteritis nearly always occurs. The whole vessel becomes friable. Emboli form, and coagulation may result in arterial thrombosis.

Etiology.—Wounds, emboli, extension of inflammation from without, and irritation from a hard vegetation may cause it. Acute aortitis is usually of rheumatic origin. A purulent aortitis is described by some as occurring in septic conditions.

Symptoms.—There are no special symptoms by which it can be distinguished. When coagula are formed thrombi result, and then the symptoms will be those of thrombosis complicated by pyæmia.

CHRONIC ENDARTERITIS.

Atheroma, or *endarteritis deformans*, is a common disease.

Morbid Anatomy.—It is an inflammation of the internal coat, with thickening in patches, the thickening being due to multiplication of the cellular elements :—granular fatty degeneration of these elements and of the middle coat follows, and a yellow atheromatous focus is produced, separated from the blood current by a thin tense pellicle. As the atheromatous changes take place, the centre of the patch contains a putty-like mass of cholesterin crystals, fat granules, and crystals of fatty acid. When there is little or no fatty change in the patches, the process is called "*sclerosis*," and they are then stained dark brown. When these processes progress slowly, calcareous granules infiltrate the tunica intima, and, later, form thin, friable calcareous plates just underneath an internal pellicle. The vessels above the heart in the thorax are most frequently affected; they become enlarged, irregular, and friable. When the stiffened internal coat breaks, chinks are formed which fill with blood and later become melanotic. In the aorta the middle coat often disappears, connective-tissue taking its place; destruction of the middle coat is the only cause of spontaneous aneurism of the aorta. The external coat finally assumes the histological characters of the altered internal coat. After the aorta, the cerebral, coronary, and splenic vessels and those of the lower extremity may become involved. In the small vessels, narrowing and stenosis are the results of chronic arteritis. Cardiac hypertrophy is a common result of the rigidity and inelasticity of the aorta and its branches.

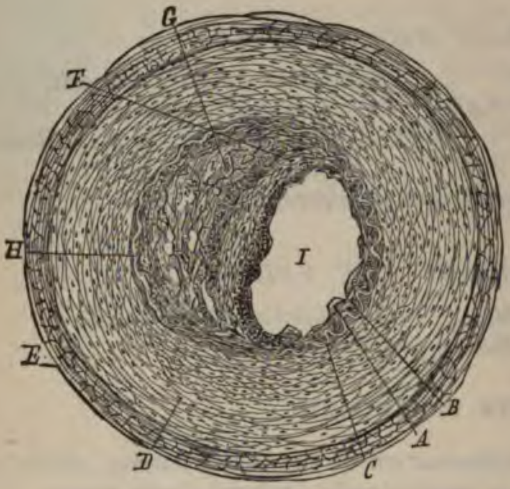

FIG. 112.

Chronic Endarteritis : Atheroma.

Magnified View of a Small Artery, partly diagrammatic.

A. *Endothelium of artery, cells turned inward.—B. Intima thrown into folds by C, the elastic lamina.—D. Muscular coat (tunica media).—E. Adventitia.*
F. *Swollen and proliferating intima.*
G. *Irregular spaces containing calcareous, granular, and fatty matter.*
H. *Elastic lamina, limiting the degenerative process.*
I. *Lumen of the vessel encroached upon one side only.*

Etiology.—Atheroma or arterial sclerosis is a disease of advanced life. Men are far oftener affected than women. It is predisposed to by gout, rheumatism, syphilis, Bright's disease, lead poisoning, and especially by alcoholismus. Over-strain of the vessel is often its immediate cause. It is said sometimes to be an extension from the endocardium into the aorta.

Symptoms.—Nearly all its symptoms are the mechanical results of rigidity of the artery. The left ventricle is hypertrophied. The peripheral arteries are enlarged, lengthened, and tortuous, and an irregular outline is readily felt along their course. The pulse is feeble, sometimes imperceptible; the sphygmograph shows a short up-stroke and a flat summit (pulsus tardus). The extremities are cold and parts may become gangrenous (senile gangrene). Apoplexy may occur, and some ascribe epilepsy and senile dementia to atheromatous arteries. The different organs atrophy, the skin becomes dry, and the lungs are frequently emphysematous. Dissecting aneurisms may be induced after a rupture of an atheromatous abscess; persistent anasarca of the legs in old men is often due to calcified arteries.

Differential Diagnosis.—Aortitis sometimes gives rise to symptoms that can establish a diagnosis. These are acute substernal pain with oppression, palpitation, quick and feeble pulse. With these symptoms may be associated a hard systolic murmur, originating at the seat of inflammation, and transmitted to a distant point of the aorta. Paroxysms of pain like angina pectoris are sometimes marked.

Prognosis.—It is a condition which cannot be cured.

Its *treatment* is altogether hygienic.

PERIARTERITIS.

In periarteritis the adventitia and very soon the surrounding cellular tissue are hyperæmic, swollen and infiltrated with cells. The external coat becomes homogeneous and gelatinous. The process terminates either in connective-tissue or pus formation. In purulent infiltration of the external coat the intima is not affected; but should the middle coat become involved pus may open into the blood current and an aneurism is liable to be formed.

Etiology.—Periarteritis occurs from wounds, extension of inflammation from adjacent parts, or during the course of pyæmia.[1] Periarteritis is the first step in the formation of those miliary aneurisms occurring in the cerebral vessels, and which are always found preceding cerebral hemorrhages.

Fatty degeneration, apart from atheroma, is rare. It occurs chiefly in the aorta. In the internal coat the fat granules occur in flat layers, and in the middle coat they are found between the fibres, and when very abundant the muscular elements cannot be distinguished. Sometimes the endothelium alone is involved, and it may desquamate, laying bare the tunica intima. This is said to accompany erysipelas and relapsing fever.

Calcification of the arteries, independent of atheroma, is even rarer than fatty degeneration.

Amyloid degeneration occurs in the small arteries, especially in the renal glomeruli, but also in those of the spleen, liver, intestines and lymph glands. Its causes, gross and microscopical appearances, tests, etc., are fully discussed under chronic Bright's disease.

Cancer only attacks the adventitia.

Tuberculous granules often stud the external coat of the small arteries.

Syphilitic disease of the arteries chiefly attacks the cerebral vessels. Great thickening and nodose swellings are due to gummatous material infiltrating the outer coats. The walls are opaque and the lumen is considerably diminished. Later, cellular growths occur in the internal coat. Thrombosis and cerebral softening are often the result. This neoplastic formation has been called *arterioma*. When such a condition is suspected, mercury and iodide of potash are to be given.

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[1] Kussmaul and Maier describe a periarteritis nodosa which is usually fatal, and Gull and Sutton have called the hyaline fibroid appearance of the external coat of the arterioles *arterio-capillary fibrosis.*

General dilatation of arteries may be due to atony or paralysis of their muscular coat, or to atheroma or degeneration of their walls. The aorta and the pulmonary arteries are those most frequently involved. Sometimes the aorta and its branches are the seat of congenital uniform stenosis. This occurs in females chiefly, and is associated with other malformations. *The symptoms* are a small pulse, frequent palpitation, cold extremities, tendency to syncope, and menorrhagia. Gastric ulcers are common. The aorta may be contracted and nearly obliterated at its junction with the ductus arteriosus.

GENERAL ARTERIAL FIBROSIS.

Dr. Bright, and most pathologists since his time, noticed that the granular contracted kidney—the "small red kidney" of the English writers—was usually associated with morbid changes in other organs, and it was generally held that, under these circumstances, the kidney was the organ primarily affected, and that the other changes were the result of the cachexia produced thereby.

In 1872 Sir William W. Gull and William Henry Sutton[1] denied the correctness of this latter opinion, and claimed that all the morbid changes, those found in the kidney, as well as those of the other organs, were equally dependent upon a fibroid degeneration of the walls of the smaller arterioles and capillaries. To this degeneration they gave the name *arterio-capillary fibrosis*, and while admitting that it commonly began in the kidneys, they claimed that there was evidence of its primary appearance in other organs, and also of its occasional localization elsewhere, to the entire exclusion of the kidney. The morbid changes peculiar to this condition have long been recognized by pathologists, as have also the corresponding clinical facts, but the connection between them was not understood. It is this connection, this grouping together, with a more detailed knowledge of the minute changes, that constitutes the present theory. Although the description of the pathological changes, as given by Gull and Sutton, has been shown to be incorrect, their comprehensive explanation of a general arterial disease, as distinct from any renal disease, is fully accepted.

Dr. George Johnson and others have shown that an hypertrophy of the arterial muscular coats may produce similar symptoms as the fibroid change, and an acceptable explanation has been given by Conheim of the relations which such muscular hypertrophy bears to renal disease. While these arterial changes are thus most frequently associated with some form of nephritis, they may be primary, and not involve the kidney in any marked degree.

Morbid Anatomy.—Two distinct pathological processes must be recognized as producing those changes and symptoms which are characteristic of high arterial tension. In the first the changes consist in a general increase in the amount of fibroid tissue throughout the body, especially marked in the excretory organs, together with hypertrophy of the left and dilatation of the right ventricle. The increase of fibrous tissue usually begins in the outer coat of the smallest arteries, and spreads thence through the connective-tissue stroma of the organ involved. The inner coat is also sometimes much swollen, granular, and thickened. In the capillaries the new tissue is granular and without any fibroid appearance. According to Gull and Sutton the muscular coat of the artery is atrophied. The result of these changes is to diminish the calibre and destroy the elasticity of the vessels. This alteration in the vessels is the primary and essential morbid process; by its reaction upon the heart, and by its spreading to the adjoining tissues, it produces the following secondary changes. By the formation and retraction of new connective-tissue, especially in the kidneys, gastro-intestinal tract, and skin, these organs diminish in size and lose more or less of their granular epithelial elements.

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[1] Medico-Chirurg. Transactions, 1872.

The lungs are firm, with prominent bronchi, and often show well-marked vesicular emphysema. There is atheroma of the aorta and of the cardiac valves, opacity of the arachnoid, and increase in the amount of the subarachnoid liquid.

Although the renal changes are not necessarily associated conditions, they are so frequently developed, late or soon, as to be considered a complication if not part of the disease.

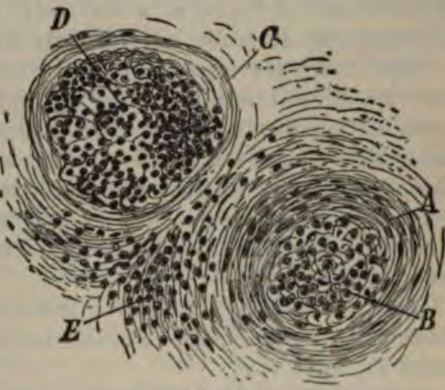

FIG. 113.

Section from the Cortex of a Kidney in general arterial fibrosis, showing two Glomeruli.

A. Capsule of a Malpighian body thickened with laminated connective issue.
B. Vascular tuft degenerated into a fibrous nodule.
C. Capsule of another Malpighian body, with fibrous thickening, smaller in amount than at A.
D. Vascular tuft, adherent to capsule, with round cell infiltration.
E. Nucleated fibrous tissue surrounding the atrophied glomeruli. × 300.

The kidneys are small, red, and granular, with adherent capsules, and with small cysts scattered through them. This condition of the kidney must be distinguished from the *mixed* or *yellow granular* kidney, which, according to this theory, is either a large white kidney that has shrunk and become granular, or else is the consequence of an attack of acute parenchymatous nephritis supervening upon the chronic interstitial change. The microscopical appearance of the kidney is such as will be described in interstitial nephritis. There is increase of intertubular connective-tissue, especially around the Malpighian bodies, and in and around the walls of the minute arteries.

The hypertrophy of the heart, which is found so constantly in connection with these changes, is readily explained. The heart is called upon for greater effort, in order to overcome the obstruction to the blood-current created by the change in the arterioles and capillaries, and, as in the case of other muscles, it increases in size and strength to meet the additional demands. The increase of arterial pressure is also felt within the heart, and ultimately produces the other changes seen upon the valves—changes which are found at the points subjected to great pressure.

This explanation is in harmony with the following facts observed by Gull and Sutton: hypertrophy of the left ventricle existed in all cases in which the vessels were generally thickened by the hyalin-fibroid change, and its degree varied directly with the degree or the extent of the change. They argue that it is due not to renal disease, but to the morbid changes in the vessels, because (1) it is often absent in cases of large white kidney, in lardaceous disease, and in scrofulous pyelitis, with almost complete destruction of the organ; (2) whenever hypertrophy of the heart coexisted with large white kidney, the hyalin-fibroid change was also present; and (3) hypertrophy is found at a very early period of the kidney affection when the excretory function is not greatly altered.

The other pathological processes which produce a similar clinical picture of high arterial tension and hypertrophied heart associated with renal disease, are, according to Dr. George Johnson, found in an hypertrophy of the muscular coats of the smaller arteries and arterioles. It seems well established that such an hypertrophy occurs, but the explanation of its cause offered by Dr. Johnson, that the hypertrophy was intended to decrease the arterial calibre, and thus shut out irritating blood from the organs, does not seem tenable.

This muscular hypertrophy is found especially in connection with parenchymatous forms of renal disease, and Conheim's explanation appears the most probable.

He claims that functional activity in the kidney varies directly with the arterial tension and blood-flow. When part of the renal parenchyma becomes inactive through disease, an increased arterial tension is a necessary condition for the extra activity demanded of the unaffected portions, in order that the sum total of the renal secretion may not be diminished. To produce this, an increase of cardiac activity is necessary, which results in cardiac hypertrophy. In order, then, that the augmentation of cardiac activity may not disturb the circulation in organs requiring no change in their vascular conditions, but may be concentrated upon the kidney, a general contraction of all the arteries is necessary—a contraction which shall vary with the varied requirements of the several parts. Such contraction can be accomplished only through the muscular coats of the vessels under the control of the sympathetic system. Its persistence results in hypertrophy of the muscular coats.

Although these changes are pathologically and etiologically entirely distinct from an arterial fibrosis, clinically they can not always be distinguished, and we are able to recognize only high arterial tension and its results.

Etiology.—The exciting cause of the fibroid change is usually some form of blood-poisoning, either temporary or chronic, such as gout, alcoholism, pregnancy, scarlet fever, lead poisoning, and certain forms of dyspepsia, mal-assimilation and functional disorders of the liver, which act by producing first a functional and then an organic increase of the arterial tension.

Aside from these direct causes, however, we recognize a fibroid diathesis which influences all inflammatory processes. Under its influence causes which would otherwise be inoperative produce extensive fibroid degeneration. The exciting cause of the muscular hypertrophy would be found, according to the theory given, in defective renal function—a defect which may be relative or absolute, inherited or acquired, and be present with or without renal disease.

Symptoms.—The symptoms vary with the organ chiefly affected and the period of the disease. The first in order of time, and the one upon which Mahomed places the most reliance as a means of diagnosis, is the increase of arterial tension, recognized by the pulse, or, better, by the sphygmograph. During this stage, if the pressure has increased rapidly, dropsy and albuminuria may be present, but ordinarily these two symptoms denote accompanying epithelial change in the kidney, or an exacerbation in the progress of the disease. Albuminuria is not itself a symptom of arterial fibrosis; on the contrary, the affection may run its course without the appearance of this symptom. In such cases the vascular changes have involved other organs, and have left the kidney unchanged or but slightly affected; such patients die with symptoms of pulmonary or gastro-intestinal troubles, or of cerebral hemorrhage or aneurism.

The condition begins as a diathesis in early life, gaining ground every year, and betraying itself by the pulse, pulmonary emphysema, or dyspepsia, and if at any time a serious exacerbation occurs, death may be caused with symptoms referable more directly to the heart or kidneys.

Diagnosis.—The diagnosis, therefore, is to be made mainly by a consideration of the character of the pulse, and it is claimed that heretofore, in the majority of cases, the disease has passed unrecognized,[1] the diagnosis being made only when the kidneys were sufficiently involved to give rise to albuminuria. The important point, therefore, is to recognize the condition of high arterial tension. The sphygmograph alone can always do this with certainty, but careful examination of the heart and pulse will usually suffice. The pulse of high pressure has been variously described as hard, cord-like, persistent, long, or slow. The most constant and characteristic quality is that designated as persistent or slow (not infrequent). The artery feels full under the finger during diastole as well as systole of the heart, and its systolic expansion is prolonged,—the so-called *pulsus tardus*, shown on the sphygmograph by a prolongation of the elevation of the trace. The heart signs of high arterial pressure are a long or reduplicated first sound heard over the inter-ventricular septum, and an accentuated second sound.

The following conclusions, taken from Gull and Sutton's first paper and Mahomed's last upon the subject, present the points in convenient form.

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[1] Mahomed says: "How often patients are allowed to die—nay, more, even killed—when their hearts are failing from the terrible arterial pressure they can no longer overcome. Their flagging, over-taxed ventricles dilate; the wretched, feeble, laboring pulse is thought to mean weakness which requires stimulation, its persistence (indicating over-distention) is passed unnoticed, and the struggling heart, failing at last in its work, stops, and the patient dies for want of a lancet or purge."

There is a disease characterized by hyalin-fibroid formation in the arterioles and capillaries, attended with atrophy of the adjacent tissues. This morbid change in the vessels is the primary and essential condition of the morbid state called arterio-capillary fibrosis with contracted kidney. The kidneys, however, may be little if at all affected, while the morbid change is far advanced in other organs. The blood condition which produces the high arterial pressure is the primary condition, and is not secondary to deficient renal excretion as heretofore held. The cardio-vascular changes, when found alone, may be taken as evidence of the existence of the disease. The condition of high pressure is almost constantly present in old age, and in one form or another brings about a large proportion of the deaths of those over fifty years. The existence of high arterial tension in the pulse of young persons indicates a diathesis, and is of grave importance. The same condition being of frequent occurrence after the age of fifty is not of such great importance, unless present in an excessive degree. It then produces serious symptoms and calls for active treatment.

Treatment.—Whether inherited or acquired, the fibroid diathesis is most intimately associated with lithæmia, and all treatment is primarily directed to this condition. Evidences in early life of faulty metabolism, of defective oxidation or deficient elimination of waste products, should lead to the adoption of a carefully regulated diet and the persistent use of those drugs which stimulate these processes. When the condition is once developed and the evidences of high arterial tension are present, even greater care must be exercised in the diet. From the earliest indications of disturbance in the nutritive processes, the persistent use of alkaline waters will be desirable. Mercury and Warburg's tincture are the most decided stimulants to the metabolic changes; and later, when fibrous tissue is being formed, minute doses of bichloride of mercury may be given continuously.

PHLEBITIS.

Phlebitis may be acute or chronic.

Morbid Anatomy.—In acute phlebitis the adventitia may be first involved and the inflammation extend inward, a clot forming in the calibre of the vein; or the inflammation may commence within, in connection with surrounding inflammation, and extend outward. If there is extensive connective-tissue infiltration around the vein, adhesive obliteration of the vein results; should the clot soften and disintegrate, pus formations result. The presence of a clot may be regarded as an essential accompaniment of all forms of phlebitis except the adhesive or chronic.

In *chronic phlebitis* the external coats of the veins are very much thickened, while the intima may be normal. The connective-tissue around the vein is greatly increased, there is hypertrophy of its muscular tissue, and the vasa vasorum are very much dilated. In rare cases thickening, fatty degeneration, and calcification ("atheroma") of the innermost venous coats are found. In these cases the outer layers will almost invariably exhibit "sclerotic" changes. This has been called "*chronic endophlebitis.*"

Etiology.—The commonest cause of phlebitis is the formation of a thrombus. Periphlebitis may be induced by wounds, ulcers, abscesses, chronic visceral disease, phlegmonous erysipelas, separation of the placenta, osteomyelitis, amputation, ligation of veins, pyæmia and septicæmia, cellulitis from any cause, and, according to some, by varicosity or permanent dilatation.

Symptoms.—If the vein is within reach of observation, it will be found hard, swollen, and tender; prominences occur at the sites of the valves, pains dart along its course, and the limb may become stiff. When superficial, the veins can be felt, and the skin over them is livid red. When deep main trunks are involved, the limb is swollen and the skin pale, tense, and shining over it.[1] Abscesses in the course of the vessel, which may or may not communicate with its interior, are of common occurrence. Should the tissues become œdematous and should constitutional hectic or pyæmic symptoms supervene, suppurative phlebitis (peri-phlebitis) may be suspected.

Gouty phlebitis occurs in those with hereditary gouty tendencies; the skin over a vein becomes dusky or livid red, the vein is hard, and the limb is somewhat œdematous. All the symptoms may suddenly disappear, quickly to reappear in some distant part (metastatic phlebitis). Varicose veins in the gouty are especially liable to these manifestations, although loss of tone and local erythema are more to be blamed than gout for these venous inflammations.

Differential Diagnosis.—Phlebitis is distinguished from *lymphangitis* by the fact that in the latter the glands are tender and enlarged from the outset; and bright red streaks are very numerous.

In *erysipelas*, redness is in the form of a general blush;—in phlebitis there is only a dusky red localized streak.

Prognosis.—This is bad only in the suppurative variety.

Treatment.—Absolute rest, splints to confine and render the affected limb immovable, and hot fomentations over the parts are the first indications. Abscesses should be opened early, and when œdema occurs the parts must be bandaged.

VARIX.

Varix or dilatation of the veins occurs most in obstructive diseases of the right heart. In a few cases the veins become dilated and varicose without any obstruction, the cause of the dilatation under such circumstances being very obscure.

Morbid Anatomy.—When veins dilate they elongate; the dilatation is most marked immediately above the valves and the affected vein assumes an irregular outline. The walls are thickened from hypertrophy of their middle coat at some points, and dilated at others. Dilatation may take place in the largest, the smallest, or in the medium sized veins; calcareous plates not infrequently form in their walls, and phlebolites and venous calculi often develop in the pouch-like protrusions in the veins, where the circulation is slowed. Local or general obstruction and a varicose condition of the veins serve primarily as important aids in diagnosis and rarely require medical treatment. They are mainly surgical disorders.

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[1] See phlegmasia alba dolens in works on Obstetrics.

THROMBOSIS.

Thrombosis is coagulation of the blood in the heart, or blood-vessels, during life. The colt or coagulum is called a *thrombus*, and is most commonly met with in the veins. Parietal thrombi are those clinging to the wall of a vessel and not completely obstructing the flow. Occluding thrombi are those absolutely obstructing the flow.

Morbid Anatomy.—In rapidly formed thrombi a considerable number of red blood discs are entrapped and the color is first dark red ; in such clots the fibrin at once completely fills the vessel, and the thrombosis is uniform or non-laminated. These are the usual characteristics of obliterating thrombi. In slowly formed thrombi fewer red blood discs are entangled, hence the color is lighter, sometimes, indeed, the clot is absolutely colorless. The structure is laminated or stratified, and the mass adhering to the wall of the vessel does not wholly obstruct the current, or at least in its early stages. These *primitive* thrombi usually extend along the vessel to a branch whose blood current is sufficiently strong to arrest their progress. The projecting conical end of the coagulum becoming softened, small pieces may be detached and thus enter the circulation.

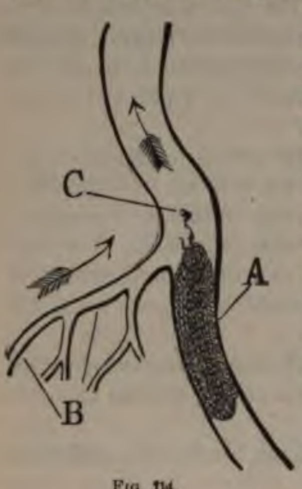

FIG. 114.

Diagram showing the manner of detachment of small portions of a venous thrombus A, by the blood current of a small venous branch, B. C, Detached portions of coagulum.

A thrombus may organize or undergo shrinking, softening, or suppuration.

Organization occurs oftenest in uniform thrombi situated in arteries. The leucocytes in the clot or those from the vasa vasorum develop new connective-tissue, and vessels permeate the new structure in whose meshes lies the débris of the clot. Progressive dilatation of the new vessels ultimately renders the original channel pervious:—"*canalization of the thrombus.*"[1]

Instead of organization a thrombus may *soften*. Stratified thrombi usually soften. Molecular disintegration commences at the centre of the clot, which will be found filled with a purulent-looking milky or pulpy material, containing albuminous granules, molecular fat, granular detritus, and changed red and white corpuscles. This is not suppuration of the thrombus, nor should it be called puriform softening. Large cardiac thrombi suffering these changes resemble cysts. Those thrombi that break down into granular matter containing *bacteria* and *pus cells*, are specific or infectious thrombi (*vide* Pyæmia). A non-infectious thrombus, after softening, may be wholly absorbed ; or, as central softening occurs, fibrin is deposited upon the periphery. Pus may enter such a thrombus from without.

Suppuration is occasionally seen in the thrombi of veins surrounded by, or leading from, inflamed parts ; a multiplication of leucocytes takes place

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rnil and Ranvier assert that there is merely an outgrowth of vascular granulations from the tunics at penetrate the thrombus ;—and that the latter gradually disappears without taking part in the *of the* reticulated tissue which occupies its place ("obliterating endarteritis").

in the thrombus either by proliferation or immigration, and the whole softens down into a purulent fluid. In these cases the wall of the vein itself is always inflamed. These softened and broken-down thrombi are a common cause of embolism. When the middle coat of the vein is involved and intensely inflamed, true suppuration of the coats may occur, and thus thrombosis may be a cause of abscess of the external coats of a vein. It is important to distinguish between thrombi and post-mortem coagula :—the latter are soft, divisible into two layers—a colored and an uncolored—are never laminated, their texture is looser, they never entirely fill the vessel, and they do not adhere to its wall. Heart clots that form during the death agony are, in color and consistence, midway between the two just mentioned. They are entangled with the columnæ carneæ and chordæ tendineæ, but can be separated with a little care.

Etiology.—Any abnormality of the vessels, but especially of the tunica intima, will induce the formation of a thrombus (atheroma, phlebitis, endarteritis, etc., etc.). Any neoplasm in a vessel may cause it. Wounds, blows, ligation, dilatation of the vessels or of the heart, and anything that will diminish heart power, or induce *slowing of the blood current*, will induce thrombosis. Hence we find it occurring in phthisis, cancer, old age, etc., etc. The veins of the pelvis and lower limbs, and in children the cerebral sinuses are the favorite seats of these "*marantic*" thrombi.

Compression thrombosis results from slowing of the current from mechanical causes outside the wall. In the heart thrombosis may be caused by endocarditis. Lymphatic thrombosis has chiefly been observed in the puerperal condition. In leucocythæmia the capillary circulation being interfered with from the vast number of white corpuscles, clots readily form in the veins. Finally, venous thrombi are especially liable to form in the pockets of the valves.

Symptoms.—The symptoms depend upon the extent of the obstruction to the circulation and the size and situation of the vessel :—for instance, when the femoral vein is plugged, phlegmasia alba dolens results. Thrombosis of the cerebral vessels gives rise to special cerebral manifestations.[1] Thrombosis of the portal vein is followed by the grave symptoms of pylephlebitis. Moist gangrene, ascites, hydrothorax, œdema and cyanosis of the face and neck, hemorrhage from stomach, intestine or kidney—each may be a consequence of the plugging of the main vein issuing from the part. The special danger of venous thrombosis is the possible detachment of a portion of the thrombus, its transportation by the circulation to the heart, and its arrest there or in one of the branches of the pulmonary artery (embolism). The result of arterial thrombi is anæmia of the part supplied, necrosis, or hemorrhagic infarction.

EMBOLISM.

An embolus is any solid body other than the corpuscles floating in the blood current. Embolism is the occluding of a vessel by an embolus. Thus it is seen that arterioles and capillaries are the usual seats of embolism, since in these vessels the current is toward ever-diminishing branches.

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[1] See Brain : Art. *Embolism.*

In general an embolus is part or all of a dislodged thrombus; for example: a clot in the femoral vein (milk-leg) crumbles; particles are swept into the ascending cava, then through the right heart into the pulmonary artery, and some of the latter's branches having a calibre smaller than the diameter of the particles they will be plugged.

Morbid Anatomy.—When small arteries are plugged the anastomoses may prevent any visible lesion from occurring. When a vessel of any size is plugged, the first result is anæmia of the district supplied by the branches of the blocked vessel. Then there is backward pressure and regurgitation of blood from the veins, through the capillaries into the arterioles, whose vitality is impaired because of this *venous substitution*. Exudation and ultimate necrosis of vascular walls are followed by hemorrhage, and the blood coagulates forming a *hemorrhagic* infarction. When an embolus causes anæmia and necrosis without hemorrhage, it produces what is called a *white* or *anæmic* infarction. The primary and essential change in a white infarction is coagulation-necrosis. The shape of an infarct is conical because of the tree-like branching of the arteries and capillaries beyond the site of the embolus; and it is usually situated with its base toward the surface and its apex toward the centre of the organ.[1] In non-infective infarctions the mass becomes decolorized, changing from dark red to dirty yellow, and then to white. At the same time it shrinks and finally may leave a depressed fibrous patch or cicatrix. In larger masses molecular disintegration and softening first occur and a pulpy granular puriform fluid forms, which becomes enclosed in a fibrous capsule, and finally becomes cheesy or calcareous. In both cases there is a circumscribing zone of congested vessels.[2]

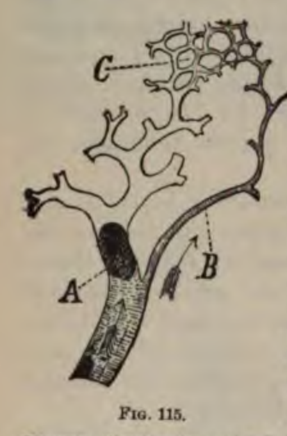

FIG. 115.

Diagram showing the establishment of circulation by anastomotic vessels after an Embolism.

A, Embolus plugging small Artery.
B, Anastomotic branch supplying blood to the area C.

When a vessel is plugged, wholly or partly, and the vessel is a terminal artery, hemorrhage does not necessarily occur, and there occurs a white or anæmic infarction, which is to be distinguished from a decolorized hemorrhagic infarction by microscopical examination. Some emboli are always followed by necrosis, others by hemorrhage. In the brain anæmic softening without hemorrhage is common. Necrosis may rapidly ensue after embolism (gangrene), or it may come on slowly as a withering or softening. The vessel wall at the site of the embolus undergoes changes similar to those described as resulting from thrombi. If an embolus does not completely fill a vessel a secondary thrombus forms, by deposition of fibrin, and this extends till a strong current of blood arrests its progress. Emboli coming from venous thrombi usually induce pulmonary infarction; emboli from the left heart, arterial aneurisms, arterial neoplasms, etc., produce infarctions in spleen, kidneys and brain, as a rule.

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[1] Cohnheim states that in order to produce hemorrhagic infarction the artery must be a terminal artery, *i. e.*, giving off no anastomotic branches before its final capillary distribution, and the veins must *not* have valves, and that these conditions are met with in the spleen, kidney, brain, certain branches of the pulmonary artery, and the central artery of the retina. Litten has opposed these views, and states that in genuine terminal arteries hemorrhagic infarction does not occur, and that infarction may take place *after ligature of the vein*. Hence venous reflux cannot be its cause. From his experiments it would seem that congestion and infarction following embolism are due to afflux of arterial blood into the territory from collateral channels; his views more nearly correspond to Virchow's original theory. —*Zeitschrift für Klinische Medicin*, vol. i.

[2] The changes that occur in infective or specific emboli are described under pyæmia. Cornil and Ranvier regard the decolorization which marks the *white infarct* as the result of fatty degeneration of the parenchymatous cell elements of the organ involved. The connective-tissue about them is infiltrated with white blood discs. But Litten regards white infarctions as due to *coagulation-necrosis* of the protoplasm of the cells, having a remarkable tendency to calcification.

When emboli produce death of part of an organ without true gangrene, Virchow gives the name *necrobiosis*. An embolic abscess is the same as a pyæmic abscess. The influence of embolism in the production of aneurism is to-day recognized, even when the particle that plugs the vessel is not a sharp calcareous or atheromatous mass.

THORACIC ANEURISM.

An *aneurism* is a more or less abrupt dilatation of the calibre of an artery; the tumor thus formed must communicate with the channel of the vessel. *Thoracic* aneurism includes all those tumors which arise from the aorta and its branches within the thorax, or from the pulmonary artery.

Morbid Anatomy.—The convexity of the ascending portion of the arch of the aorta is the most frequent seat of the aneurismal development, next the transverse portion of the arch; next the descending portion of the arch, and least frequent of all the descending aorta. Aneurisms of that portion of the aorta which is embraced by the pericardium are of small size and are apt to pass unrecognized.[1] The junction of the ascending and transverse portions of the arch at the *sinus magnum* is a favorite seat of aneurism, it being nearly at right angles to the blood-stream from the heart.[2]

The only logical clinical classification of aneurisms is based on their shape.

The whole surface of the artery may be dilated, and the aneurismal tumor be cylindrical, fusiform or globular in shape.

There may be a lateral bulging or sacculation of a portion of the circumference of the artery:—a *sacculated aneurism*. In both of these classes the arterial coats may be all intact, or any one or two of them may be absent or diseased.

When the walls of an aneurism are made up of the surrounding tissue, it is called a consecutive *diffuse* aneurism; and when blood finds its way between the coats of an artery, it is called a *dissecting* aneurism.

The post-mortem appearances of aneurism will vary with its location, size and variety. In some cases nothing abnormal will be found except an unruptured aneurismal tumor; in others the tumor will be found ruptured, the pericardium filled with blood, or extravasated blood will be found either in the bronchi, trachea, stomach, or pleural cavity, or an external rupture may have been the immediate cause of death. Aneurisms arising from one of the sinuses of Valsalva, within the range of the valves, rarely attain a size larger than that of a small billiard-ball. They are sacculated and not infrequently pedunculated, communicating with the aorta by a small orifice. They further exhibit a remarkable tendency to *descend* in the progress of growth, involving in their course the heart or the root of the pulmonary artery. By their position they are sheltered from direct influx from the ventricle, whilst they are exposed to the maximum force of reflux from the aorta. When, however, the orifice is partially or entirely above the level of the valves, the main pressure sustained by the sac is that during influx from the ventricle; hence the direction of growth is upward.

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[1] Of 708 cases of Sibson's (Medical Anatomy), 87 were within the pericardium, *i. e.*, about 12 per cent.

[2] Anatomically, aneurisms are divided into *true* and *false*. True aneurisms are those in which all the coats of the artery are found in the walls of the aneurismal sac. False aneurisms are those in which a rupture of one or more coats of the artery has occurred.

Aneurisms near the sinus magnum produce erosion of the ribs and their cartilages, the sternum and the right clavicle :—sections of the bones show the lesions of osteitis. The adjacent muscular and connective-tissue is extensively infiltrated. The descending cava and the left innominate vein may be so compressed as to have their channel completely closed. The left recurrent laryngeal, the left sympathetic, or the trunk of the vagus may be compressed, atrophied, or entirely destroyed. The thoracic duct may be compressed and ruptured into. Should aneurisms about the arch enlarge backward, the trachea, œsophagus, and right lung will suffer from the pressure.

In aneurism of the descending arch, or thoracic aorta, the spinal extremity of the ribs and the bodies of the vertebræ in the dorsal region may be destroyed, and the left bronchus may be obliterated, causing consolidation of the entire lung. The dorsal spinal nerves and sympathetic trunk may be destroyed by pressure. All the secondary changes in thoracic aneurism are "pressure effects," and they are never alike in any two cases.

The *aneurismal sac* also varies greatly in the appearance it presents at the autopsy. All the tunics of the artery may be preserved ; but in large aneurisms while the external and internal coats can be traced all over the tumor, the middle coat ceases abruptly where the sac opens into the artery. When an aneurism begins with rupture or disease of the inner coat of the artery, a lining membrane of new formation meets and coalesces with the *intima*, so that the appearance is the same as if no rupture or change had occurred. The aneurismal walls may undergo fatty or calcareous degeneration. The contents vary with the size and shape of the sac and with the rapidity with which it has formed. The sac may be nearly filled with concentric layers of firm laminated fibrin containing small calcareous plates ; or it may be partially filled with looser layers of fibrin inclosing recent coagula. Sometimes fresh coagula and fluid blood are alone found. Those laminæ of fibrin nearest the aneurismal wall are the firmest.

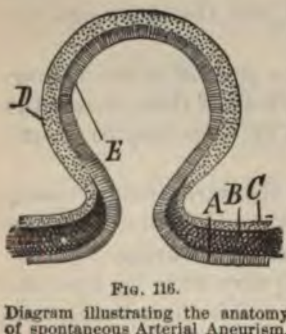

FIG. 116.

Diagram illustrating the anatomy of spontaneous Arterial Aneurism.

A. Internal coat of Artery; B. Middle coat of same ; C. External coat.

D and E. External and internal coats of the aneurismal tumor showing the absence of the middle arterial coat.

Etiology.—The chief predisposing cause of aneurism is disease of the arterial walls, the most common of which are chronic endarteritis and atheroma. Age and occupation may also be regarded as predisposing causes, the period between forty and fifty being the favorite period of its development. It is a question whether aneurisms occurring in middle life are the result of senile changes or violent physical exertion. Atheroma and calcareous degenerations are commonest after sixty; hence muscular effort probably has much to do in developing the more frequent aneurisms in those who are younger, although it is doubtless aided by commencing degeneration of the arterial wall. Mechanics, porters, soldiers, and those liable to sudden and violent physical exertion are frequent subjects of aneurism; the irregularity and violence of the action is to be considered, rather than its severity. Habits of life, intemperance in eating and drinking, chronic alcoholismus and tight fitting garments (uniforms) predispose to aneurismal developments. The majority of these who develop aneurism before forty-five will give a syphilitic history; hence syphilis must be ranked as a predisposing cause. Chronic Bright's, rheumatism, gout, lead and mercurial poisoning are included in the predisposing causes to arterial diseases, and consequently to aneurism. In aortic insufficiency the hypertrophied left ventricle throws a larger column of blood with abnormal force against aortic walls; chronic aortitis results and an aneurism may follow.

The *exciting* causes are blows, falls from heights, wounds, excess or prolonged venereal excitement, and sudden violent strains, exerted on a degenerated artery.

Symptoms.—The early *rational* symptoms of thoracic aneurism vary with the site of the tumor. If the aneurism is near the sinuses of Valsalva, it will give rise to no symptoms until rupture discloses its existence. If a second murmur is heard over the pulmonary artery it may be caused by displacement of the valve, or diminution in the calibre of the pulmonary artery from pressure of an aneurismal tumor, and if it is accompanied by venous stasis and congestion of the upper half of the body, an aneurism may be suspected. With all aneurisms within the pericardium there will be some hypertrophy of the left ventricle. The development of an aneurism near the sinus magnum is usually accompanied by very positive symptoms. The patient often states that after some violent effort, some blow, or during an excess of some kind, "he felt something suddenly give way," and then followed a "boring" pain near the sternum with dyspnœa, palpitation, and perhaps hæmoptysis.

As a rule there are no subjective signs of thoracic aneurism until the tumor presses on the adjacent parts. By the direction of the pressure the seat of the tumor may be determined. Aneurism of the ascending arch usually presses forward, upward, and to the right;—of the transverse arch, backward and upward; and of the descending portion of the arch, backward and to the left. In whatever direction an aneurism presses, *pain* is its first symptom. The pressure may be exerted upon (1) *the nerves;* (2) *the blood-vessels;* (3) *the trachea, œsophagus,* the *large bronchi, the lung-tissue,* the *thoracic duct,* and, indirectly on the *heart.*

The pain when present is constant. It is increased by acceleration of the circulation, and is localized in the region of the tumor; usually it is associated with a sense of constriction. The *pressure pain* may be neuralgic, paroxysmal and wandering. It radiates to the neck and shoulder and may shoot down either arm. If the intercostal nerves are pressed on, there will be attacks of excruciating intercostal neuralgia. If erosion of vertebræ, sternum, or ribs occurs, there is a peculiar, constant "boring" pain. When one or both vagi or recurrent laryngeal nerves are pressed on, spasms and partial or complete paralysis of the laryngeal muscles cause dyspnœa and voice-changes; the voice becoming husky. Sometimes there is complete aphonia. Violent paroxysms of dyspnœa are liable to occur, attended by a congested, anxious countenance, and violent respiration followed by exhaustion. Cases are recorded where vomiting and pyrosis resulted from pressure on the pneumogastric. Pressure on the pulmonary plexus gives rise to a harsh metallic "brassy" cough. Pressure on the vagus may be followed by congestion of the lungs, œdema and gangrene. An inequality of the pupils may come from irritation or pressure on the cervical sympathetic: irritation causes dilatation of the pupil; and pressure (when annulling the function) causes its contraction on the affected side. Disordered vision may thus become a symptom of thoracic aneurism.

When *blood-vessels* are compressed only the main trunks of *one* side are involved, hence a delayed, even a suppressed radial pulse will be found only on that side. In a few cases I have found no pulsation in either carotid or subclavian on the affected side. Then cerebral anæmia and signs of impaired nutrition in the limb on that side were present. The effect of impeded venous return may lead to a diagnosis of the seat of an aneurism. When an aneurism near the sinus magnum enlarges forward, the upper half of the body shows congestion and œdema; there is headache, drowsiness and other cerebral symptoms, and the eyeballs protrude. Aneurism of the innominata or of the right common carotid in the thorax, presses on the external jugular, and hence the right side of the head and neck is turgid. Such a condition on the left means aneurism of the left common carotid.

When *tracheal* symptoms are urgent, they point to aneurism of the transverse portion of the arch enlarging backward. The flattening of the trachea induces difficult breathing, then follows a stridulous cough (with *no* expectoration), having a metallic ring, like a "nervous cough." Such compression may result from an accumulation of mucus which cannot be expectorated; hence, dyspnœa arises. The pressure may even produce gangrenous patches, which lead to rupture and fatal hemorrhage. It is readily seen why congestion of the lungs and pneumonia sometimes follow compression of the trachea. The signs of pressure on a *large bronchus* are, principally, a metallic cough, with tenacious mucous sputa, at times blood-streaked, and, *possibly,* evidences of pneumonia and gangrene. Pleurisy may be excited by a tumor's pressure, and it is always an important sign taken in connection with signs of pressure upon the trachea and bronchus.

Dysphagia may be induced, but the œsophagus is rarely ruptured into by an aneurism. Dyspepsia, reflex in origin, may be a symptom of thoracic aneurism. The lower third of the œsophagus is said to be widely dilated in some cases of this kind. Enlargement of the lymphatics below the sac results from pressure of an aneurism on the thoracic duct. Symptoms of mal-assimilation, wasting and inanition would also be present in such cases.

All these symptoms are never present together in any one case, but when three or four of the prominent ones exist, they are strong evidence of thoracic aneurism.

Physical Signs.—*Inspection.*—If the aneurism press on the cava descendens, the face, neck and upper extremity will be swollen, livid, or œdematous, the veins being turgid and varicose. Sometimes there is a thick, fleshy collar around the lower part of the neck, due to capillary turgescence. Bulging is seen at some spot on the chest, probably along the course of the aorta, and this may be as large as a cocoanut, or, again, may be perceptible only after careful inspection. Non-existence of a tumor does *not*, however, disprove the existence of an aneurism ; aneurismal tumors deeply seated will not produce bulging. When the visible tumor is large it is generally conical. The skin over it is smooth, tense and shining. Inspection may reveal pulsation in it, which is synchronous with the cardiac systole, and when this bulging occurs on the anterior surface of the chest there seem to be two beats within the thorax at the same time. Pulsations are, at times, only detected by bringing the eye to a level with, and looking across the chest. Aneurisms of the ascending arch usually enlarge first to the right of the sternum near the second costal cartilage, but if it is very large it may extend into both the mammary and infraclavicular region. Aneurisms of the transverse arch protrude above the sternum, those of the descending arch to its left. In the latter case a visible tumor is uncommon. Aneurisms of the descending aorta enlarge to the left, rarely to the right, of the spine. They may sometimes give rise to violent pulsations near the heart and simulate extensive cardiac hypertrophy.

Palpation discovers more accurately the size and the condition of the walls of the aneurism. The pulsation imparted to the hand is like that of a blow from the centre outward in all directions, dilating or *expansile ;* there may be a diastolic pulsation as well as systolic. The impulse is sometimes perceptible only when one hand is pressed over the sternum and the other over the interscapular space. When the transverse arch is involved the aneurismal thrill may be communicated to the hand by pressing the fingers down behind the sternum. Palpation should be employed to detect lung changes, fremitus, expansion, etc., etc.; it is noteworthy that consolidation of lung substance induced by thoracic aneurism is characterized by *absence* of vocal fremitus.

Percussion elicits circumscribed dulness at some point along the line of the aorta, corresponding to the seat and size of the aneurism. A resistance, peculiar to aneurism, and increased by the force of the percussion-blow, will be noticed over all large aneurisms. Consolidation of adjacent lung-tissue may increase the area of dulness.

Auscultation.—The heart sounds accompanied by " murmurs " peculiar to aneurism may at times be audible over the seat of the tumor, or both heart sounds may be replaced by murmurs, the character of which varies. They may be sawing, rasping, or grating. A diastolic murmur is rarer than a systolic, and is usually softer. With aneurisms near the sinus, the murmur is booming or splashing, and is accompanied by a thrill not transmitted in any direction. When a large bronchus is compressed, the respiratory murmur is weak or suppressed on one side and exaggerated on the other. There is loss of vocal resonance over the aneurism and over the side on which the bronchus is compressed.

Differential Diagnosis.—It is always of the first importance to determine at what point in the course of the aorta an aneurism is developed.

An aneurism near the sinuses of Valsalva may be mistaken for *aortic insufficiency*. The latter is distinguished by the previous history, absence of arterial degeneration, transmission of the murmur to the xiphoid cartilage, absence of a murmur over the pulmonary artery, and the existence of left ventricular hypertrophy and dilatation. Should the sinus of the right auricle be pressed on, *both* cavæ will be obstructed and the liver will show evidences of congestion.

The diagnosis between *aneurism* of the *arch of the aorta* and of the *innominate artery* is difficult. In the latter the tumor appears earlier in the neck, and on the right side at the sternal end of the clavicle; while aneurisms of the arch are usually limited to the second right intercostal space, or appear at the manubrium sterni or in the episternal notch, frequently extending to the *left* of the median line. Pressure on the right subclavian or common carotid does not lessen the pulsation in aneurism of the arch; while if the innominate alone is involved, the impulse will be markedly diminished. Impaired venous return and neuralgic pains are confined to the right side in innominate aneurism, while the venous congestion is bilateral and pain is on both sides in aneurisms of the arch. The *bruit* of an innominate aneurism is less intense than that of an aortic. The radial pulse is seldom altered in aortic aneurism, while a suppressed radial pulse on the right side is a common and important sign of aneurism of the innominate. The larynx and trachea are often pushed to the left by an innominate aneurism; rarely by an aortic.

Cancer of the pleura, mediastinal tumors, bony exostoses, pulsating empyema, abscesses between *œsophagus* and *trachea, laryngeal disease, intercostal neuralgia, angina pectoris, consolidation* of the *lung* near the *apex*, and *hydropericardium*,—all may be mistaken for a thoracic aneurism.

In *cancer* of the pleura the personal and hereditary history is important. The pain in cancer is constant; in aneurism it is wandering, and shifts with change in direction of the tumor. Anything increasing heart action increases the pain of an aneurism; this is not so in cancer. The pulsation is dilating in aneurism; heaving and lifting in cancer. A harsh double *bruit* is present in aneurism; while if one is present in cancer it is soft and blowing. In aneurism the centre of dulness and the point of maximum dulness coincide; this is not the case in cancer. Enlarged veins and glands (axilla, neck, etc.) accompany cancer; they are not present in aneurism. In aneurism there is a subjective sense of throbbing, *never* present in cancer. Infiltrated cancer of the lung induces *retraction* of the chest-walls, and is not likely to be confounded with thoracic aneurism.

Localized empyema which *pulsates* must occupy the cardiac area and push the heart to the right, and it has no murmur. Besides, the peculiar wandering pain of aneurism is absent in empyema, and in this condition the pulse is not altered. Irregular diurnal fever, chills, and sweatings occur in empyema, never in aneurism. The exploring needle will settle the question.

An *abscess* between the *trachea and œsophagus* is attended by no *bruit*, no pulsation of an expansile character, no shifting pain, no pulse-difference. Deep-seated fluctuation, chills, fever, and sweats accompany it, however.

An *exostosis* below the sterno-clavicular articulation may pulsate, but the pulsation is lifting, *not* expansile, and there is no *bruit*.

Laryngeal disease may be recognized by the vocal changes. A physical examination of the chest, and the laryngoscope will enable one to make a correct diagnosis.

In *intercostal neuralgia*, the three diagnostic points of tenderness, *i. e.*, at the exit of the nerve from the spine, midway between this and the sternum, and at the edge of the sternum where the terminal branches become superficial, will decide between it and aneurism.

Angina pectoris may occur with thoracic aneurism. But in all such cases valvular disease or degeneration of the heart-walls will be found to co-exist. Hence the diagnosis rests on the signs of a tumor in the one case, and the symptoms of structural heart-disease in the other.

Pulmonary consolidation at one apex, with a murmur in the subclavian or pulmonary artery, will be attended by the signs of phthisis and not by those of a tumor. *Fluid in the pericardium* gives a triangular outline of dulness never met with in aneurism.

Prognosis.—Although cases of thoracic aneurism have apparently recovered, the rule is that they terminate fatally. The average duration is about two and one-half years:—some terminate in a few months, others live five or six years. There is always a liability to sudden death. The better the general health and the smaller the swelling, the better the prognosis. The prognosis in aneurism of the ascending arch is better than in any other form of thoracic aneurism. Death may occur from pressure on important organs, or from rupture of the sac.

The sac may open into one of the serous cavities from sloughing, erosion or laceration of its wall; or it may open externally, or into a mucous canal.[1] When the sac bursts into the pericardium or pleura, it ruptures at the thinnest part; if into the œsophagus, trachea, or a bronchus, it breaks at some point of adhesion between the two, which has subsequently become thinned. External openings are produced by gradual atrophy from pressure, or by sloughing of the skin over the tumor. Pneumonia, pleurisy, bronchitis and gangrene may occur as complications to cause death. Pressure on nerves, lymphatics or ducts may induce death from exhaustion. Emboli may arise and become a cause of death.

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[1] In twenty-six ruptures, ten were into the pericardium, five into the left lung or pleura, four into the trachea, three into the right lung or pleura, three into the left bronchus or œsophagus, one externally.

Treatment.—The treatment of thoracic aneurism is divided into those measures which come strictly within the province of the physician, and the more recent surgical procedures. In both, *absolute rest* is one of the essentials. Anything that accelerates or increases the force of the heart's action will do harm, in accordance with the simple physical law that every abnormal dilating force applied to the walls of an aneurismal sac must favor its growth and hasten the fatal issue. Blood rich in nutritive elements more readily deposits its fibrin, thus favoring that formation of laminated layers of fibrin within the aneurismal sac which is the first step in the curative process. Fluids must be taken in minimum quantities. Mr. Tufnell restricts the food taken to two ounces of bread and butter, and two ounces of milk for breakfast; two or three ounces of bread with two or three ounces of meat for dinner, with two to four ounces of milk or claret wine; and two ounces of bread and butter and milk for supper. Mr. Tufnell says that this dietetic treatment, combined with absolute rest in a recumbent position for two or three months, resulted in cure in a large percentage of cases.

Various internal remedies have been used to favor the formation of a coagulum within the aneurismal sac, either by increasing the coagulating power of the blood, or by acting in some specific manner upon the walls of the aneurism itself or upon the adjacent arterial walls. The principal drugs used for this purpose are ergot, iodide of potassium, acetate of lead, and the vegetable astringents. Iodide of potassium and ergot are the only ones that have stood the test of experience. Both are used at the present time, and seem to have power in staying the growth of aneurisms and relieving painful phenomena. A combination of the dietetic, rest, and iodide treatments has arrested the progress of aneurisms in a number of cases under my observation, and I have the records of ten cases of thoracic aneurism where cures were effected and have persisted for over two years. In using the iodide of potash, the patient should be placed in bed and all medication withheld for three or four days until his pulse rate is established. The iodide is then to be given in increasing doses and finally maintained at the highest point possible without increasing this previously established pulse rate.

The only remedy to be relied on for relief of the excruciating pain attending aneurismal development is the hypodermic use of morphine. It not only relieves pain, but by its quieting and regulating influence on the heart it delays the growth of the aneurism. It also diminishes restlessness and impatience, and enables persons who are naturally irritable to obtain the necessary rest which is so important a factor in any plan of treatment. The external application of belladonna to the aneurismal tumor will often afford temporary relief to the local pain. The continued application of an ice-bag to an external aneurismal tumor will often afford temporary relief of the pain and reduce the tegumentary inflammation; its use should not be continued too long. When a patient with aneurism has an undue fulness of the vessels, free purgation with salines will be attended by marked relief for a time.

Surgical Treatment of Thoracic Aneurism.—Thoracic aneurism seldom presents features which justify surgical measures. The methods employed are ligation of one or more of the great vessels in the neck, galvano-puncture, the injection of coagulating substances into the sac, and the introduction of solid bodies with the object of starting consolidation. The two latter methods have only been employed in a few desperate cases, and death has always followed so rapidly that no deductions can be made. On theoretical grounds it is improbable that either method could do good, except in cases of pouched aneurism. Experience shows injection of coagulating fluids to be very dangerous, usually inducing suppuration of the sac. The permanent introduction of wire, horse-hair, and catgut has never been followed by good results; but in at least one case of (ileo-femoral) aneurism no harm resulted. The temporary introduction of several acupuncture needles and their retention from one to two days has been tried with good results in a few cases; it is less dangerous than the other methods mentioned. Galvano-puncture has been employed by Ciniselli in twenty-three cases, with five cures. The same method has been tried by other surgeons, but the clot is liable to break down and cause inflammation of the sac. This plan has been adopted when rupture of the sac was impending, to delay for a time the fatal result. In some cases of supposed innominate aneurism, which proved to be aortic, ligation of the carotid, or of the subclavian, has been followed by marked relief. In two cases of aneurism involving the transverse arch, the left carotid has been tied and the disease cured or arrested for a very long time. It would seem best to perform this when the sac involves *only* the arch. Tracheotomy may be performed only to insure a quiet death.

ABDOMINAL ANEURISM.

An aneurism of the abdominal aorta, or of any of its branches situated within the abdominal cavity, is called an abdominal aneurism. The cœliac axis, the mesenterics, the renal, and the common iliacs are the branches usually involved. The morbid changes are similar to those of thoracic aneurisms, except that the pressure effects are different. The splanchnic nerves, semilunar ganglia, and the solar plexus may be involved. The bile-duct or the renal vessels, the stomach and the duodenum may be pressed on and narrowed. The bodies of the vertebræ may be eroded. Abdominal aneurisms are not so often caused by "atheromatous" changes in the walls of the artery as are thoracic aneurisms.

Etiology.—Its development is always preceded by some form of arterial degeneration. It is rare before thirty-five, and is met with in men oftener than in women.

Symptoms.—Intermittent, paroxysmal pain is its prominent symptom. Agonizing *pain in the back* darts along the branches of the lumbar plexus. This pain is apt to be continuous, and indicates erosion of the spinal column.

Nausea and vomiting may result from pressure on the stomach; dysphagia from pressure on the œsophagus; jaundice from pressure on the bile-duct; changes in the urine from pressure on the renal vessels; and anasarca of the lower limbs from pressure on the inferior cava, or in one limb from pressure on one of the iliac veins. Aneurisms here may burst into the peritoneal cavity, the retroperitoneal tissue, the spinal canal, or into the substance of the mesentery, meso-colon, or great omentum, and in the last-named instances there will be more or less obstruction about the region of the pylorus. They may also open into the intestinal canal, the lung, the pleura, the inferior cava, the pelvis of the kidney, the ureter, bile-passages, or the œsophagus. Rarely are the liver and heart displaced.

Physical Signs.—*Palpation* discovers in some instances a smooth, elastic tumor to the left of the median line. It has an expansive, dilating impulse (rarely double), and synchronous with the radial pulse.

There is dulness over the tumor.

On *auscultation* a single prolonged post-systolic murmur may be heard.[1] A double murmur over the aneurism in front is rare. Seldom can any murmur be heard when the patient is in any other than a recumbent posture.

Differential Diagnosis.—If an abdominal aneurism is of considerable size, the constant pain in the back and the presence of a dilating tumor will establish a diagnosis; forcible *pulsation of the aorta* may simulate an aneurism, but the throbbing is felt along the entire course of the aorta and its branches, and is not localized as in aneurism; then the absence of pain and of the "expansive" impulse and murmur will establish the diagnosis. A *cancerous* or other solid tumor may have a pulsation communicated to it by the underlying aorta; but the knee-chest position will remove doubts. In thin subjects especially, by grasping the solid, uneven mass, it is easy to decide for or against an aneurism.

Prognosis.—Hayden gives fifteen days to eleven years as the extremes; a year or eighteen months is the average duration. After rupture the patients have lived for some time; but death is certain sooner or later.

Treatment.—Posture, rest, a restricted diet, and mild laxatives are advocated in the treatment of abdominal aneurism by Bellingham. Tufnell's plan may also be followed. Iodide of potassium and ergot reduce vascular tension and are highly recommended. Aconite is highly recommended by English surgeons. Pressure, ligation, tourniquets, etc., etc., are measures resorted to by surgeons.

MEDIASTINAL TUMORS.

Cancer and *sarcoma* are, independent of aneurism, the most frequent mediastinal tumors. In rare instances lymphadenomata, lipomata, cysts, enlarged lymphatics, fibromata and osteomata may develop in the mediastinum. The lymphatic glands in the anterior mediastinum are most frequently the seat of these developments, although they may originate in any mediastinal tissue. In exceptional cases the thymus gland is the original seat of the new growths. The primary cause of their development is unknown; they occur at any age, but are most frequently met with between twenty and forty.

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[1] If the patient be placed in the knee-elbow position, and a murmur still persists, then the tumor is in all probability an abdominal aneurism, *not* a tumor to which aortic pulsations have been transmitted.

The *symptoms* of mediastinal tumors are those of pressure, *e.g.*, aphonia, dysphagia, cyanosis, pain, and a sense of constriction about the chest. Displacement of the heart without any other recognizable cause is an almost diagnostic symptom.

The *physical signs* vary with the size and site of the tumor, which may pulsate and have a distinct *bruit*. Mediastinal tumors, mediastinal abscesses, aneurism, pericardial or pleuritic effusions and chronic pneumonia all produce symptoms which are strikingly similar; and the diagnosis of a mediastinal tumor is arrived at mainly by exclusion. The exploring trocar is often the only means by which a diagnosis can be reached.

Prognosis.—Mediastinal tumors sooner or later terminate fatally. Lebert states that their average duration is thirteen months. In a case of Jaccoud's, death occurred on the eighth day.

The *treatment* is palliative.

DISEASES OF THE KIDNEYS.

THE URINE.

The urine in health is a clear, amber colored liquid of acid reaction, saline taste, and having a peculiar aromatic odor. The amount voided in twenty-four hours ranges between forty and fifty ounces. Its specific gravity varies from 1.012 to 1.030; the average being about 1.020. After exposure to the air the acidity of the urine, which is due mainly to the presence of the acid phosphates, continues for a few days, and then an acid or alkaline fermentation takes place. The former is caused by the growth of a round cell vegetable ferment, and is accompanied by the crystallization of uric acid and the precipitation of the acid urate of soda. The alkaline change is the result of the growth of the *micrococcus urea*, and is marked by decomposition of the urea and the formation of carbonate of ammonia and the triple phosphates.

Normal Constituents of the Urine.—Generally speaking, these may be regarded as the products of the metamorphosis of the tissues of the body; the most important *organic* constituents are urea, uric acid, hippuric acid, oxalic acid, kreatinin, xanthin, and the coloring and extractive materials.

Urea.—This substance represents the result of the retrograde metamorphosis of the nitrogenous body tissues and the excess of the nitrogenous elements of the food. It is formed in the tissues, taken up by the blood and lymph, filtered by the kidneys, and appears in the urine to the amount of five to six hundred grains daily. Urea is abnormally *increased* in amount in all febrile and nervous affections, in pyæmia and diabetes; it is abnormally *diminished* in nephritis, anæmia, cholera, and starvation, and may be entirely absent in acute yellow atrophy of the liver. Urea is a feeble base, extremely soluble, and cannot be detected except by chemical examination.

Uric acid is generally found in the urine combined with some base, especially lime or soda. Its origin is similar to that of urea. In health six to nine grains are passed every twenty-four hours; this amount is increased by a highly albuminoid diet and in certain febrile conditions. It is diminished by out-door exercise. Uric acid appears in the urine as a crystalline deposit, which will be described hereafter. Besides the above constituents, the urine normally contains small quantities of kreatinin, hippuric acid and xanthin, which may be said to represent the less completely oxidized products of tissue change.

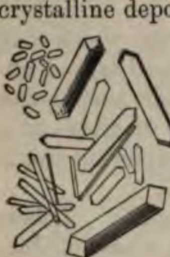

FIG. 117.
Hippuric Acid. × 250.

Coloring and *extractive materials.*—The normal color of the urine is due to the presence of a pigment, called urohæmatin; a substance closely allied to the coloring matter of the bile, and derived from the blood by the action of the liver and spleen. Another normal pigment of the urine is indican, a peculiar substance which under certain conditions gives rise to indigo-blue. The extractives are certain volatile organic acids which give to the urine its peculiar aromatic odor.

The inorganic constituents of the urine are chlorine, sulphuric acid, phosphoric acid, potassium, sodium, calcium, magnesium, oxygen, hydrogen and nitrogen.

Chlorine.—The average amount of chlorine passed daily is about one hundred grains; an increase in this amount has no special significance, but a diminution or absence has been noticed in all acute febrile diseases with the one exception of intermittent fever.

Sulphuric Acid.—The amount daily passed averages about thirty grains. Sulphuric acid in the urine arises from the animal and vegetable food taken into the system, and from changes in those tissues which contain sulphur and sulphates.

Phosphoric Acid.—About fifty grains of this acid are eliminated in the twenty-four hours. It is abnormally increased in all inflammatory diseases of the nervous system, in severe nerve lesions and in rickets. It is abnormally diminished in most febrile and inflammatory diseases, especially in pneumonia and Bright's disease. The rest of the inorganic constituents of the urine, which amount to about one hundred grains daily, will be considered under the head of urinary sediment, as they generally appear in that form.

Albumen.—When albumen appears in the urine it may have its origin in the kidneys or depend upon the presence of pus or free blood; this question can only be settled satisfactorily by the microscope. Albuminous urine is generally of low specific gravity. In diseases of the kidney the serum-albumen which is found in the urine has its origin in the blood, and either by the increase or diminution of the blood pressure within the glomerules, the albumen is transuded within the capsule of Bowman, and then is washed along the uriniferous tubules with the urine. When albumen appears in the urine in acute and chronic Bright's disease some structural change in the kidney is indicated; but it may appear independent of any structural lesions under the following conditions, viz.: febrile and inflammatory diseases, impediments to the circulation of the blood, pregnancy and the puerperal state, saturnine intoxication, hydræmia and atony of the tissues, after the use of certain drugs, and in some people after taking certain articles of food.

Urinary Sugar.—When the urine contains much sugar it is of a pale, yellow color, sweetish taste, and increased in amount. Its specific gravity is always high, generally between 1.030 and 1.040, although cases are occasionally met in which it is as low as 1.008. Sugar, except in a very small quantity, is not found in normal urine, so when it is constantly present in large amounts a grave pathological lesion is indicated. Diabetes mellitus is the only disease in which sugar is found in the urine in large quantities, but traces of it appear after disturbances of the abdominal circulation, after injuries to certain portions of the nervous system, after interference with respiration, in the urine of women just after weaning a child, and sometimes it is temporarily present without any assignable cause.

Bile.—Urine containing bile varies in color from a deep reddish brown to a dark green, and generally has an acid reaction and high specific gravity. The coloring matter of the bile, such as bilirubin, biliverdin, and biliprasin, is the portion which usually appears in the urine in disease. It is especially noticed in those who are jaundiced. The bile salts are sometimes present.

Lactic acid has been found in the urine in diabetes, acute yellow atrophy of the liver, trichinosis and osteomalacia.

Fat.—Fat is not very often found in the urine, but when it is present it gives to that fluid a milky appearance, for it is held in the form of an emulsion by the albumen present. The urine shows a tendency to spontaneous coagulation, and in a short time a white layer rises to the top which disappears on the addition of ether. Under the microscope minute globules of fat, sometimes with blood and lymph corpuscles, are seen.

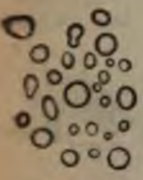
FIG. 118.

Fat globules from chylous urine. × 250.

Leucin and *Tyrosin.* — The urine often contains large quantities of these substances, which arise from the prolonged action of the pancreatic ferment upon the nitrogenous elements of the food.

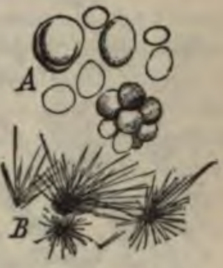

FIG. 119.

Leucin and Tyrosin.

A. Globules of Leucin; B. Tyrosin. × 250.

Leucin appears either in the form of white crystalline scales freely soluble in water, or as small, round, yellow bodies looking something like spherical fat cells.

Tyrosin is in the form of white masses consisting of long shiny needles arranged in star-shaped groups. Leucin and tyrosin appear in those diseases in which oxidation is very greatly impaired, such as acute yellow atrophy of the liver, typhoid fever, small-pox, and in hepatic diseases generally.

URINARY SEDIMENTS.

Many deposits, in crystallizd and non-crystallized forms, appear in the urine, some of which are passed with it, and others are separated from it after its passage. The following are the most important:

Uric Acid.—The strong acids which appear in the urine during the stage of acid fermentation quickly decompose the urates, and set the uric acid free. This is deposited in the form of yellowish-red colored crystals which assume a multitude of forms. The most common are lozenge-shaped and rhomboidal crystals, having angles more or less rounded; they also appear, especially when abundant, as aggregated or flat stellate crystals. Should any doubt arise as to the character of the crystals, dissolve the sediment in a drop of potassic hydrate, then add a little hydrochloric acid, and the uric acid, if present, will recrystallize into one of its numerous forms. The clinical importance of uric acid crystals has already been referred to.

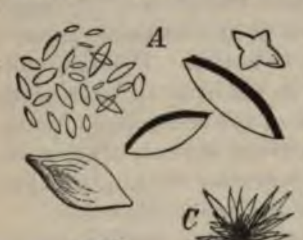

Fig. 120.

Uric Acid.

A. The most common form.
B. Disintegrated crystals.
C. Formation of rounded masses.
× 250.

Urates.—When the urine contains abundant amorphous urates it is generally turbid, but becomes clear on heating. On careful observation a fine powdery sediment will be seen, which clings to the glass and varies in color from a light fawn to a pink. The urates are in a state of solution in normal urine, but when that fluid becomes concentrated or has lost the temperature of the body, the urate of soda will become deposited in an amorphous condition, appearing under the microscope as mossy granules. Urate of ammonia appears only after the urine becomes alkaline, occurring as brownish spherical bodies, with or without fine projecting spiculæ. The urates may be deposited after slight indigestion, great mental and physical exertion, and in acute febrile and inflammatory diseases; they are often the forerunner of urinary calculi, and of derangements of the liver and of the other chylopöetic viscera.

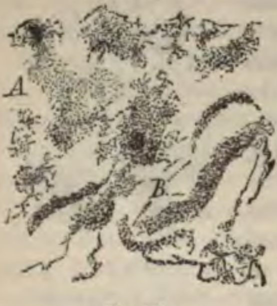

Fig. 121.

Urate of Soda.

A. Amorphous granules in clusters resembling moss.
B. Granules in strings sometimes mistaken for granular casts.
× 250.

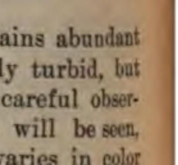

Fig. 122.

Urate of Ammonia.

A. Cluster of Brown Spherules.
B. Isolated Spherules with spiculæ—the "chestnut-bur" crystals. × 250.

Oxalate of Calcium.—Calcium oxalate is often held in solution in the urine, but when it is precipitated it takes one of two forms, either as small, colorless, sharp-edged, octahedral crystals resembling envelopes, or as dumb-bell shaped crystals, entangled with mucus. The presence of the oxalate of lime crystals is due to the reduction of the compounds of oxalic and carbonic acids which are normal to the urine, or to the ingestion of certain articles of food. When oxalate of calcium occurs constantly in the urine it produces the so-called *oxaluria* or oxalic acid diathesis, and is apt to lead to the formation of the mulberry calculi, and in time exert its poisonous effects on the brain and spinal cord. The crystals of calcium oxalate are found in the urine in cases of disturbed respiration, emphysema of the lungs, rachitis, and after epileptic convulsions.

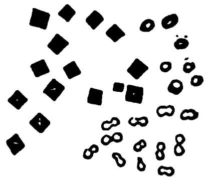

FIG. 123.

Oxalate of Calcium.

The octahedra, most frequently present, are seen on the left. The comparatively rare form of dumb-bells is also shown. × 250.

Earthy Phosphates.—The earthy phosphates are the most common sediment met with in the urine, in fact, when the urine is alkaline they are never absent; they present themselves as the ammonio-magnesian or triple phosphates, or as the phosphate of calcium. During the stage of alkaline fermentation, the ammonia produced combines with the phosphate of magnesium present, and the result is that the crystals of the triple phosphates, being insoluble in an alkaline fluid, are thrown down in large quantities, as also are the crystals of the phosphate of lime, the separation of the latter depending upon the presence of one of the fixed alkalies, as the carbonate of sodium. The crystals of the triple phosphates vary according as they are the result of rapid or slow crystallization: in the former case they assume a feathery form, looking something like two ferns crossing at an acute angle; in the latter case they appear as triangular prisms with bevelled edges. The phosphate of lime forms an amorphous transparent sediment like the urates, but is distinguished from them by the action of heat, which causes an increased precipitation, and by that of nitric acid, a few drops of which clear up the urine. A sediment of the earthy phosphates does not of necessity indicate that there is an abnormal amount in the urine, but it does show the alkaline state of the urine and the possible results of such a condition, and it points out the danger of the formation of phosphatic calculi. An increase of the earthy phosphates has been noted in certain diseases of the bones, such as rachitis.

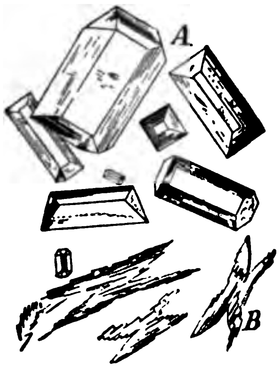

FIG. 124.

Ammonio-Magnesian, or Triple Phosphate.

A. Large colorless prisms.
B. Forms rapidly deposited.
× 250.

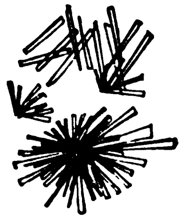

FIG. 125.

Phosphate of Lime.
× 350.

Cystine, which is a crystalline body derived from the liver, is not often found in the urine, but when it is it presents itself in the form of flat hexagonal plates, which are of neutral reaction and can be dissolved by the caustic mineral alkalies. When this substance occurs in the urine it is apt to give rise to calculi. Cystinuria seems to run in families.

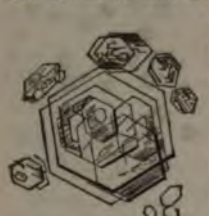

FIG. 126.
Cystine. × 250.

Organized Sediments.—Mucus and Epithelium. All urine contains a varying amount of mucus, derived from the urinary passages and from the bladder, which separates in the urine as a light, flaky cloud. Under the microscope mucus presents itself in one of two forms, either as mucous corpuscles in the form of small, round granular cells containing one or more nuclei, or as transparent masses of mucous coagula, which look very much like granular casts and for this reason have been called mucoid casts. An abnormal amount of mucus in the urine shows that there is irritation at some point along the urinary tract; this may be the result either of a local inflammation or of a general constitutional disease, such as pneumonia or typhoid fever; when there is a mucous sediment in the urine, there is always found entrapped in it a large number of epithelial cells of different varieties, which for convenience of description may be divided into three classes. *First,* round, spherical cells having distinct nuclei derived from the tubules of the kidney, or from the deep layers of the mucous membrane lining the pelvis or from the male urethra. *Second,* columnar and ciliated cells derived from the cervix of the uterus. *Third,* flat cells with large distinct nuclei which have their origin in the bladder or vagina; in the former case they are much larger and granular. The situation of an inflammation confined to some portion of the urinary tract may sometimes be determined by noting the character of the epithelial cells passed in the urine whenever it can be determined whether the cells came from the tubules or pelvis of the kidney, or from the bladder or lower part of the urinary passage.

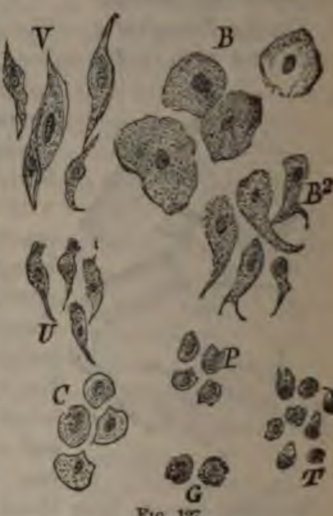

FIG. 127.
Epithelium from Urinary Deposits.
B. Large, flat bladder cells.
B^2. From bladder—deeper layers.
V. Cells from vagina.
U. Ciliated cells from cervix of uterus.
C. From mucosa of uterus.
P. Cells from pelvis of kidney.
T. From collecting tubules.
G. From prostatic portion of urethra.
× 250.

Blood may appear in the urine in varying amounts, and may come from any portion of the tract; when the urine contains blood it will have a reddish or smoky appearance, and deposit, on standing, a coffee-ground like sediment, and will show by chemical analysis the presence of both albumen and fibrin. The appearance of the urine, the amount of blood, and the cause of its presence will vary greatly according to the portion of the urinary passages from which it comes:—*first,* when the quantity of blood is small, and it is equally diffused throughout the urine, in all probability, it is derived from the parenchyma of the kidneys, and especially from the Malpighian tufts; this condition is met with as the result of Bright's disease, congestion of the kidney, injury, the use of certain drugs, such as cantharides, the formation of abscesses secondary to renal infarctions, and from the presence of adventitious growths. *Second*, when the urine contains much blood and distinct clots are visible, it is safe to infer that the blood is derived from the pelvis of the kidney, from the ureters, or from the bladder; in the former case it is generally the result of pyelitis, renal calculi, parasites, or morbid growths; in the latter case it is present as the result of vesical calculi, or erosions and ulcerations of the mucous membrane. Blood may appear in the urine as the result of disease of the urethra, but then the cause of its presence can easily be determined. Certain constitutional causes may give rise to bloody urine, for it appears in the following diseases: fevers, scurvy, purpura, cholera, myelitis, and in the hemorrhagic diathesis.

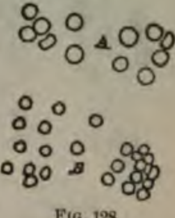

FIG. 128.

Blood.

A. Swollen red corpuscles seen in urine of low specific gravity.
B. Crenated corpuscles from dense urine. × 250.

Pus, when present in the urine, gives to it a milky appearance, and, on standing, a yellowish-green sediment is precipitated which, as long as the urine is acid, can easily be mixed with it, but when the urine becomes alkaline, the sediment will have a gelatinous, ropy appearance and soon undergo ammoniacal decomposition. Albumen is always found in urine containing pus, and varies in quantity with the amount of pus present. The microscopical appearance of pus is sufficient to determine its presence; for the shape, size and granular appearance of the pus corpuscles, with their granular nuclei rendered more distinct by the addition of acetic acid, cannot be mistaken for anything else. Pus appears in the urine either as the result of some suppurative inflammation along the genito-urinary tract or from the rupture of some neighboring abscess, but it must be remembered that in women it may be derived from the genital organs. The significance of pus in the urine depends upon its source, which may be determined by remembering the following points:—if the urine is acid, when voided, the pus probably has its origin in the kidneys; if it is alkaline, its origin is in the bladder; if its presence is accompanied by slight colicky pains over the course of the ureters, probably suppuration is going on in them. In inflammations of the urethra pus can be pressed out of the meatus.

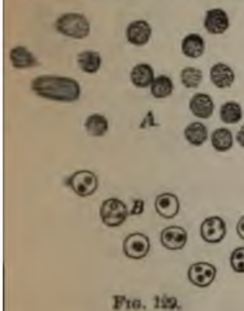

FIG. 129.

Pus.

A.—*Pus corpuscles as ordinarily seen in urine.*
B.—*The same, treated with acetic acid.* × 300.

Casts are peculiar cylindrical bodies consisting of exudative material or coagulated matter formed in the urinary tubules of the kidney as the result of disease, and then washed out by the urine secreted behind them. They vary in size and number according to the nature of the disease which gives rise to them, but it may be rightly stated that the more numerous the casts and the longer time they are present, the more extensive will be the structural lesions in the kidney. The following are the principal varieties of casts met with in the urine:

Epithelial casts consist of tubular masses of renal epithelium, especially from the tubules of Bellini in the medullary portion of the kidneys; they are also, at times, derived from the epithelial lining of the pelvis and calices of the kidney. Occasionally the epithelial cells present a normal condition, but generally they show granular degeneration. This variety of casts indicates desquamative nephritis. When they are found mingled with pus corpuscles a serious inflammatory condition is indicated.

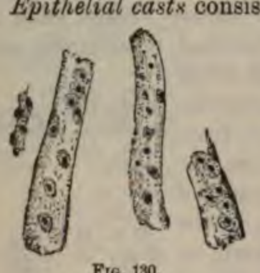

FIG. 130.
Epithelial Casts.
× 250.

Hyaline casts are structureless, transparent cylinders, having a tendency to fracture transversely, and are derived from the fibrinous exudation which has passed through the degenerated walls of the renal vessels and coagulated in the tubules of the kidney. *Large* hyaline casts are met with in the atrophied stage of all forms of Bright's disease. *Small* hyaline casts are found in the acute stage, and sometimes when no lesion can be appreciated.

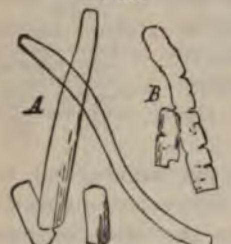

FIG. 131.
Hyaline Casts.
A. Delicate hyaline casts.
B. Dense, so-called waxy casts.

Granular Casts.—These casts are solid, fibrinous cylinders, which have a finely granular appearance caused by the presence of the débris of the degenerated renal epithelium. Blood and pus corpuscles and granular cells are often found embedded in this granular matter. These casts are most often found in the advanced stages of Bright's disease, and indicate the large white or granular kidney, or that extensive destruction of the parenchyma of the kidney is taking place.

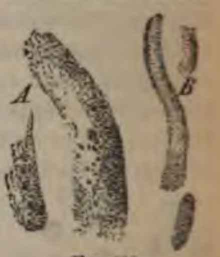

FIG. 132.
Granular Casts.
A. Large granular casts.
B. Small finely granular casts. × 250.

Fatty casts are made up of a material supposed to be mixture of olein with cholesterin, and some albuminous matter; fat globules, varying greatly in size, and also some epithelial cells and granular material may be found in them. These casts show that the degenerative changes in the kidney are fatty, and they indicate the same conditions as granular casts.

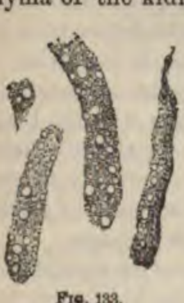

FIG. 133.
Fatty Casts.
× 250.

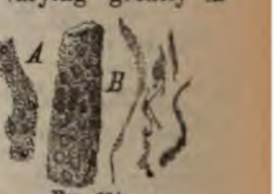

FIG. 134.
Blood Casts.
A. Collecting-tube blood casts.
B. Mucous casts.

Blood casts consist of coagulated fibrin and red blood corpuscles. By some they are thought to indicate the disease of the renal blood-vessels, especially amyloid or fatty degeneration of the Malpighian tuft.

Spermatozoa are occasionally met with in the urine, and give to it, when present in abundance, a milky white appearance. If a drop of the urine be placed under the microscope the characteristic tadpole appearance of the spermatozoa can easily be recognized. Spermatozoa appear in the urine after coitus, involuntary nocturnal emissions, and occasionally after defecation. They have also been found in the urine of typhoid fever patients.

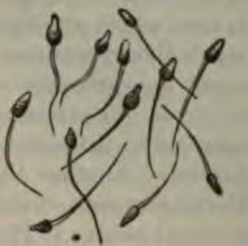

FIG. 135.
Spermatozoa.
× 500.

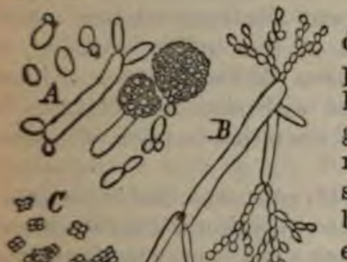

FIG. 136.

A. Torula cerevisiæ.—B. Penicillium glaucum.—C. Sarcinæ Vent. × 300.

Animal and Vegetable Organisms.—Fully developed hydatids and echinococci, or only portions of these, may appear in the urine, having been developed at some portion of the genito-urinary tract or poured into it by the rupture of some hydatid cyst. A small unisexual parasite, the Bilharzia hæmatobia, has been found in the urine, especially during the epidemics of hæmaturia occurring in Northern Africa. Bacteria, or fermentation spores, form in urine which is undergoing decomposition. Sarcinæ have been observed in alkaline urine. Penicilium glaucum has been found in acid albuminous urine. Torula cerevisiæ often forms in diabetic urine.

URÆMIA.

Decrease in the elimination of the solid constituents of the urine is followed by two well marked combinations of symptoms,—one in which nausea, vomiting, and convulsions are indicative of cerebral irritation ; and a second in which headache, drowsiness, and coma depend upon decrease and abolition of nervous function. These conditions, with associated symptoms, are called uræmia.

The primary cause of both these conditions is to be found in a failure of the kidneys to perform their normal function of elimination, and the consequent accumulation in the circulation of some or all of the poisonous elements of the urine. This condition may occur in the course of any disease in which suppression of the renal secretion takes place ; such arrest of the function of the kidneys most frequently occurs in the different forms and stages of Bright's disease, in the puerperal state, in connection with the surgery of the urethra, cystic, tubercular, and cancerous disease of the kidney, and in suppurative nephritis ; by far the largest number occur in acute Bright's disease.

A number of theories have been advanced in regard to the exact element which acts as the poisonous agent in uræmia. The earliest accepted view is that which attributes the symptoms of uræmia to *retained* urea. Frerich's theory was that urea, as urea, was innocuous, and that the poisonous agent was carbonate of ammonia resulting from decomposition, in the blood, of urea into carbonate of ammonia and water, which decomposition was ascribed to the action of a ferment in the blood; this theory has been disproved. Another hypothesis which has attracted much attention is that the phenomena of uræmia are due to cerebral anæmia and the attending cerebral œdema. This is the *mechanical* theory. Still more recent experimenters have claimed that urea is formed in the kidneys from nitrogenous matter in the blood, and that uræmic manifestations mainly depend upon the accumulation, in the blood, of kreatin and kreatinin. Again, others have claimed that the phenomena of uræmia are due to the retention, in the circulation, of the products of nerve waste. It has also been claimed that some forms of uræmia *may* be associated with structural changes in the brain, similar to those which occur in the retina in cases of neuro-retinitis.

Mahomed (Brit. Med. Jour., 1877, ii. 10–42) calls attention to the cerebral lesion which, he says, accounts for the epileptiform convulsions of uræmia, viz., numerous punctiform hemorrhages in the gray matter of the cerebral convolutions. They are true hemorrhages. He ascribes other cerebral symptoms to œdema of the brain following increased tension. Similar to œdema is the "hyaline exudation into nervous tissue" found by Gull and Sutton. Similarly, headache results from increased tension *without* exudation. Gubler (Paris, 1878) describes the "diminished number and impaired respiratory capacity of the red blood corpuscles" in uræmia, due (G. states) to kreatinin *and* ammonium carbonate. Hence there is more or less "asphyxiation of the nerve centres." He also believes *arterial spasm* to result in *sudden* retention of effete products. He regards the dyspnœa as at times due to kreatinin, at times to ammonium carbonate; in one the breathing is hurried, in the other it is the "*Cheyne-Stokes respiration.*"

The experiments and facts upon which these theories are based lead to the following conclusions: that uræmic toxæmia depends upon the presence in excess, in the blood, of poisons normally eliminated by the kidney. This excess may be due to failure in kidney function or abnormal formation of the poisonous elements. Four classes of poisons are now recognized:

(1.) Products of tissue disintegration.
(2.) Aliments; prominently the potash salts.
(3.) Absorbable products of intestinal decomposition.
(4.) Various secretions; as, the bile and saliva.

The uræmic poison is complex in character, and may vary not only in the proportion of its component elements, but in the elements themselves. The potash salts are regarded by many authorities as the most poisonous elements of the urinary secretion.

Symptoms.—An acute uræmic attack is usually preceded by certain premonitory signs, such as œdema in various parts of the body, restlessness or an almost irresistible desire to sleep, vertigo, headache, delirium, nausea, vomiting, diarrhœa, and impaired vision. The countenance has a pale, waxy or dingy appearance, and the urine is scanty, high-colored, bloody, albuminous, and contains casts; the body and extremities may become violently convulsed, or the patient may pass rapidly into a state of coma.

The *convulsions* may consist of a single paroxysm, or a succession of paroxysms may follow one another at intervals of a few minutes or several hours, the patient during the interval lying in a state of more or less profound insensibility. They may almost exactly simulate epilepsy, or they may be unattended by loss of consciousness. During the convulsions the face becomes livid, the eyes are glassy, and the pupils are contracted or dilated; at the commencement of the convulsive attack, they are generally contracted. Frothy mucus, which is sometimes bloody, collects around the mouth, and there is a strong urinous odor emanating from the perspiration. The pulse is accelerated, and the temperature is raised in some instances as high as 107° F. A low temperature may be present in the aged. Sudden coma may occur *with* convulsions. Restless delirium is the chief symptom in many cases. Intense dyspnœa, and articular symptoms are very rare.

Uræmic coma may come on gradually, twenty-four or forty-eight hours elapsing before the stupor is complete, or the patient may fall suddenly into a state of profound coma, its advent resembling an attack of cerebral apoplexy. Headache, giddiness, disorders of vision, vomiting, or delirious excitement may precede the coma. There are periods when the coma is so profound that nothing arouses the patient; at other times he is easily aroused, or arouses himself and attempts to speak and sit up, swallowing fluids with difficulty. If the system has become accustomed to the presence of the urinary poisons, a considerable excess of urea and effete urinary products may exist in the blood for a long period without giving rise to any but the premonitory symptoms of acute uræmia. When once this balance is destroyed and a certain excess of urea and its allies in the blood is reached, the kidneys become embarrassed by the excessive demand made on their excreting power, and rapid and intense renal congestion follows, but either convulsions, coma, or both, result in this way; acute uræmia may be developed in the chronic as well as the acute stage of renal disease. Uræmic coma is always accompanied by stertor. The stertor is peculiar: it is not the "snoring" of apoplexy, but a sharp, hissing sound produced by the rush of expired air against the teeth or hard palate. At first the respirations are accelerated, but they soon become slow and labored; the pupils are dilated, but they are not irregular; they may be normal; they react slowly to light. The pulse is increased in frequency and lacks firmness; at first the temperature is raised, but after a time it falls below the normal standard; the face is pale.

Differential Diagnosis.—Acute uræmia simulates in some of its phenomena those diseases in which convulsions and coma are prominent symptoms.

The phenomena of an *epileptic seizure* are almost identical with those of an uræmic convulsion, and it is exceedingly difficult to distinguish one from the other. If the previous history is known, the chronic character of the epilepsy will in some instances distinguish it from acute uræmia, and an examination of the urine will generally determine the uræmic character of the convulsions; in epilepsy one side is convulsed more violently than the other; while in uræmia both sides of the body are equally convulsed. In epilepsy the temperature is *not* elevated, and although there is a loss of consciousness, reflex sensibility continues from the beginning to the end of the paroxysm, which is not the case in uræmia. Immediately following uræmic convulsions there is deep coma; following an epileptic seizure there is merely a deep sleep, from which the patient may be aroused. The initial cry and corpse-like pallor of the face in epilepsy are wanting in uræmia.

In *cerebral apoplexy* coma always precedes convulsions, and with the convulsions there is facial paralysis and hemiplegia; there is also clonic spasm of the paralyzed parts. The urinary symptoms of uræmia are absent and the stertor is less sharp and hissing.

In *hysterical convulsions* the patient falls with a scream into a convulsive, tetanic or cataleptic condition. Close inspection shows that the patient is not unconscious, and the pupils are normal, as are the pulse and temperature. The limbs are jerked irregularly, the breathing is spasmodic and is attended by a choking sensation; opisthotonos is very common. There is no lividity of the face, nor distention of cervical blood-vessels, and the close of the paroxysm is usually accompanied by the discharge of a large quantity of pale urine,—non-albuminous and free from casts.

Cholæmic convulsions very closely resemble uræmia, but may be distinguished from them by the jaundice which precedes or accompanies their development, and by the antecedent history of hepatic disease. Convulsions originating in meningitis and other cerebral affections are distinguished by the accompanying characteristic symptoms of these affections.

The points in the differential diagnosis of *uræmic coma* are similar to those of uræmic convulsions. It may be distinguished from the coma of *apoplexy* by the absence of paralysis.

From *opium poisoning* it may be distinguished by the rise in temperature. The temperature in opium poisoning is often below the normal. In opium coma the respiration is slow and peculiar, and the pupils are uniformly contracted. Uræmic coma is distinguished from *epileptic coma* by the antecedent history of the patient, the presence of bloody froth about the mouth, and the indentations on the side of the tongue; from *alcoholic coma*, by the temperature, and the character of the breathing, which is "puffy" in alcoholic coma, and a hissing stertor in uræmia. In all cases of coma, an examination of the urine is important before reaching a diagnosis.

Prognosis.—From experiments as well as from the clinical history of acute uræmia, it is evident that the primary cause of death is a poison, the exact nature of which is obscure, but which resembles in its action narcotic poisons. This poison acts primarily on the nerve centres, and produces changes in the blood which interfere with or arrest oxygenation. This action is followed by certain structural changes in the different tissues of the body. When these poisons are introduced into the circulation in small quantities, so that its elimination can be effected in a short time, it only temporarily disturbs the functions of organic life; but when it is introduced in large quantities, oxygenation of the blood is arrested, and it undergoes certain changes which render it incapable of supporting life. The prognosis, then, in each case will depend upon the amount of the poison, and the length of time the system is under its influence.

If the symptoms of excessive uræmic toxæmia are present, and there are evidences that the poisoning has been going on for a considerable time, the prognosis is much more unfavorable than when the acute uræmic symptoms are mild and of recent date.

Treatment.—It is claimed that the most important thing to be accomplished in the treatment of acute uræmia, is to secure as rapidly as possible a free eliminative action of the skin, bowels, and kidneys. The favorite method of elimination is by diaphoresis, accomplished by the hot-air baths. Pilocarpin has recently been used to accomplish the same results. In connection with diaphoresis, a vicarious action of the bowels is induced by the administration of drastic purgatives, such as elaterium, *pulvis purgans* and scammony. The testimony in regard to the use of diuretics is conflicting. Many object to their use, on the ground that inflamed organs should not be stimulated.

Digitalis acts efficiently—is *diuretic* without stimulating the kidneys. It increases the power of the heart's action and increases the tension in the Malpighian tufts. The diminished secretion of urine is due to obstruction in the capillary circulation of the kidneys. Digitalis, by increasing the heart power, overcomes such obstruction. To obtain its effects in such conditions, larger doses are required than are usually administered. My rule of practice in these cases is to give half an ounce of the infusion every three hours for twenty-four hours, or at least until its specific effect is produced. In the majority of severe cases of acute uræmia, when the patient is in convulsions or coma, the excretory functions of the skin, bowels and kidneys are completely arrested, so that diaphoresis cannot be induced, or, if induced, it is not eliminative, and the bowels do not respond to purgatives although the patient may swallow them in large doses, and digitalis in large doses fails to restore the urinary secretion. At one time under such circumstances free general blood-letting was used extensively, but the result was unsatisfactory.

If there is acute uræmia, the avenues of elimination are shut off, and the question arises:—what means have we to counteract this uræmic poison, and open again the avenue of its elimination, or, at least, to hold the patient until the normal eliminating process shall be re-established? The first indication is to diminish reflex sensibility, and subdue spasmodic muscular paroxysms, for these, if continued, will either directly terminate life, or end in equally fatal insensibility. The remedy which for some years has been employed for the accomplishment of these results is chloroform. It has been extensively used, and, I believe, is regarded as the safest and most reliable means for controlling uræmic convulsions. Hydrate of chloral and bromide of potash are also more recently recommended, but their action is not swift or powerful enough. Although many authorities recommend the use of chloroform in uræmic eclampsia, few make mention of its employment in acute uræmia independent of the puerperal state. Its only known clinical effect is to control muscular spasm, and in a large proportion of cases it fails to give more than temporary relief to those patients who pass from successive convulsions into a state of complete coma, and die without any apparent neutralizing effect from the chloroform. In the few cases in which I have administered chloroform in non-puerperal uræmic convulsions, it has seemed to me to have no other effect than to arrest convulsive movements by rapidly hastening the patient into a state of insensibility. In no instance have I known its administration to be immediately followed by diaphoresis or a return of the urinary secretion. It has seemed to be more difficult to establish diaphoresis or diuresis by diaphoretics and diuretics in patients with uræmia to whom chloroform had been administered, than in those who had not taken it.

Therefore, I believe that while chloroform temporarily controls muscular spasm, it prejudices the chances of ultimate recovery by the changes its inhalation produces in the blood, which changes increase rather than retard the development of the uræmic toxæmia. With these impressions one naturally seeks an agent which not only has the power to control muscular spasm, but shall also at the same time tend by its action to reopen the avenues of elimination, either by counteracting the effects of the uræmic poison on the nerve centres, and thus facilitating the action of diuretics and diaphoretics, or by acting itself directly as an eliminative.

I believe *morphine* administered hypodermically to be such an agent.

There are two questions that very naturally present themselves in connection with the use of morphine in acute uræmia.

First. Can morphine in full doses be administered without danger to patients with acute uræmia?

Secondly. What are the effects of such administrations?

If one turns to recognized authorities for an answer to the first of these inquiries, he will find that nearly all make mention of the use of opium in uræmic toxæmia only to warn against the danger attending its administration. During the first years of my professional life, I regarded opium as one of the most dangerous remedial agents that could be administered to uræmic patients, rarely daring to give more than five grains of Dover's powder to a patient with albuminous urine, and if fatal coma followed such administration, more than once do I remember to have felt that a Dover's powder which I had administered might have been the cause of the fatal coma. In 1868 I administered my first hypodermic injection of morphine to a patient with acute uræmia. The effects which followed its administration in this case taught me that in some cases with marked uræmic symptoms morphine could be administered hypodermically not only safely, but with apparent advantage. Since that time I have used morphine hypodermically in the treatment of these patients, not only during the premonitory stage, but also during the active manifestations of uræmic intoxication, and its administration has been uniformly followed by good results. In no instance have I caused a fatal narcotism.

From the histories of quite a large number of puerperal and non-puerperal cases of acute uræmia, in which morphine was successfully used, I have reached the following conclusions :[1]—*first*, that morphine can be administered hypodermically to some if not to all patients with acute uræmia without endangering life. *Secondly*, that the almost uniform effect of morphine so administered, is, first, to arrest muscular spasms ; secondly, to establish profuse diaphoresis ; thirdly, to facilitate the action of cathartics and diuretics, especially the diuretic action of digitalis. Thus, morphine administered hypodermically becomes a powerful eliminating agent. The rule is to give small doses at first, not to exceed one-sixth of a grain. If convulsions threaten, and a small dose does not arrest the muscular spasm, it may be increased to one-quarter or one-half of a grain, and the hypodermics may be repeated as often as every two hours. It must be given in sufficient quantities to control convulsions; neither the contraction of the pupils nor the number of the respirations is a reliable guide in its administration.

I would not discard all (perhaps none) of those means which have been relied on for the relief of patients in acute uræmia, but would urge that hypodermic injections of morphine not only control muscular spasms, but aid in establishing the eliminating processes, and thus become another means of saving life in these often fatal cases. Dry and wet cupping, leeching and poulticing over the loins may be employed to aid in this re-establishment of the suppressed renal function.

DISEASES OF THE KIDNEYS

will be considered in the following order :

I. *Renal Hyperæmia.*
II. *Renal Hemorrhage.* (Embolism and Infarction.)
III. *Nephritis.* (Bright's Disease.)
IV. *Pyelitis.*
V. *Hydronephrosis.*
VI. *Cystic Kidney.*
VII. *Renal Calculi.*
VIII. *New Growths.* (Cancer, etc.)
IX. *Parasites.* (Hydatids.)
X. *Perinephritic Abscess.*
XI. *Hæmaturia.*
XII. *Chyluria.*
XIII. *Cystitis.*

RENAL HYPERÆMIA.

(*Congestion.*)

Renal hyperæmia may be active or passive. Passive renal hyperæmia, or congestion, is almost always due to a mechanical obstruction of the venous circulation, and is sometimes called "chronic renal congestion."

Morbid Anatomy.—*Active renal hyperæmia* has its seat mainly in the renal arteries and in the Malpighian tufts. The kidneys are much increased in size ; the hyperæmia may involve the cortical or medullary portion, and may be more intense in one portion than another ; it is usually most marked at the base of the pyramids. The kidneys are of an unnaturally dark color, their capsule is non-adherent, their surface is smooth, and they are softer and moister than normal.

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[1] New York Med. Rec., Aug. 1, 1873.

On section, dark spots are noticed scattered over the cut surface which correspond to the Malpighian tufts, and the vessels of the cones are filled with blood. A dark fluid follows the section, which is partly serum and partly blood. The stars of Verheyen are prominent. A *microscopical* examination shows that these changes are due to an engorgement of the blood-vessels, and a more or less abundant infiltration of serum into the intertubular structure of the kidneys.

Passive hyperæmia, or "chronic renal congestion," has its seat in the veins, which are overfilled with venous blood, while the amount of blood contained in the arteries may be even less than normal. The kidneys are but slightly, if at all, increased in size, are firmer than normal, their capsule is non-adherent, and their surface is smooth and of a uniform red color. In chronic cases the surface is uneven. The tubular epithelium is granular, opaque and flattened from coagulated fibrin which may partially fill the lumen of the tubes. The convoluted tubules may be filled with blood. The stroma is unaltered. Hyperæmia of, and hemorrhages into the mucous membrane of the pelvis and ureters may occur in very severe cases.

Upon section, it will be noticed that the medullary portion is of a darker color than the cortical, that the cortical portion has streaks of red rather than an uniform redness. The Malpighian tufts are not prominent. The veins are dilated, tortuous, and varicose, the abnormal hardness of the kidney being due to the constant distended condition of the efferent capillary vessels. The epithelium of the convoluted tubes may have a peculiar stiff appearance, not the result of an inflammatory process. Coagula of yellowish hyaline material are sometimes found in the glomeruli, arranged in concentric layers.

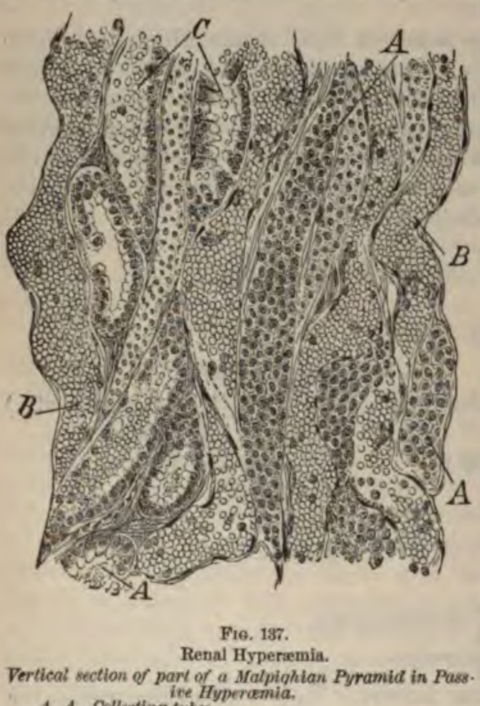

FIG. 137.
Renal Hyperæmia.
Vertical section of part of a Malpighian Pyramid in Passive Hyperæmia.
A, A. Collecting tubes.
B, B. Dilated, tortuous vessels.
C, C. Collecting tubules containing blood. × 300.

Both these varieties of hyperæmia may lead to, or be accompanied by inflammatory processes in the tubular and intertubular structures of the kidneys. Active hyperæmia may be the stepping-stone to acute parenchymatous nephritis. It is only a step from congestion to some of the more chronic inflammatory forms of kidney disease. When to passive hyperæmia there is added inflammatory swelling of the epithelium of the tubules, the kidneys will be enlarged, and the epithelium of the convoluted tubules will be swollen, granular, fatty, and disintegrated. When the inflammation extends to the stroma the kidneys will be diminished or enlarged, but retain the stony hardness of non-inflammatory congestion. The capsule will be adherent, and on section the cortical portion will be slightly diminished, and there will be patches of new connective-tissue throughout its substance,—the process not being unlike the "cirrhotic" form of Bright's. If the passive hyperæmia is due to heart disease, the kidneys will be increased in size, the capsule will be non-adherent and the surface smooth. The cortical substance will be pale instead of red, and the medullary portion of a darker color than normal, the gross appearance very closely resembling that of the "large white kidney." There will be well-marked changes in the epithelium of the tubules, in the stroma, and in the walls of the arteries. These are the *large stony kidneys* of chronic heart disease. Chronic passive hyperæmia is sometimes called "cyanotic induration."

Etiology.—*Active renal hyperæmia*, or fluxion, may be produced by exposing the body to sudden changes of temperature, by any of the blood poisons which give rise to the acute infectious diseases, by malaria (and it is sometimes a prominent feature of a violent malarial paroxysm), by the prolonged and excessive use of certain drugs which give rise to irritation of the urinary passages, as cantharides, copaiba, turpentine, cubebs, nitrate of potash, carbolic acid, etc., and by the irritating condition of the urine in diabetes, cholæmia, etc. It sometimes accompanies Basedow's disease. The early stage of acute inflammations of the kidneys is attended by active renal hyperæmia. Paralysis of the vaso-motor nerves of the kidneys (supposed to occur in hysteria and allied states, polyuria, etc.) is said to be a cause of active renal hyperæmia.

Passive renal hyperæmia, or renal congestion, has its most frequent cause in organic disease of the heart. All valvular lesions of the heart, or structural diseases of the cardiac valves which interfere with venous return, come under this head, as well as all those forms of pulmonary disease which interfere with the pulmonary circulation, and are followed by dilatation of the right heart, *e. g.*, emphysema and fibroid phthisis. Congestion may also be produced by pressure on the emulgent renal veins or inferior vena cava in pregnancy and by other abdominal tumors. The formation of a thrombus is also followed by it. Some of the cases of so-called albuminuria in pregnancy are examples of passive hyperæmia from the pressure of the pregnant uterus.

Symptoms.—The symptoms of both varieties of renal hyperæmia are for the most part confined to changes in the urine. In active hyperæmia the urine is scanty, high colored, of high specific gravity, containing a large percentage of albumen, with few blood casts and hyaline tube casts.

In *passive hyperæmia,* without any structural changes in the kidneys, the quantity of the urine is not much diminished, its specific gravity remains about normal, the amount of albumen is small, and only small hyaline casts are present. It is often of an acid reaction, and deposits urates. The amount of urea is a little below normal. The simultaneous appearance of blood and albumen in the urine is so common in renal congestion that the presence of albumen alone, without blood globules, almost excludes it. Besides the changes in the urine in active hyperæmia, there is usually slight œdema of the face and lower extremities, with nausea and a persistent headache.

Passive hyperæmia is often produced by chronic cardiac or pulmonary disease, attended by a cough with a watery blood-stained expectoration, and by dyspnœa that often becomes so severe as to prevent the patient from lying down. The cough and dyspnœa depend in part upon the accompanying heart or pulmonary disease, but there is also a nervous element in it which is characteristic of the renal complication. There is loss of appetite, nausea and occasional vomiting; there is a continuous headache, restlessness and insomnia, which, added to the dyspnœa, make the patient's condition distressing. There is loss of flesh and strength, and steadily increasing anæmia. These symptoms gradually become worse, and general dropsy develops, and the patient may die from the general anasarca, or from convulsions and coma.

The history of these cases varies greatly: some get progressively worse, others pass from an apparently hopeless condition to one of comparative comfort, and these attacks are repeated at intervals for a long period. However desperate the condition may appear, a return of a comparatively comfortable condition is always possible. That form of renal congestion which is so often met with in pregnancy is usually accompanied by the presence of albumen and casts in the urine before any other symptoms are developed; afterward the patients become anæmic, and suffer from persistent headache, vomiting, and œdema of the face, feet and legs; they become "water-logged." In a few cases the first symptom may be a convulsion. In all cases the thing to be dreaded is the onset of convulsions, which rapidly follow each other until coma is reached.

Differential Diagnosis.—Renal congestion is distinguished from *Bright's disease* by the general condition of the patient, the presence or absence of cardiac or pulmonary disease, or venous obstruction. The urine, though scanty, is above normal in specific gravity, and always deposits blood, renal epithelium, or tube casts.

Prognosis.—The prognosis in active renal hyperæmia, when the cause is of a transient character, is good. Renal congestion which occurs in the advanced stage of cardiac disease and pulmonary emphysema has much to do with causing a fatal termination, and after it is once developed it is never recovered from. That form of active hyperæmia which occurs in congestive malarial fevers is sometimes so intense as to entirely arrest the function of the kidneys, and then it becomes a direct cause of death. The renal congestion of pregnancy is usually relieved by the removal of its cause, which should never be delayed if the symptoms become urgent.

Treatment.—The most important thing to be accomplished in the treatment of active renal hyperæmia is to find out, and as quickly as possible remove its cause. The treatment is to be addressed to the kidneys. Place the patient in bed in a room with a temperature of 80° F., and apply a dozen wet cups over the lumbar region. Administer a powerful drastic purge, induce free diaphoresis, and let the patient drink freely of diluted mucilaginous drinks. The hot-air or warm vapor-bath, and even blood-letting in intense fluxion, are to be employed. Camphor is advocated in some cases of active hyperæmia. In passive renal hyperæmia the main thing to be accomplished is to relieve the venous congestion ; it is to be remembered that there is too much blood in the veins and too little in the arteries.

There are three ways of restoring the natural state of the circulation :—

1st. By general bleeding.

2d. By increasing the force of the heart's action.

3d. By causing the dilatation of the capillary arterioles.

A free bleeding from one of the large veins will temporarily relieve the venous congestion and cause a better filling of the arteries, but it exhausts the patient, and is only admissible in the renal congestion of pregnancy when the symptoms are urgent. By increasing the propelling power of the heart, the amount of blood in the arteries is increased and that in the veins diminished. This is the usual mode of procedure in the passive renal hyperæmia which depends upon chronic heart and lung disease. Digitalis is the drug which has been most extensively employed to accomplish this. It must be given in full doses and continued until the desired effect is induced. The best mode of its administration is in the infusion ; a tablespoonful of the infusion of the leaves may be given every three or four hours until its specific effect is produced or the quantity of urine is greatly increased. Recently the fluid extract of *convallaria* in half-drachm doses has been recommended as a substitute for digitalis. My experience with it has been very unsatisfactory ; its action is not only temporary, but far less certain than digitalis.

The drugs that seem to have some power in dilating the capillaries and arterioles are nitrite of amyl and nitro-glycerine. The nitrite of amyl may be given by inhalation in doses of from three to five drops every four hours. The nitro-glycerine may be given in a one per cent. alcoholic solution, one drop every three or four hours. Under its use albumen has disappeared from the urine in quite a number of instances, and remained absent so long as the patient continued the drug.

If counter-irritation is employed it must be mild in character—a few dry cups over the lumbar region, or some mild embrocation is all that is necessary. The intestines should be occasionally unloaded by a full dose of calomel combined with rhubarb. When the venous obstruction is directly mechanical, as in pregnancy and fluid accumulation in the abdominal cavity, something may be accomplished by so changing the position of the patient as to relieve the pressure on the renal veins.

If passive renal hyperæmia, especially in heart disease, is attended by great restlessness and dyspnœa, morphine may be given hypodermically in sufficient quantities to give relief and make the patient comfortable, even though but a small quantity of urine is being passed.

RENAL HEMORRHAGE.

(*Embolism and Infarction.*)

Renal congestion and renal hemorrhage are very often associated, for renal hemorrhage often occurs as a result of renal congestion.

Morbid Anatomy.—The anatomical changes in a kidney which is the seat of renal hemorrhage do not differ essentially from those already described as present in a renal hyperæmia, unless there are hemorrhagic infarctions or renal calculi. Blood may be effused into the uriniferous tubules or the interstitial tissue, giving rise to ecchymotic spots varying in size, from which, on section, blood flows freely. The vessels will be found ruptured, and the epithelia and stroma of the kidney are stained with blood pigment. The epithelia soon become opaque, granular, and infiltrated with fat, and finally disintegrate. Incident to the great increase in the blood pressure, diapedesis of the red corpuscles may occur; this is true renal hemorrhage, having its origin in the Malpighian tufts. The blood escapes between the vascular tuft and its capsule, which is slightly distended.[1]

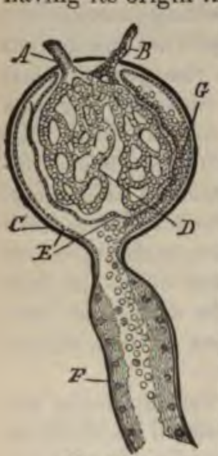

Fig. 138.

Renal Hemorrhage.

Diagram showing Hemorrhage from the vascular tufts of the glomeruli.

A, B. Afferent and efferent vessels; C. Capsule; D. Tuft; E. Epithelium covering tuft and lining capsule; F. Origin of convoluted tubule. At G, the blood is seen escaping from the tuft and entering the tubule.

The most frequent form of renal hemorrhage is that which occurs in connection with *renal embolism* and *infarction*. Its occurrence is marked by the development of hard uniform masses in the cortical portion of the kidney; these masses are usually wedge-shaped, and have their sharp edges toward the hilus of the kidney and their base toward its surface.[2] They vary in size according to the size of the vessel obstructed; they may be capillary, and then are of very small size. These infarctions when first formed are of a dark red color, and are as firm as normal kidney-tissue; very soon they begin to change in color, losing their dark red hue and becoming lighter, and their centres present a yellowish appearance; sometimes they undergo cheesy change, which always commences at their centres. Around these infarctions a zone of redness is formed; this zone is in the normal kidney-tissue, beyond the infarction. A congestion takes place in the vessels, due to changes in the uriniferous tubes adjacent to the capillaries in that portion of the kidney which surrounds the infarction; there is also a more or less rapid production of cells in this surrounding zone. If the infarction does not disappear by absorption, this zone-change continues until there is more or less cicatricial tissue developed about the infarction, which shrinks in consequence of the contraction of the tissue, gradually diminishes in size, and after a time disappears altogether, leaving only cicatricial tissue to mark its former site. The surface of the kidney may be depressed over an atrophied infarction.

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[1] Cornil and Ranvier.

[2] Rayer's so-called "*Rheumatic Nephritis.*"

On the other hand, the production of cells may be so rapid and abundant that the entire mass undergoes purulent transformation, producing *abscesses* which will occupy the seat of the infarction. This is one of the ways in which abscesses are formed in the kidneys. In these cases there is always a certain degree of suppuration occurring at the margin of the affected area. Again, these infarctions may undergo a still more rapid degeneration, increasing in size and becoming necrotic, so that at the autopsy a gangrenous mass is found as the result of the necrotic change which has taken place in the infarction. More or less suppuration also attends it. Again, there may be little masses found scattered throughout the substance of the kidney, especially in the cortical portion, looking very much like ecchymotic spots, which are simply capillary thrombi: these are usually due to some slowing of the circulation in the capillary vessels. These capillary thrombi may be very numerous, and they may undergo changes similar to those which take place in the larger infarctions.

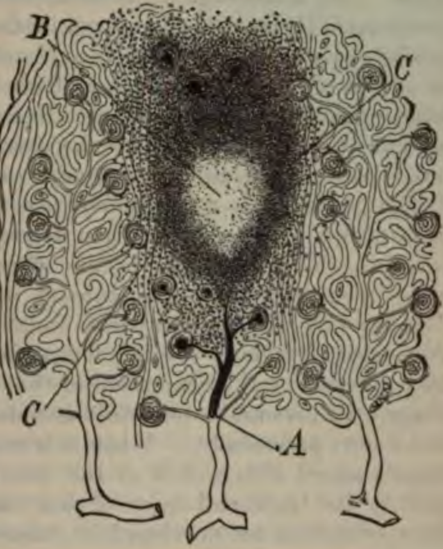

Fig. 139.
Renal Hemorrhage.
Diagram illustrating Renal Infarction.
A. Embolus in an interlobular artery.
B. Cheesy centre of the infarct.
C, C. Zone of redness.

At the autopsy the kidneys may be found studded with minute abscesses; unquestionably these little collections of pus are nothing more than minute capillary infarctions or thrombi which have undergone purulent transformation. Thus a single abscess or many abscesses of the kidney may form as the result of infarctions. This form of renal hemorrhage is especially liable to occur in passive renal hyperæmia.

Etiology.—Intense hyperæmia of the kidney is a cause of renal hemorrhage, especially in the first stage of acute nephritis. It may also result from injuries, and in connection with the development and passage of renal calculi. It may also occur in connection with the development of morbid growths in the kidney, especially cancer. Blood changes, such as occur in purpura, scurvy, etc., may cause it. Passive obstructive hyperæmia from cardiac disease may become so intense as to give rise to it, with or without the occurrence of infarctions.

Symptoms.—It is attended by no constant or distinctive subjective symptoms. Our knowledge of its occurrence, during life, rests almost exclusively upon the results of an examination of the urine. Its existence cannot be recognized, unless the blood is effused into the uriniferous tubules or into the hilus of the kidney and discharged in the urine. At autopsies, large infarctions of the kidney are often found which, during life, have given no indication of their existence, because there was no extravasation of blood into the uriniferous tubules, and consequently no blood appeared in the urine.

The course of a renal hemorrhage depends to a great extent upon the cause which produces it. When dependent upon the presence of a renal calculus, the hemorrhage occurs after every violent exertion. When it arises from cancer or other tumors, it is generally profuse and persistent. The bleeding which accompanies inflammation of the kidneys in the infectious diseases is never severe ; it may be so slight as only to be recognized by a microscopical examination of the urine. That form of renal hemorrhage which occurs in malarial districts in hot climates is usually profuse and occurs periodically. When it is caused by an infarction, the patient is usually seized with a chill at the time the infarction occurs, followed by pain in the back, and more or less nausea and vomiting. If, therefore, these symptoms are developed in connection with cardiac disease or pyæmia, it is evidence that renal infarctions have occurred.

When valvular disease of the heart exists with ulcerative endocarditis or extensive calcareous degeneration of the arteries, embolic infarction may be suspected, when in addition to the sudden appearance of blood and albumen in the urine there is fever and vague pains in the lumbar regions. Small abscesses, the sequelæ of infarcts and circumscribed spots of gangrene, cannot be diagnosticated.

Prognosis.—The prognosis depends upon the conditions and circumstances under which the hemorrhage occurs. If it occurs in connection with renal calculi or cancerous disease of the kidney, the prognosis is bad ; life is endangered under these circumstances by the exhaustion produced by the continued loss of blood. Occurring in connection with infectious diseases, it has no particular significance ; it merely is an indication of intense renal hyperæmia. When there is reason to believe that a hemorrhagic infarction has occurred in the kidney, the event must always be regarded as attended with danger to life ; not that it is necessarily fatal, or that the prognosis is necessarily unfavorable, but the fact that infarctions exist will cause anxiety as to the development of the other degenerative changes in the kidneys, and as to the lodging of emboli in other parts, particularly the brain.

Treatment.—The first thing to be accomplished in its treatment is to find out and, if possible, remove its cause. In many cases where the main causative disease is amenable to treatment, the hemorrhage does not require any special attention. During the occurrence of the hemorrhage, the patient should be kept absolutely at rest. If there is danger of exhaustion from repeated and profuse hemorrhages, ice-bags may be applied to the lumbar region, and styptics administered internally.

The remedial agent which seems to have the greatest control over these hemorrhages is tannic acid, it being expelled from the system through the kidneys in the form of gallic acid ; a powerful astringent is thus brought directly in contact with the uriniferous tubes and urinary passages. Ergot, muriate of iron, alum, the acetate of lead, and turpentine are sometimes of service. Ergotin given hypodermically in connection with morphia is indicated if hemorrhages are profuse. If the hemorrhage is of malarial origin, large doses of quinine and arsenic are indicated. The danger from acute renal inflammation must always be borne in mind when renal hemorrhage occurs in connection with the infectious diseases ; the proper measures for the subduing or arresting of such inflammations must be promptly resorted to.

NEPHRITIS.

(*Bright's Diseases.*)

A great variety of inflammatory changes in the kidney have been generally classed under the comprehensive name of Bright's Diseases. Dr. Bright first called the attention of the profession to this class of diseases in the year 1827, at which time he described, and represented by colored drawings, various morbid appearances of the kidneys, which he showed were of every-day occurrence and were frequently associated with general dropsy. Dr. Bright regarded granular degenerations as the principal, if not the only pathological lesion present in this class of diseases ; he accordingly designated it as a *granular nephritis.*

Recent and more extended investigations have shown that there are several morbid processes in the kidneys of those who are the subjects of this class of diseases, and that the kidneys in the course of these morbid processes present a great variety of appearances.

A great number of terms claiming to be expressive of these different morbid appearances have been employed, such as the large white kidney, the large granular kidney, the small fatty granular kidney, the large and small red granular kidney, the waxy kidney, and the cirrhotic kidney.

In studying the morbid changes which occur in this group of diseases it is important to remember that there are three distinct anatomical elements in the kidney which are primarily or secondarily involved in these changes ; namely, the uriniferous tubules, the blood-vessels, and the intertubular tissue (or stroma).

I shall describe nephritis under the following heads :

I. Acute Nephritis ; which may be—
  (*a*) Parenchymatous ; also designated glomerular, catarrhal, croupous, desquamative, etc.
  (*b*) Interstitial ; also designated diffuse interstitial nephritis, and surgical kidney.

II. Chronic Nephritis ; which may be—
  (*a*) Parenchymatous ; known as large white kidney, large and small granulo-fatty kidney.
  (*b*) Interstitial ; known as cirrhotic, gin-drinker's, or hobnail kidney.
  (*c*) Amyloid ; known as waxy or lardaceous kidney.

ACUTE NEPHRITIS.

(*Acute Bright's Disease.*)

Acute nephritis may be wholly recovered from, prove fatal, or lead to chronic nephritis.

Morbid Anatomy. — The gross and microscopical appearances of the kidneys in acute nephritis will vary according to the intensity and character of the processes which attend its development. The kidneys are usually increased in size, their capsule non-adherent, their surface smooth and mottled, presenting an irregular combination of red vascular engorgement and unnatural pallor; sometimes they are of a dark purplish color, dotted here and there with spots of ecchymosis. The stars of Verheyen are more or less prominent, and the kidneys are softer than normal.

On section, the cortical portion is relatively increased in thickness, and is dotted over its entire cut surface with dark or bright red points, which correspond to the situation of the Malpighian tufts, which in some instances stand out prominently upon its cut surface. The cortical substance between the Malpighian tufts may be of a paler color than natural. Some distinguish between a "red" and a "pale" kidney in acute nephritis. The engorgement will usually be most marked at the base of the pyramids, at the junction of the cortical and medullary substance—in the arterial arcade. The medullary portion will be of a darker color than normal, darker even than the cortical portion; sometimes it will present a striated appearance (red and white lines alternating), the lighter lines corresponding to the changed uriniferous tubes, the red lines to the congested intertubular vessels.

The lining membrane of the pelvis of the kidney is usually congested. The inflammation of the mucous membrane of the pelvis and calices is attended by exudation of a turbid fluid containing cells.

When such a kidney is examined *microscopically,* it may be found to present quite a variety of appearances. First, The epithelium of the convoluted tubes and of the Malpighian tufts may become swollen, increasedly granular, and their nuclei more or less obscured; as a result, the lumen of the tubes becomes obliterated and filled with swollen cells—the changes corresponding to those which occur in catarrhal inflammations of the mucous surfaces. The cells of the convoluted tubes of the cortex will have undergone cloudy swelling. These changes are common in the acute nephritis of fevers. There will be no cell infiltration around the tubules in the intertubular tissue.[1] Some cases are very mild. Even though albumen and casts were found during life, no marked structural changes are found in the kidney. In all probability, in these cases, changes have taken place in the capillaries of the Malpighian tuft which allow of the escape of albuminous material from the blood.

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[1] Cell accumulations about the glomeruli and tubuli contorti are said by Traube to be primary, and the epithelial changes to be secondary.

In another class of cases the epithelial cells, especially of the convoluted tubes, become filled with albuminous and fatty granules, desquamation follows, and the tubes become filled with broken-down epithelium and fatty matter. Colloid material is often seen in the tubes. The epithelia, in a few cases, undergo simple atrophy. In some cases of acute nephritis all these processes may be present at the same time in the same kidney. The centre of the uriniferous tubes will often contain a hyaline material which resembles coagulated fibrin; this hyaline material may have mingled with it, or may be surrounded by disintegrated epithelium and blood globules; it resembles fibrinous inflammatory exudation. In addition to these tubular changes, more or less cell development takes place in the intertubular structure, especially around the Malpighian tufts and the small veins.

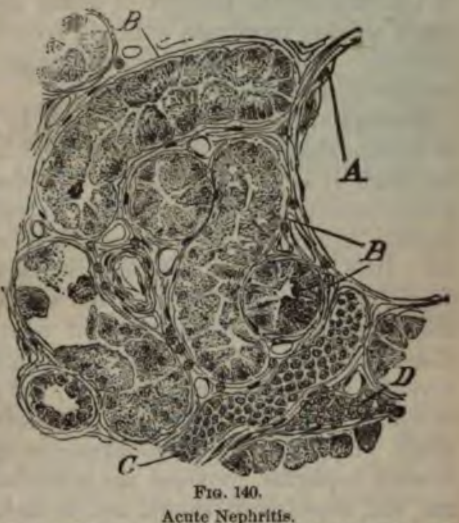

FIG. 140.

Acute Nephritis.

Section of the cortex of a Kidney showing "cloudy swelling," etc.

A. Part of a capsule of a Malpighian body.
B, B. Convoluted tubes showing the cloudy, swollen epithelium. The nuclei are obscured.
C. Ascending limb of Henle's loop.
D. Slight round-cell infiltration in intertubular tissue. × 350.

Again, in another class of cases, a typical example of which is furnished by the kidney of scarlet fever, the changes seem to be confined principally to the glomeruli. The epithelia covering the capillary tuft, and those lining the capsule of Bowman, proliferate, and the capsular space becomes filled with desquamated epithelium, often to such an extent that the capillary vessels of the tuft are compressed, and the circulation through them impeded. In the vessels of the Malpighian body, and in the capillaries between the tubes, are found an abnormal number of white corpuscles and some detached endothelial cells. The epithelia of the convoluted tubes are often swollen and filled with fat

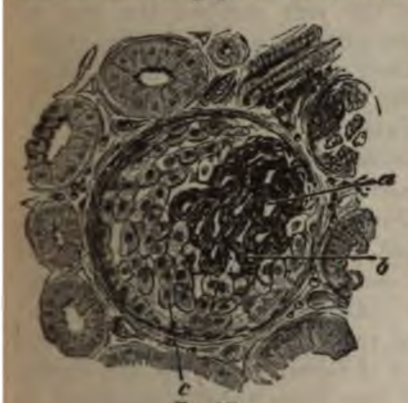

FIG. 141.

Glomerulo-nephritis, case scarlet-fever.

a, Compressed vascular tuft.
b, Increased glomerular nuclei.
c, Proliferated lining cells of glomerulus filling capsular space. × 300.

granules. Some make this a distinct form of Bright's disease and call it glomerulo-nephritis. Minor changes in and about the glomeruli are found in the majority of kidneys of acute nephritis. In most cases these processes quickly terminate either in recovery or death; in a few they become chronic. When blood extravasations are abundant, the name "hemorrhagic nephritis" has been given to it.

Etiology.—Its most common cause in the adult is exposure of the surface to sudden changes of temperature, as is indicated by the class of subjects in which it is most liable to occur—bakers, firemen, moulders, and those whose occupation subjects them to sudden, repeated changes of temperature. Again, it occurs among those who are addicted to the use of alcohol; they may not be habitual drinkers or greatly intemperate, but they occasionally "go on a spree," and while in a state of intoxication expose their surface to sudden changes of temperature or to prolonged cold after violent exercise. Under these circumstances it is not the alcohol that develops the tubular inflammation, but the sudden changes of temperature to which these persons subject themselves in consequence of such indulgence. The daily use of alcohol may be indulged in for years without leading to acute nephritis, provided the individual exercises care in regard to exposure, and therefore it should not be included in the list of its direct causes.

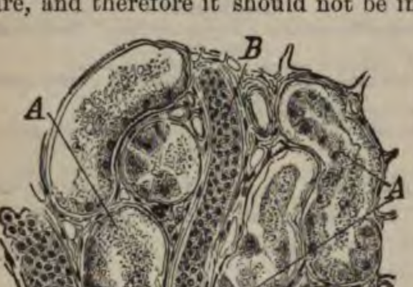

FIG. 142.

Acute Nephritis.

Section of the cortical portion of a Kidney, showing advanced degenerative changes in the parenchyma.
A, A. Convoluted tubules filled with broken-down epithelium and granulo-fatty matter.
B. Swollen epithelium of one of Henle's tubes.
C. A collecting tubule. Epithelium nearly normal. × 350.

Occasionally it happens that a very trifling exposure to sudden changes in temperature is sufficient to develop it, such as sitting in a draught of air and exposing the lightly covered loins to a current of cold air while the individual is in a heated condition. In this climate the failure to wear flannel next the body throughout the year involves the risk of developing at some time an inflammatory process in the uriniferous tubules. It is not clear how such exposure excites tubular inflammation. Runeberg states that congestion (*e.g.*, passive hyperæmia) and the consequent slowed circulation and diminished pressure in the glomeruli, is the cause of the albuminuria or tubular inflammation. The theory that the nephritic inflammation is due to the reflex influence of the nervous system—there being a connection between the sympathetic nervous system and the surface of the body—rests on the same basis which is employed to explain the occurrence of pneumonia and bronchitis after exposure to cold.

Another very common cause of acute nephritis is the circulation of morbid elements in the blood; such elements are very numerous, embracing all those poisons which give rise to specific forms of infectious diseases. The infection of scarlet fever is one of its most frequent causes, especially in childhood. Every epidemic, however, is not attended by renal complications, for there are some seasons when a type of scarlatina prevails, in which scarcely a case will have renal complications; while during other seasons almost every case will be attended by them. Such variations can only be accounted for by regarding the occurrence of nephritic complications as dependent upon a difference in the scarlatinal infection.

Another class of causes is included under the head of renal irritants which may be introduced into the stomach: among these are the balsam of copaiba, spirits of turpentine, cantharides, phosphorus, arsenic, and lead. The prolonged use of these remedies, or their administration in overdoses, not infrequently gives rise to tubular nephritis. Another cause is acute internal inflammations, especially inflammation of the lungs: one should always be on the watch for its occurrence during a pneumonia.

Another frequent cause is pregnancy. It was formerly supposed that pregnancy produced Bright's disease by interference with the renal circulation from pressure on the renal veins; but probably this is rarely its cause. During pregnancy there is an abnormal quantity of excrementitious material to be carried out of the system by the kidneys, which not only calls upon these organs for increased labor, but the material eliminated acts as an irritant on the uriniferous tubes, and tubular inflammation is the result. It may occur at any period of pregnancy, but it is rare before the third month, and is of more frequent occurrence during the later months. In connection with pregnancy, this form of kidney disease rarely terminates in chronic nephritis. It often disappears rapidly and never recurs, or it may appear in successive pregnancies, and finally lead to the development of chronic Bright's disease. Again, passive renal hyperæmia dependent upon obstruction to the return circulation from cardiac or pulmonary lesions may cause acute Bright's disease.

There is a degeneration of the epithelium of the uriniferous tubes which occurs under certain circumstances independent of inflammation. It is not amyloid; it is not, strictly speaking, a fatty change; but it occurs during the degenerative processes of old age, and is a result of senile change. In this sense, extreme old age may be regarded as a cause of tubular epithelial degeneration in the kidneys.

Symptoms.—The presence of urea and its allies, kreatin, kreatinin, etc., in the blood, in abnormal quantities, gives rise to the phenomena which attend the development of acute nephritis. The symptom which usually first attracts the attention of a patient is œdema of the face. There may have been signs of gastric disturbance prior to its occurrence, but they have not been distinctive in character. After exposure to sudden variations in temperature, or after an attack of some form of acute infectious disease, or without any known cause, there is noticed a slight puffiness about the eyes in the morning; if the patient is anæmic, the œdema may appear in the feet and ankles at the same time This œdema usually increases very rapidly. With the occurrence of the œdema there is great restlessness. Toward evening there is a little quickening of the pulse and a slight rise in temperature—never typical; the patient complains of headache, which increases in severity from hour to hour; at times he is very drowsy. Complete, sudden, but temporary blindness may occur at its very onset, the ophthalmoscope showing *no* morbid appearances.

If the patient is closely questioned, he will state that he has recently noticed some change in his urine, that it has been scanty and high colored, and he has had a frequent desire to pass it. Perhaps he has had some pain in the back and loins; he may complain of nausea and perhaps of vomiting; the latter is sometimes so troublesome that the physician may direct his attention to the stomach as the seat of the disease, and treat the patient for some form of gastric lesion. There is more or less acceleration of the pulse, which is irritable in character. The skin is usually unnaturally dry; occasionally it is moist, but when it is so the perspiration has a peculiar urinous odor. These, in brief, are the symptoms which attend the development of a mild form of acute Bright's disease.

In a favorable case, after the patient has reached the condition described, he begins to improve; the urine is increased in quantity, the œdema gradually disappears, the headache moderates, the gastric disturbances disappear, and in the course of two or three weeks he has entirely recovered.

In a certain proportion of cases, instead of improving, the patient steadily grows worse; the œdema steadily increases until the cellular tissues of the entire body become œdematous. As a result of the pulmonary œdema there is dyspnœa. Dyspnœa in this connection is not always dependent upon an œdematous condition of the lung, for uræmic dyspnœa may occur independent of any change in the lung-tissue. When there is general anasarca the dyspnœa is usually due to pulmonary œdema; it may be accompanied by more or less pulmonary congestion, giving rise to a watery expectoration, which may be streaked with blood. If the disease progresses, the anasarca will gradually increase until the patient becomes "water-logged." With the general anasarca the surface of the body assumes a peculiar, pale, waxy appearance; there is œdema of the scrotum and penis, or labia, and more or less effusion into the peritoneal, pleural, and pericardiac cavities. Hydrothorax may so impede respiration as to cause death.

As the uræmia becomes more profound, a series of nervous phenomena are developed: the patient becomes exceedingly restless, muscular twitchings occur, and these may soon be followed by convulsions, coma, and death. If this class of patients do not die from the direct toxic effect of the urea, they may have complications, such as meningitis, pericarditis, endocarditis, pneumonia, etc., which may rapidly lead to a fatal issue. This is the most unfavorable of all the types of acute parenchymatous nephritis. Such cases sometimes follow scarlet fever. The same type of cases is also met with in connection with other infectious diseases.

There is still another type of acute nephritis which is occasionally met with, called by some acute diffuse nephritis. It is ushered in by violent symptoms: the patient is seized with a chill, intense pain in the back and along the ureter, with retraction of both testicles; there is delirium, great nervous disturbance, urgent cerebral symptoms, and the patient may pass quickly into a state of coma and die within two or three days. The chill in these cases is followed by high temperature ranging from 104° F. to 106 F.; there is often almost complete suppression of urine, perhaps not more than two ounces being secreted in twenty-four hours. The delirium which is present so closely resembles that of meningitis that it is often difficult to differentiate between the two conditions. In these cases there is intense, active renal hyperæmia, and the tubules are extensively filled with an inflammatory exudation. Very soon after the accession of the ushering-in symptoms, œdema of the face will be developed, and soon after its occurrence the patient will pass into a state of coma, which is usually followed by death. If these patients recover from the acute stage, the kidneys will be permanently damaged.

Connected with the history of acute nephritis, there are symptoms which are of special importance, and which I shall consider more in detail; these are the changes in the *urine*, the *dropsy*, and the *nervous phenomena*. These are present to a greater or less degree in all cases, and their existence is necessary for its diagnosis.

The *urine* in all varieties is diminished in quantity, high colored, and sometimes smoky in appearance; it is of high specific gravity, perhaps as high as 1.030. A sediment, in which there are red and white blood-corpuscles, forms soon after the urine is voided. The amount of urea eliminated in the twenty-four hours is diminished to one-half or one-quarter the normal amount. When tested for albumen, from one-third to one-half of the entire bulk of the urine will coagulate. In testing for albumen it is well to employ both heat and nitric acid. Albuminous urine is usually coagulated by heat below the boiling point, and by nitric acid. If both of these tests are carefully used, one will rarely be led into error; but mistakes are often made when only one of these agents is employed, for the reason that heat alone will not coagulate albumen in urine which is neutral or alkaline; in such cases the addition of nitric acid coagulates and precipitates the albumen. In true albuminuria, where *serum-albumen* appears in the urine, there is some kidney change. In false albuminuria, where albumen, not serum-albumen, appears in the urine, the kidneys may be healthy.[1] Recently, opinions have changed in regard to albuminuria.[2] If a portion of the urinary sediment be *microscopically* examined, casts will be found which correspond to the contents of the uriniferous tubes already described; these casts consequently vary in appearance and composition. Those which are most characteristic are the epithelial casts, which may contain blood-globules; in very active forms of the disease, the casts may be entirely composed of coagulated blood, called *blood casts;* casts of this form and composition are found in no other form of nephritis, unless it is complicated by acute tubular inflammation. In addition to epithelial and blood casts, small and large hyaline casts may be found. The small hyaline casts are formed in tubes the epithelium of which has not been removed. In addition to the casts, free epithelial cells and blood-globules may be seen. Hyaline and epithelial casts are sometimes found independent of Bright's disease, and saccharine urine may be loaded with them.[1] Distinctly formed cell elements in a cast point to an origin in the straight or collecting tubes.

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[1] See text-books on "Urinary Analysis" for modes of determining the different albumens; also Appendix to Foster's "Physiology."

[2] Albuminuria itself is, according to Gull's statements, as common in young men and boys as spermatorrhœa; Moxon confirms this. Young girls from fourteen to seventeen have it. Depressing mental emotions cause a lowered pressure in the vessels, and this, according to Runeberg's ingenious theory, is the one cause of albuminuria. Leube and Furbruger incline to the opinion of an individual permeability of membrane. Temporary nervous innervation may in some instances induce transient albuminuria, with or without healthy kidneys. Drs. Brunton and Power (St. Barthol. Hosp. Rep., 1877) take issue with Bartel's statement that albuminuria is always of renal origin. There are different albumens, some derived from the blood, others from the digestive organs. Diminution and increase of blood-pressure in the glomeruli have both been advanced as prime causes of the albuminuria. Probably blood-pressure plays but an unimportant part. Cohnheim regards changes in the epithelium covering the glomeruli as an important factor. These changes are, no doubt, in part produced by the stagnant inflammatory current. The vessels of the glomeruli unquestionably allow most of the albumen (in acute albuminuria) to exude.

Dropsy occurs early; there have been several theories advanced as to its cause, but none are perfectly satisfactory. One theory is, that it is due to the sudden removal from the body of a large amount of albumen; whereas in the most rapidly developed dropsies no albumen is carried out of the body, for the reason that the patient passes little or no urine.[2] Another cause assigned for the dropsy is, that the kidneys fail to eliminate the watery portion of the blood in the form of urine, and that the dropsy occurs as the result of the retention of the watery elements; yet very extensive dropsies occur while the patient is passing more than the normal quantity of urine.[3] Again, it is said that dropsy occurs in consequence of the anæmic condition of the patient. The anæmic condition undoubtedly contributes to the ease with which the transudation of fluid through the walls of the vessels takes place; but a patient may be exceedingly anæmic and yet no dropsy be present, and dropsy very often occurs before the patient shows any evidence that he is in an anæmic condition.

I regard dropsy as a necessary symptom of acute nephritis, but the exact cause of its occurrence in many cases cannot be satisfactorily determined.

Nervous symptoms are of great importance and prominence. Undoubtedly these are due to the presence of some irritating poison in the circulation, which acts directly upon the nerve centres.[4] Usually the nervous symptoms first manifest themselves by headache; therefore headache is a symptom which must not be lightly regarded, for it is often the precursor of more dangerous symptoms. If persistent and severe, and permitted to pass unrelieved, it may be followed by convulsions. The larger proportion of cases of acute nephritis will suffer from more or less severe headache, without any subsequent convulsions; but the fact that convulsions do follow it is sufficient to cause one to watch for the indications of their occurrence. If the poisoning goes on gradually, the patient will first become drowsy, the drowsiness passing into stupor, and frequently into coma. A large number of patients with acute nephritis unquestionably die from the direct effect of urea and its allies upon the nervous centres; but a still larger number die from complications.

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[1] Southey regards it as an error to suppose that the larger casts are derived from the larger tortuous tubes. Nothing but cellular elements can pass through Henle's loops (diameter 1-1200 to 1-1000 in.); "when," he says, "a cast is assumed to come from the profounder renal tissue and to be of grave significance, an error is committed, based on ignorance of anatomy." Hyaline casts probably form in Henle's loops.

[2] Cohnheim, in his work on pathology, regards inflammatory changes in the walls of the cutaneous and subcutaneous vessels—whose causes are the same as those of acute Bright's—to be the reason of anasarca in many instances,—the vessels being rendered more permeable.

[3] Cohnheim (*Virchow's Archiv.* 1877, 96, p. 106), after most elaborate experimentation, regards œdema as the result, *not* of dilution of the blood or of increase in the relative amount of water, but of *hydræmic plethora, i. e.*, increase in the absolute amount of water. This fact, with changes in the walls of the vessels, is accepted by most authorities as the most plausible and probable cause of œdema in acute Bright's.

[4] The different theories in regard to the causation I have considered under the head of *acute uræmia.*

Differential Diagnosis.—If a patient has headache, some fever, more or less œdema, nausea, and perhaps vomiting, with scanty, high-colored urine of high specific gravity, containing epithelial, small hyaline, or blood casts, it is certain that acute renal disease exists. There may be other pathological conditions existing in the kidneys at the same time, but this train of phenomena gives unmistakable evidence that some of the uriniferous tubes are the seat of acute inflammation; the acute may be engrafted upon the chronic. In every case which presents this train of symptoms, frequent examinations of the urine should be made. The general symptoms and the changes in the urine in acute nephritis are so obvious that it can scarcely be overlooked or mistaken for any other disease. The only circumstances under which it is possible for this affection to pass unrecognized are those in which dropsy is not a prominent symptom, and when a careful examination of the urine has not been made.

It is not always easy to determine whether acute nephritis is *primary* or *secondary*—that is, whether it has occurred in kidneys that were healthy previous to its occurrence, or in those that were already the seat of chronic disease. The previous history of the patient, and the presence or absence of cardiac hypertrophy, are the only means to guide one under such circumstances.

The points of differential diagnosis between *congestion of the kidneys* and acute Bright's disease have already been given.

Acute nephritis is distinguished from paroxysmal *hæmaturia* and *albuminuria* by the abrupt commencement and brief duration of these affections, by the marked nervous and gastric symptoms, the slight jaundice, and the absence of dropsy. Granular pigment in hæmaturia, and a very great quantity of albumen and tube casts in paroxysmal albuminuria, are characteristic urinary symptoms. Hæmaturia with a tendency to suppression has few tube casts.

Prognosis.—The tendency of acute nephritis is to recovery, but the chances of recovery are much better in the young than in those past middle life. In those cases which terminate in recovery the characteristic symptoms of the disease disappear within two or three months from the commencement of the attack. So long as albumen continues in the urine, however small in quantity, recovery cannot be regarded as complete. The indications of a fatal termination are very scanty urine, frequent and distressing vomiting, extensive anasarca, severe and persistent headache, convulsions, coma, typhoid symptoms, and the occurrence of complications.

The pulmonary complications which render the prognosis unfavorable are œdema, pneumonia, and capillary bronchitis. The great danger in pneumonia which complicates acute Bright's disease is the sudden development of pulmonary œdema in portions of the lung not involved by the pneumonia. Another dangerous complication is inflammation of the serous surfaces, especially endocarditis and pericarditis. Acute meningitis is a rare but always a fatal complication. There may be complete loss of sight; this form of amaurosis is usually temporary, and is unattended by any change in the retina recognizable by the ophthalmoscope; it is probably due to the direct effect of the urea upon the retina. Subacute gastritis, functional hepatic derangement, and œdema glottidis are also complicating conditions which render the prognosis unfavorable.

In a small portion of cases patients pass rapidly from acute into chronic nephritis. The passage from acute into chronic is indicated by a copious secretion of paler urine containing few casts. The individual may be able to resume his ordinary avocations, but the œdema of the feet and ankles does not entirely disappear, and the urine remains albuminous.

Treatment.—Formerly, general and local blood-letting was practised in the treatment of all acute renal diseases. At the present time general blood-letting is never resorted to, unless in the very acute form which is attended by violent cerebral symptoms.

This was followed by the diaphoretic and cathartic plan of treatment in which it was proposed to supplant the function of the kidney by increasing the functions of the skin and bowels, and thus afford the kidney comparative rest. Under this plan of treatment the patient is given two or three hot-air baths each day, and hydragogue cathartics to produce three or four watery discharges from the bowels in twenty-four hours. Such treatment will decrease the dropsy, and for a time this class of patients will appear very much relieved; but after a few active purgations, and a few hot-air baths, they will begin to complain of extreme weakness, and very soon reach a point at which the combined action of these agents fails even to relieve the distressing symptoms, and their condition is then worse than before their administration was commenced.

Several years ago I became convinced that this depurative plan of treatment was wrong, because it rapidly depleted patients who could not bear depletion. Exhaustion can as certainly be produced by diaphoresis and hydragogue cathartics as by repeated general bleedings. Besides, the repeated use of hydragogue cathartics interferes with the processes of digestion and assimilation.

In the treatment of acute nephritis, there are three important things to be accomplished. *First:* the elimination of urea and its allies. *Second:* the removal, as rapidly as possible, of the inflammatory products which obstruct the uriniferous tubules. *Third:* to counteract the effect of urea and waste products upon the nervous system. Although no organ can assume the renal function, and any attempt to secure such vicarious action of the skin and bowels as will fully relieve the kidney, has been proven dangerous to the patient, nevertheless, something may be done to render the solid elements of the urinary secretion less irritating to the organ. The hepatic function is most closely connected with that of the kidney, and stimulation of the liver will be found to afford most decided relief to the kidneys. For this purpose some preparation of mercury is to be preferred. It should not be given in such doses as to induce profuse catharsis, but in small amounts frequently repeated, and sufficient to maintain a free action of the bowels. While I do not accept the extreme views of those who believa uræmia to be an intestinal absorptive disease, I am convinced that *cholagogue* cathartics used in moderation are of decided value in renal disease, but such catharsis must be carefully distinguished, both as to its objects and results, from the use of *hydragogue* cathartics, which are intended simply to reduce dropsy and remove such poisonous elements as may be held in solution by the dropsical fluids.

Mercury thus employed is also a direct stimulant to the parenchymatous action of the kidney necessary for the removal of the urinary poisons, and at the same time becomes a factor in accomplishing the second indication, the removal of the inflammatory products in the uriniferous tubes. These products not only interfere with the elimination of the urinary solids, but if they remain in the tubes they induce degenerative processes.

In connection with the increase of parenchymatous function, an augmentation of the watery elements in the renal secretion tends to accomplish the desired result. Digitalis is the most valuable drug for this purpose. Its action must be watched, however, and if the amount of urine is not increased its use is to be discontinued. In most cases it increases the urinary secretion without stimulating the kidneys; it overcomes the obstruction in the renal circulation, and thus causes an increased flow of the watery portion of the urine through the Malpighian tufts into the upper portion of the uriniferous tubules. Thus the obstruction in the tubes is washed out, and at the same time the eliminative function of the kidneys is increased, so that the urea is carried out of the system much more rapidly and completely than it can be by the skin or bowels. If diluent drinks are given, *water is the best*. Spirits of nitrous ether, acetate of potash, tincture of the perchloride of iron, or squills, may often be advantageously combined with digitalis.

In connection with the administration of digitalis I would recommend the application of dry cups over the region of the kidneys. In order that the dry cupping may be more effective, each cup should be removed as soon as the vessels beneath are well filled. The object is to draw the blood from the arteries into the capillaries, but not with sufficient force to cause extravasation. After dry cupping, warm poultices over the kidneys may be applied with benefit; digitalis leaves may be used for a poultice, and thus applied they will increase the diuretic effect of the drug administered internally. After the free administration of digitalis and the application of dry cups, if the uræmic symptoms are still urgent, hot-air baths and hydragogue cathartics may temporarily be resorted to, to aid in carrying the patient over the period of greatest danger; but their use should not be continued after free diuresis is established.

The next object to be accomplished is to relieve the nervous symptoms: the means to be employed to accomplish this are the same as in the treatment of acute uræmia. For the successful management of acute Bright's disease, whatever may have been its exciting cause, the patient must be kept in bed, in a large, well-ventilated apartment with a temperature of 75° F. Milk should be the only article of diet. Skimmed milk is advocated highly; besides being nourishing, it is a good diuretic. If this plan is systematically carried out from the very commencement, the urine soon becomes copious, the albumen gradually diminishes, and the dropsy passes away. As soon as the flow of urine commences, the administration of digitalis must be discontinued, and diluent drinks are to be given. If the renal secretion be not re-established in twenty-four hours, hot-air baths, hydragogue cathartics, or pilocarpin hypodermically in one-eighth or one-tenth grain doses, are to be used.

The usual method of producing profuse diaphoresis is to place the patient in bed and cover him with flannel blankets, and then by means of the hot-air apparatus introduce a constant current of hot air beneath the bedclothes, until profuse perspiration is induced and the excretory power of the skin is taxed to its utmost. The bath should be continued from half an hour to an hour; then the patient should be allowed to gradually become cool, and, when so, to resume his clothing and walk about the room or ward, the temperature of which should be above 70° F. These baths may be repeated once or twice each day, or every other day, as the condition of the patient may demand. The effect usually produced by these baths is a rapid subsidence of the œdema. It may not require more than half a dozen baths to entirely remove the dropsy from a "water-logged" patient, and, as far as that one symptom is concerned, to give complete relief; but the relief is only temporary.

ACUTE SUPPURATIVE INTERSTITIAL NEPHRITIS.

(Surgical Kidney.)

Morbid Anatomy.—The kidney is intensely hyperæmic, softer than normal, and the fat about it is œdematous. When the thickened and opaque capsule is stripped off, pus often flows from beneath it. The surface shows arborescent injection.

On section several purulent foci are seen in the cortex and pyramids, about the size of a pea; these, coalescing, may form a large abscess. When pyæmia is its cause, the abscesses are wedge-shaped, and colonies of bacteria are found surrounding the shreddy necrotic tissue, and in the centre of the suppurating mass the epithelium is cloudy and desquamated.

Cell-infiltration takes place in the adjacent connective-tissue, and secondary thrombi are found in the small *veins*. Micrococci are found in the arterioles.[1] When the abscesses are wedge-shaped they are called "*metastatic*; but when circular they are merely spots of "*suppuration in foci*."

In *chronic suppurative nephritis,* decomposing pus, calcareous salts, and a serous, fetid fluid are contained in a sac whose wall is connective-tissue. With these (so-called) chronic abscesses, cysts and renal atrophy are present. In large pyæmic or non-pyæmic abscess, ulceration may take place at the tips of the pyramids, and the abscess may open into the pelvis, the intestine, externally, or into the peritoneum. The liver has been involved from the breaking of a renal abscess into its softened parenchyma. (Rayer.)

Diffuse purulent infiltration[2] is of rare occurrence; then the whole kidney seems to be a mass of pus; the surface and cut section are homogeneous-looking. Pus is readily scraped off, and ecchymoses are seen studding its surface.[3]

Etiology.—Any of the causes of pyelitis may be, secondarily, causes of surgical kidney. Pyæmia, ulcerative endocarditis, typhoid fever, and puerperal fever may be complicated by it. Wounds, blows, and severe contusions cause it. Reflex irritation and some as yet unknown nervous conditions are supposed by many to be the cause. Certain spinal diseases are attended by it, perhaps from disturbance of "trophic influences."

Symptoms.—There is lumbar pain, tenderness on pressure over the kidney, recurring chills, fever, languor, anorexia, emaciation, perhaps diarrhœa, nausea, and vomiting; the mouth and skin become clammy, sordes may collect on the teeth, the breath becomes offensive, and there is drowsiness which rarely passes into coma. These symptoms (especially the chills and febrile movement) are often severe, and then the disease is of short duration. The patient passes rapidly into a state of stupor without convulsions and with a subnormal temperature. The *urine* may be in excess of the normal quantity or be scanty; albumen is present in varying quantities; hyaline and pus casts and renal epithelia are also present in varying amounts. Blood is *always* found in the acute cases. The specific gravity is never very high. The urine is in many cases ammoniacal. Should a tumor be felt, it will fluctuate; but rarely is there a distinct tumor.

Differential Diagnosis.—From *pyæmia* it is distinguished by the absence of recurring chills *and sweats*; by its lower temperature; by absence of joint and lung symptoms, and by the purulent bloody urine. It is often difficult to distinguish it from *septicæmia.*

A *perinephritic* abscess is distinguished from suppurative nephritis by its *tumor*, and by the fact that in uncomplicated perinephritis urinary symptoms are absent.

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[1] This is Kleb's *parasitic nephritis*: the infecting particles or spores ascend (presumably from the bladder) to the pelvis; thence up the tubules. Hyaline casts in "parasitic nephritis" have spores and algæ on their periphery. Cornil and Ranvier think they may be formed in the kidney during life.

[2] Full descriptions are given in Erichsen's Surgery, p. 712 *et seq.*, vol. ii.

[3] Marcus Beck (in "Quain's Dict. of Med.," pp. 1562-5) describes acute interstitial nephritis *without* suppuration as one variety of the surgical kidney: infiltration of small round cells in the intertubular structure and *about the Malpighian tufts* being the chief pathological event. An acute or sub-acute interstitial (non-suppurative) nephritis I have already described.

Pyelitis has the characteristic angular "tailed" cells from the mucous membrane of the pelvis in the urine, and the constitutional symptoms are insignificant compared with those of suppurative nephritis.

Prognosis.—The prognosis is always grave. The free discharge of a large abscess may prolong life, and, if unilateral, be followed by recovery. Death from complications is its frequent termination. In the aged it is almost necessarily fatal. Asthenia, uræmia, and complications cause death.

Treatment.—The treatment is for the most part surgical. Tonics, stimulants, and condensed nutriment are indicated from its onset. A pure milk diet is advantageous. Dry cupping, fomentations, and poultices or leeching over the loins are of service. The bladder is to be washed out with any of the antiseptic fluids usually employed for that purpose, two or three times a day. Benzoic acid or benzoate of ammonia may be given to relieve the offensiveness of the urine.

CHRONIC PARENCHYMATOUS NEPHRITIS.

As already stated, *chronic parenchymatous nephritis* may be a sequela of the acute form, but it is oftener a chronic process from its onset. Under this head may be included *large granular fatty kidney*, the *large white kidney* and the *small granular fatty kidney*.

Morbid Anatomy.—Chronic parenchymatous nephritis may develop with any form of renal lesion in which there is a protracted interference with the normal nutrition of the tubes. In the *large granular fatty kidney*, but few of the epithelial cells of the uriniferous tubes at first undergo change; but, as the transformation becomes general, the affected tubes become loaded with exfoliated epithelium in all stages of the process, from the earlier condition of cloudy swelling, to the final development of granulo-fatty metamorphosis. The mucous membrane of the pelvis and calices is thickened, opaque, and anæmic, or it presents a varicose dilatation of its veins. The kidneys are enlarged, their capsules are non-adherent, and their surface smooth; their color is paler than normal, presenting a more or less yellow appearance; sometimes they are mottled with red and white.

On section, the enlargement of the organ will be found to be due chiefly to an increase in the size of its cortical substance, which is of a pale yellowish color. There is but little change in the medullary portion. The Malpighian tufts do not stand out prominently, for there is more or less fatty material in the dilated portion of the uriniferous tubes around the Malpighian tufts, which gives them a somewhat pale appearance. The vascularity of the whole kidney seems to be very much diminished; but here and there spots of hemorrhage or congestion are seen. The principal changes take place in the convoluted tubes of the cortical portion, especially in those which surround the Malpighian tufts.

These sections of the cortical substance in this form of degeneration are very opaque; under a low power they show little more than uriniferous tubules irregularly distended with fatty granules, and varicose. To the unaided eye the tubules look like streaks of sebaceous matter. At some points they are greatly increased in size, at others they are of normal calibre. In the Malpighian bodies are found oil granules in varying quantities, but the capillaries of the tuft are unchanged. Under a high power, fine fat granules are seen about the nuclei in the protoplasm of the epithelial cells, and *also in the cells of the external coat of the small vessels.* Granulo-fatty material covers a homogeneous vitreous substance in Henle's tubules. The lacunæ and cells of the intertubular connective-tissue are also filled with fine fat granules.

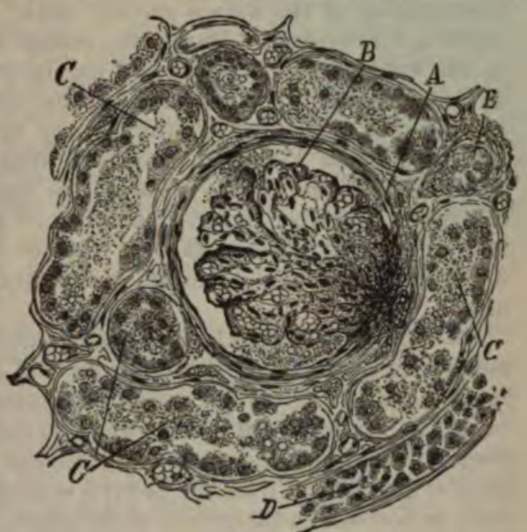

FIG. 143.

Chronic Parenchymatous Nephritis.

Section from the Cortex of a Kidney.

A. Slightly thickened capsule of the glomerulus.
B. Vascular tuft, nearly normal. A small amount of granular matter is seen beneath the capsule.
C, C, C. Convoluted tubules—epithelium nearly destroyed. Some of the tubes are entirely filled with fatty granules. The nuclei of the epithelia are yet plainly seen.
D. Longitudinal section of Henle's looped tube—ascending portion.
E. A small artery. × 350.

Large White Kidney.—In this variety of parenchymatous nephritis, the kidneys may be twice their normal size, of an "ivory-white" color, their surface smooth, and their capsule non-adherent.

On section, the enlargement is found to be due to an increase in the volume of the cortical substance. The medullary portion shows no appreciable alteration. The microscope will show that the morbid changes are confined almost exclusively to the epithelium of the convoluted tubules and that lining the Malpighian bodies. The epithelium is granular, and so much swollen that the lumen of the tubes is obstructed and may be distended with a hyaline material. There is a dilated and varicose condition of the tubes, with some thickening of their walls. In some cases Henle's loops present alterations similar to those that occur in the convoluted tubes.

Small Granular Fatty Kidney.—The atrophic alterations in the kidney in this variety (or stage) of parenchymatous nephritis are entirely different from those of atrophy produced by interstitial nephritis. The epithelium which may have been the seat of fatty or granular change, disintegrates, liquefies, and is absorbed or passes off in the urine. The tubes, deprived of their epithelium, collapse. Some claim that renal atrophy and granular degeneration of the kidney are the *same*. That these processes are associated is very evident. During the process of atrophy, developments occur in the walls of the tubes and in the intertubular tissue, which lead to, or are followed by, thickening of the tubules and blood-vessels. The processes of inflammatory atrophy are always slowly progressive. An atrophied white kidney is markedly diminished in size, its surface is uneven and more or less nodular; its capsule is adherent and slightly thickened, and when removed portions of kidney tissue may be removed with it; the denuded surface is more or less granular, its color varies, it may be white, have a stellate vascularity, or present a mottled appearance.

On section, it will be found that the diminution in the size of the kidney is mainly due to atrophy of its cortical substance; the medullary portion retains very nearly its normal dimensions; the cortical substance between the pyramids will be somewhat atrophied. The kidney is firm and tough. The granulations on its surface and in its substance are the pyramids of Ferrein. Under low power a section of the cortical substance will show an increase in the stroma of the organ, the walls of the vessels will be thickened and the Malpighian tufts will have lost their distinctness. The uriniferous tubules will be denuded of their epithelium, in some places filled with granular or fatty material, and distended; in others they will be entirely obliterated, atrophied, and more or less shrivelled. This form of kidney degeneration may be distinguished from the contracted kidney of interstitial nephritis by the larger size of the organ, its less firm consistency, its more uneven surface, its pale yellow and large granulations not only on its surface but throughout its substance, evidently formed by the accumulation of fat in the tubules, and by the absence of cysts either on its surface or in its substance. It is not necessary that the small atrophic kidney of chronic parenchymatous nephritis should have been preceded by an enlarged fatty or granular kidney.

Etiology.—Chronic parenchymatous nephritis may be the sequela of acute. It is more common in males than in females, it occurs in early adult life rather than past middle life. Exposure, moderate alcoholism, bad hygiene, phthisis, diabetes, arthritis deformans, emphysema, and chronic cardiac diseases predispose to its development. The cause is sometimes undiscoverable.

Symptoms.—This form of chronic Bright's disease may be ushered in by acute symptoms or come on insidiously; in either case, when once fully developed, the symptoms are identical. There are two symptoms which are always present, viz.: albuminuria and dropsy. If its advent is marked by acute symptoms, its development is attended by the phenomena of acute Bright's; the patient rapidly reaches a condition of general anasarca; his countenance assumes a pallid appearance; the pallor is not like the clear pallor of phthisis, nor the dingy pallor of cancer, but is peculiar to the disease. When he has reached an apparently hopeless condition his urine becomes gradually increased in quantity. His appetite returns, nausea disappears, he suffers less from restlessness, the anasarca gradually diminishes, the sleep becomes refreshing—in short, there is a gradual improvement in all his symptoms. The improvement may be continued or relapses may occur, but after a few weeks or perhaps months he may reach a condition of comparative health; this class of patients never so far recover that no traces of the disease remain. There will always be some œdema along the line of the tibia and over the internal malleolus, and the albumen will never entirely disappear from the urine. Patients in such a condition are always inspired with the hope that they will reach complete recovery; but they are liable at any time to a sudden return of their dropsy.

When the disease comes on gradually without any acute symptoms one of its earliest indications is increased frequency of micturition; the œdema may not be very extensive, but it is always present; perhaps there is at no time pain in the back or loins; but there is a time, early in the history of the disease, when the urine is scanty and high colored; afterward it becomes copious, of a pale color and low specific gravity. The gastric and nervous symptoms, so prominent in acute Bright's, are never severe; there is gradual loss of energy with progressive emaciation; the skin becomes dry and harsh, the surface assumes a peculiar pale, sallow appearance, there is often great thirst, very troublesome dyspeptic symptoms, and often marked signs of anæmia. The pulse becomes feeble and irregular in force, and the patient grows old rapidly.

The urine after a time becomes more abundant than normal, of low specific gravity, sometimes as low as 1.010, and the quantity of albumen is increased. Fatty and hyaline casts are present; when the stage of atrophy is reached the urine sometimes becomes very abundant, and, although the albumen at times may be small in quantity, it never entirely disappears, and large hyaline and fine granular casts are always present. As the elimination of urea is steadily diminished, it is important to subject the urine to frequent quantitative analyses. Cardiac hypertrophy develops, and albuminous retinitis is of frequent occurrence. Degeneration of the cardiac walls and dilatation of its cavities develop secondarily to, or coincidently with, the hypertrophy.

It is to be remembered that the symptoms and course of this form of Bright's disease are not continuous; there will be remissions, periods when these patients seem to be recovering, and suddenly the urgent symptoms of chronic anæmia will develop, and the patient passes into a state of listlessness, stupor, or coma, and death rapidly ensues.

Differential Diagnosis.—When the urine is abundant, of a pale color, low specific gravity, highly albuminous, and contains fatty, granular and hyaline casts, accompanied by œdema of the lower extremities, one readily makes a diagnosis of chronic parenchymatous nephritis, especially if a careful analysis of the history of the patient corresponds to the usual course of its developments. A state of uræmic stupor, with a dry tongue and sordes on the teeth, may be mistaken for *typhoid fever*, yet the history of the case, and a careful examination of the urine will soon remove all doubts.

If the urine is carefully examined it is hardly possible for one to confound the *anæmia* and cachexia which sometimes attend the stage of atrophy of chronic parenchymatous nephritis with the cachexia of other *chronic diseases.* The mistakes that are made in diagnosis, or rather the failures to recognize its existence, are usually due to the fact that a careful examination of the urine has not been made. In every case of persistent dyspepsia careful examination of the urine should be made.

Prognosis.—One of the most constant attendants of the advanced stage of this form of Bright's disease is the development of cardiac hypertrophy. It is probably due to interference with the systemic capillary circulation, and it is an evidence that the renal disease has existed for a long time; it suggests the possible occurrence of cerebral hemorrhage, and therefore renders the prognosis unfavorable; visceral inflammations, especially pneumonia and bronchitis, are liable to occur, and often are the direct causes of death.

The most frequent serous inflammations in this connection are pleurisy, pericarditis, and meningitis. They are usually insidious in their development, and always render the prognosis unfavorable. Another complication which may render the prognosis unfavorable is subacute inflammation of the mucous membrane of the stomach. Patients never entirely recover from the structural changes which occur under such circumstances. Amaurosis is first indicated by the patient's inability to see distinctly; subsequently he has more or less difficulty in reading print which formerly he had read with ease; lenses do not improve his vision; after a time the sight may be entirely lost. This amaurosis is due to a neuro-retinitis; it is present to a greater or less degree in a large number of these patients. The structural changes in the kidneys in the advanced stage of this form of Bright's disease are such that they do not admit of repair. All portions of the kidney, however, are not equally involved; consequently the depurative function of the organ is not suspended, but only imperfectly carried on. So long as the degenerative process is not progressive this class of patients may get on quite comfortably, but its tendency is to progress until it reaches a point beyond which life cannot be sustained. In a large number of cases, long before this limit is reached, some one of the complications to which reference has been made will cause death.

In the advanced stage, the most trustworthy prognostic indications are to be obtained by comparing the evidences furnished by examinations of the urine with the general symptoms; one must always be cautious in giving a prognosis, for the uræmic symptoms may suddenly be greatly aggravated by exposure to cold or errors in diet, and the patient quickly passes from a condition of comparative good health into uræmic coma. Although in all advanced cases the prognosis is unfavorable, still there is reason to hope that by judicious management, even in the most unpromising, relief may be obtained from many of the more distressing symptoms, and life be prolonged.

Treatment.—At one time mercurials were extensively employed in the treatment of this form of Bright's disease, with the idea of keeping up their constitutional effects for months. This plan is now abandoned. It is important that the diuretic plan of treatment should be continued when a patient passes from acute into chronic parenchymatous nephritis. Digitalis in moderate doses, or at intervals, is always indicated; it is important that the accumulations in the uriniferous tubules should be removed the same as in the acute stage. When the eliminating function of the kidney is decreased, as indicated by low specific gravity with abundant flow, or when digitalis alone fails to increase the quantity when the urine is scanty, then calomel and squills should be combined with digitalis.

There is another element which enters into the treatment. The most important thing to be accomplished in the treatment of this form of Bright's is the establishment of healthy nutrition; the nutrition of the kidneys is always imperfectly performed, and these patients are always more or less anæmic. For this reason it is important that the nutritive processes be carried to their highest point; that after the degenerated material is removed from the uriniferous tubes, the degenerative inflammatory processes may be arrested and the epithelial lining of the tubes restored. Digitalis combined with iron should be given in sufficient quantity to produce moderate diuresis. In most instances milk is the best article of diet. Adults will often take two or three quarts in twenty-four hours; when taken freely it supplies an abundance of liquid, which acts to some extent as a diuretic. In most cases a moderate amount of stimulants will be of service. Wines are to be preferred, and they should be taken with the food.

The patient must be placed under the best hygienic conditions, in a uniform temperature, and the surface of the body must be covered with flannel; over-indulgence of every kind, and exposure of the surface to cold must be carefully avoided; a residence in a uniformly dry climate is of the utmost importance. The urinary secretion must be carefully watched both as to its quantity and quality. In the stage of atrophy there will be no necessity for the administration of diuretics, for the urinary secretion is abundant. The disease is attended by great feebleness, and on account of their feeble digestive power this class of patients will be compelled to take food in small quantities and at short intervals; they will generally be greatly benefited by cod-liver oil, combined with iron. Wines are always indicated in moderation. Whenever the urine becomes scanty, two or three full doses of digitalis should be administered and dry cups applied over the kidneys.

The urgent symptoms, such as dropsy, etc., must be relieved by an occasional hot-air bath, hydragogue cathartics or stimulating diuretics, and at the same time great care must be exercised lest the depletion be carried too far. Jaborandi or the hydrochlorate of pilocarpin may be cautiously used in very urgent cases; they are prompt and efficacious, but sometimes dangerous. Iron and cod-liver oil are the two great remedial agents in this disease, and should be daily administered, unless the condition of the stomach of the patient shall contra-indicate their use. Milk should be the principal article of diet. By living in a warm climate, by constant watchfulness, and by following the rules given in acute Bright's disease, a fatal termination may be long delayed, although complete recovery cannot be hoped for.

Let me impress this fact: that no depleting remedies should be employed, except in times of emergency, when, from some sudden renal congestion, the function of that portion of the kidney structure which is still performing the work of elimination becomes suddenly arrested or impaired, and acute uræmic symptoms are developed.

CHRONIC INTERSTITIAL NEPHRITIS.

(Cirrhotic Kidney.)

In this form of nephritis the morbid processes do not pass through distinct stages. The changes consist essentially in an increase of the intertubular structure, and a consequent atrophy of all the other structures. As has been stated, it has been called the gouty, hob-nailed, or small red kidney.

Morbid Anatomy.—Kidneys that are the seat of interstitial nephritis are at no time very much increased in size. The changes are characterized by a gradual increase in the connective-tissue of the kidneys and by atrophy of the tubules. In its early stage the capsule is somewhat adherent, the surface uneven, and the stroma of the cortical substance somewhat increased.

In the advanced stage of the process the kidneys are diminished in size, sometimes to one-fourth their normal bulk; their capsule becomes thickened and very adherent; the thickening of the capsule is quite characteristic, and there is more or less prolongation of the connective-tissue from the capsule into the cortical substance, in consequence of which a portion of the kidney structure will be removed when the capsule is torn off, leaving the surface of the organ uneven and ragged, having sometimes a finely granular appearance and of a reddish color. Such kidneys have a dense fibrous feel, and dilated veins are sometimes seen upon their surface.

On section it is found that the diminution in the size is due to decrease in the cortical substance. It is more markedly diminished in this than in any other form of Bright's disease; it will also be noticed that the blood-vessels are more distinctly visible than in the normal kidney. The Malpighian tufts, however, are not as prominent; the medullary portion retains very nearly its normal appearance and is not markedly diminished in size.

The principal change, so far as retraction is concerned, takes place in the cortical portion. This portion may be reduced to one-sixth its normal thickness. The shrinking is not only apparent in the cortical substance beyond the bases of the pyramids, but also in the tissue between the pyramids. Cysts are usually found in the cortical portion, especially near its surface. These cysts are of varying size, and may be the result of a variety of changes.

The changes which occur are usually as follows :—

First, there is cellular infiltration of the intertubular connective-tissue of the cortical substance, most abundant around the capsule of the Malpighian tufts; this gradually develops into a fibrillated structure; in this stage of the process the tubes and their epithelium are but slightly, if at all, implicated. The Malpighian tufts are diminished in size, their capsule thickened, and around the tufts are laminated, concentric zones of connective-tissue, between whose lamellæ are flat, stellate, or small round cells. The intertubular growth, by its pressure and contraction, causes atrophy of the tubes, which in some places are obliterated, in others irregularly distended, and they contain degenerated epithelial products; as the atrophy proceeds the intertubular tissue becomes filled with granular and fatty débris. The walls of the small arteries[1] become thickened by hypertrophy of all their coats, especially the middle, but they have an irregular outline. The firm, dense mass of connective-tissue between the Malpighian tufts completely obliterates the expanded uriniferous tubules, bringing the tufts much nearer to each other than in the normal kidney, but it does not as a rule obliterate them. The shrinking and even total disappearance of the convoluted tubes near the tufts, cause the tufts to almost touch one another. The Malpighian tufts are sometimes obliterated, but their obliteration is usually due to the development of cysts. The cysts are often "colloid cysts."[2]

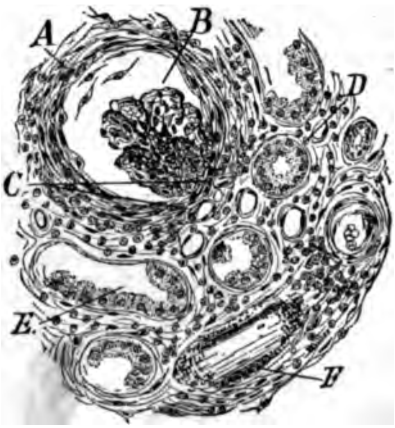

FIG. 144.

Chronic Interstitial Nephritis. Early.
Section from the Cortex of a Kidney in Cirrhosis.

A. Capsule of a Malpighian body thickened with concentric layers of connective-tissue, containing flat and round cells.
B. Vascular tuft of the glomerulus diminished in size.
C. Afferent and efferent vessels of tuft.
D and E. Convoluted tubes in transverse and longitudinal section—Epithelium nearly normal.
F. Small artery in longitudinal section. × 350.

Sometimes connective-tissue formations extend into the medullary portion, and more or less shrinking of the pyramids occurs as a result. It is usually, however, confined to the cortical portion. Those tubes that retain their normal diameter are filled with fatty, granular, or colloid cells; and their lumen contains hyaline or colloid casts. Blood pigment often stains the cells of the tubules. The tubules in an uncomplicated cirrhotic kidney contain coagulated fibrin, which will be indicated by the presence of hyaline casts in the urine : all the tubular changes are secondary.

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[1] Johnson regards induration of the arterial walls as due to an hypertrophy of the muscular coat: Gull and Sutton regard it as a deposit of a hyalin-fibroid, or hyaline-granular mass infiltrating the walls of the arterioles and capillaries. Cornil and Ranvier say it is neither: it is but a chronic arteritis to them, both intima and adventitia being involved, *i. e.*, endarteritis and periarteritis.

[2] Concerning colloid casts, Cornil and Ranvier state that "after inflammatory destruction of the normal cells of the convoluted tubules there are developed cells—not having the character of secreting cells—but assuming the cubical or flat form; these cells undergo colloid transformation and fuse into a colloid mass, which is increased by the deposit of successive layers, while at the same time new cells at the periphery become colloid."

The pelvis and calices are congested ; the submucous tissue is dense and thickened ; sometimes the pelvis and calices are dilated. In the advanced stage of this form of kidney degeneration the organs are very greatly diminished in size—their capsules exceedingly thickened, their surface finely granular, and the vessels on the surface varicosed and much enlarged. The cortical substance is tough and fibrous ; the kidneys are of a red or buff color, and usually a number of small cysts are scattered through their substance.

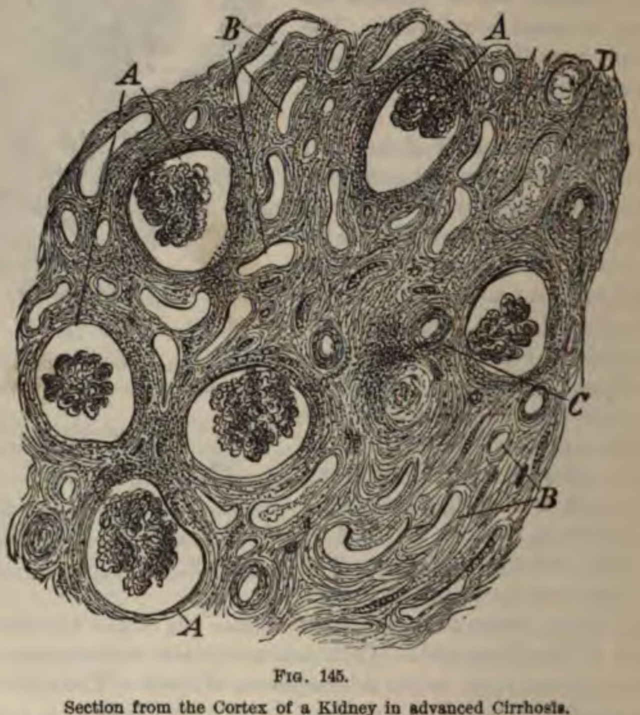

FIG. 145.

Section from the Cortex of a Kidney in advanced Cirrhosis.

A, A, A. Malpighian bodies with shrunken tufts and thickened capsules which fuse with the intertubular connective-tissue.
B, B. Nearly obliterated convoluted tubes.
C. Small Arteries with thickened walls.
D. Convoluted tubes containing colloid material. × 60.

Etiology.—The two most common causes of this form of kidney degeneration are gout and rheumatism. One of these causes is so frequently associated with its development that it has given the name of "gouty kidney" to it. The constant and continued use of alcohol may be regarded as another cause of cirrhotic kidney, for we not infrequently find this condition of the kidney associated with cirrhosis of the liver ; and the same steady and prolonged indulgence in the use of alcoholic drinks which produces cirrhosis of the liver, may produce cirrhotic kidney. These are its three principal causes.

It is occasionally met with in connection with lead poisoning. It has been claimed that the passive hyperæmia of the kidneys which occurs in connection with some forms of heart disease leads to the development of cirrhotic kidney. Cold, especially in a variable climate, exposure, poverty, and bad hygiene are strong predisposing causes. It is met with most often in and after middle life. Active brain workers are more liable to it than those who are indolent and phlegmatic.

Symptoms.—The early symptoms of the cirrhotic form of Bright's disease are always obscure. It is so insidious in its development that its commencement can rarely be determined. One of its earliest and most constant signs is a frequent desire to pass urine, which may contain neither albumen nor casts. Dropsy may be absent, and there may be none of the symptoms which usually mark the presence of kidney disease. There may be only ill-defined nervous symptoms during life, and yet at the autopsy extensive cirrhotic degeneration of the kidneys may be found.

Usually the disease is developed in the following manner: an individual notices that he is growing feeble without any apparent cause; he is suffering from dyspeptic symptoms; he notices that he is passing a larger quantity of urine than normal, and perhaps at the same time there will be a slight swelling of the lower extremities after prolonged exertion, such as standing or walking. This œdema comes and goes, is more marked at night on retiring, and disappears in the morning on rising. The complexion assumes a dingy hue. His disposition changes, he is morose, fretful, and his memory is treacherous. Insomnia and headache are tormenting, and there may be sudden loss of sight. The appetite is lost or is capricious. It is for the relief of their dyspeptic symptoms that this class of patients usually consult a physician, and a plan of treatment is adopted for their relief, with the assurance that they will be better as soon as they can leave off work and take rest. A single or repeated examinations of the urine may fail to detect either albumen or casts, and the promises of speedy recovery become more positive. The case goes on; the patient becomes more and more feeble, he has a careworn look, the complexion is altered, the eye has a peculiar expression on account of the œdema of the conjunctiva, nervousness and restlessness increase, and insomnia becomes constant; suddenly under great excitement convulsions occur and the individual passes into coma, remains insensible for twenty four hours and dies. Perhaps the urine was examined the day before the convulsion and no albumen was found; but if it is examined at the time of the seizure both albumen and casts are present.

The three prominent symptoms of this form of Bright's disease are changes in the urine, the dropsy and the nervous phenomena.

The *urine* is increased in quantity and of low specific gravity. It is characteristic of the urine in this form of nephritis that albumen is sometimes present and sometimes absent. In the other forms, albumen is always found in greater or less quantities. It may be necessary to examine several specimens before casts will be found, but when found, they usually are of the large hyaline variety; granular casts are infrequent; often several examinations of the urine are necessary before any satisfactory evidence of the disease can be obtained.

Dropsy is never very marked. Slight œdema of the feet and ankles after exertion is present in most cases. When œdema of the feet and ankles is constant, and is associated with the general symptoms and conditions of the urine which have been described, the diagnosis is readily made. When ascites is present, it is due to changes which have taken place in the liver rather than to those in the kidney.

Its most prominent symptoms are associated with its *nervous phenomena*: they come and go in a manner not well understood. The earliest and most constant is headache, which is often violent; occurring as it very commonly does with gout and rheumatism, it is very apt to be regarded as gouty or rheumatic in character. With these headaches there is more or less disturbance of nerve function, such as vertigo, temporary inability to speak, loss of sight and hearing, diplopia, myopia, presbyopia—numbness, neuralgic pains, muscular cramps, chorea, temporary and partial paralysis in one arm or leg, hemiplegia or paraplegia. Nervous dyspnœa is not uncommon, and it may be accompanied by "Cheyne-Stokes' respiration." There may be confusion of thought or impairment of memory; confirmed mania may be developed. Uræmic vomiting inducing great prostration, and anæmia—unaccompanied by dropsy—are alarming symptoms. There may be excessive itching of the surface. These patients are always liable to convulsions after severe mental or physical exertion; from the convulsions they may pass directly into coma, or become delirious, with a brown, dry tongue, dilated pupils, and thus gradually become comatose.

It is always important to remember the dangers to which these patients are constantly exposed. Cardiac hypertrophy is present in a greater or less degree in the advanced stage. The hypertrophy is usully confined to the left ventricle. The presence of left ventricular hypertrophy without valvular insufficiency is sufficient to direct attention to the kidneys. If, in connection with the cardiac hypertrophy the urine is abundant and of low specific gravity, containing only a trace of albumen, the evidences of contracted kidney are almost positive. Many theories have been advanced to explain the connection between cardiac hypertrophy and the cirrhotic kidney; some regard it as purely mechanical, produced by "the obstruction to the renal circulation and the consequent increased pressure in the aorta;" but *there is no condition* of renal obstruction that will explain the hypertrophy. Others claim that the walls of the renal and of all the other arteries progressively hypertrophy from the altered condition of the blood and the retained urinary excretion, until the heart becomes hypertrophied as "a result of the antagonism of forces." The order of its occurrence seems to be, first, capillary resistance; second, high arterial tension ; third, cardiac and arteriole hypertrophy ;[1] fourth, fibroid degeneration of the cardiac walls and secondary dilatation.

Amaurosis is a frequent attendant of cirrhotic kidney ; the loss of sight comes on gradually ; one eye only may be affected, but usually both eyes are equally involved ; the cause of the loss of sight is a true neuro-retinitis, which can readily be recognized by an ophthalmoscopic examination. The optic papilla is cloudy and swollen ; the retinal veins are distended and tortuous, and there are white patches on the retina. White dots and streaks in the perimeter of the *macula lutea*, are thought to be characteristic.

Differential Diagnosis.—This variety of Bright's disease may be mistaken for *diabetes*. The thirst, the large quantity of urine passed, the dyspeptic symptoms, the progressive emaciation, the absence of casts and albumen lead toward diabetes ; but the low specific gravity of the urine and the absence of sugar soon settle the question. The presence of a gouty or rheumatic diathesis, the insidious development of the disease, the large quantity of urine, its low specific gravity, with little or no albumen and only occasional casts, are sufficient to distinguish this from the other forms of Bright's disease.

Prognosis.—When the anatomical changes which characterize this form of renal disease are once established, their tendency is to progress ; and although a long period may elapse between their commencement and the fatal termination, yet whenever there is reason to believe that the morbid processes are advanced the patient is constantly in danger from complications. Serous inflammations are not as liable to occur as in other varieties of Bright's diseases, but mucous inflammations are more frequently met with, especially bronchitis, which assumes a chronic type.

Its *complications* are pericarditis, pneumonia, acute and chronic bronchitis, pleurisy, chronic gastric and intestinal catarrh, cirrhosis of the liver, atheroma and sclerosis of arteries, eczema, and psoriasis. In its advanced stage hemorrhages from mucous and serous surfaces, as well as into the substance of organs, are liable to occur. The most serious of these hemorrhages are the cerebral. It is more frequently associated with cerebral apoplexy than any other form of kidney disease. Hemorrhages in the retina are common.[2] It must be remembered that inflammation of the uriniferous tubes may be ingrafted upon cirrhotic kidney, and that the three forms of degeneration may be present in the same kidney.

Treatment.—In this form of kidney disease no special plan of treatment can be adopted. It has been claimed that the long-continued administration of mercury in small doses has the power to arrest or prevent connective-tissue development. When cirrhotic kidney is developed in connection with a gouty or rheumatic diathesis, the prolonged use of mercurials is contra-indicated. When it is developed in connection with lead-poisoning, mercurials are most decidedly contra-indicated. Mercurials are of especial advantage in those cases in which cirrhotic kidney is developed in connection with a cirrhotic liver. The bichloride is the preparation usually employed in such conditions. If the disease develops in connection with gouty or rheumatic manifestations, the same means which are employed to relieve gouty or rheumatic articular manifestations will afford relief.

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[1] Dickenson regards the vascular lesions as partly hypertrophy, and partly fibroid.

[2] In 100 cases reported by Mahomed seventeen died of heart disease and fifteen of apoplexy, *i. e.*, thirty-two per cent. of cardio-vascular changes. Of thirteen who died of surgical diseases, he says many died indirectly from failing heart. Eighteen died of lung diseases (eleven from severe bronchitis and emphysema, and seven from pleurisy and pneumonia).

Many of these patients will derive great benefit from residing for a time in those localities where they may constantly use water from alkaline springs. The Germans and French recommend very extensively the use of alkaline waters in the treatment of this class of diseases. Milk, skimmed milk, and butter-milk have all been vaunted as possessing curative properties; hence the once famous "milk cure." Although these patients appear anæmic, their nervous symptoms are aggravated rather than relieved by the use of iron. In a certain proportion of cases cod-liver oil will be found of service, especially when combined with the hypophosphite of soda; diuretics are not indicated, but when a marked diminution in the urinary secretion occurs, their temporary employment may be of service. When the disease is developed in connection with cirrhosis of the liver, an occasional hydragogue cathartic may be attended with benefit. It is of the utmost importance that this class of patients should make a permanent residence in a warm climate, and that all the exciting causes of cirrhotic development should be carefully avoided. Although a cure cannot be hoped for, the progress of its development may be delayed, and by carefully watching the condition of the nervous system, and by timely interference, the development of the graver forms of nervous disturbance may be delayed or prevented, and the life of the patient prolonged. For symptoms or complications that demand a narcotic or anodyne, *opium* is to be used in preference to all others. In its advanced stages, inhalation of oxygen has caused disappearance of albumen.[1]

Whenever there is extensive general anasarca, and the respiration becomes impeded by œdema of the chest-walls, or by an œdematous condition of the lungs, and all other means have failed to relieve the dropsy, prompt and sometimes permanent relief may be afforded by making free incisions through the skin into the areolar tissue above the ankles, or by pricking the parts with needles in many places.[2] Those dyspeptic and gastric symptoms which are so obstinate and distressing can usually only be relieved by a carefully regulated diet.

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[1] Dujardin-Beaumetz.

[2] See *London Lancet*, 1877, i. 649. Southey uses drainage tubes in anasarca.

WAXY KIDNEY.

("*Amyloid Form*" *of Bright's Disease.*)

Amyloid degeneration is always chronic ; it has no acute stage, and cannot, strictly speaking, be regarded as a nephritis aside from the associated changes. It usually invades several organs of the body simultaneously ; when the kidney is the seat of this degeneration its tissues become infiltrated with amyloid material. Cornil and Ranvier found that waxy degeneration in the kidneys was *invariably* associated with chronic parenchymatous nephritis ; they are, moreover, convinced that the latter condition *always precedes* amyloid degeneration.

Morbid Anatomy.—The primary waxy changes take place in the walls of the minute arteries ; secondarily the secreting tubes and cells are involved. At first, when the walls of the vessels are principally involved, there is little change in the appearance of the kidneys. They may be slightly increased in size, firmer, and of a paler color than normal.

On section the Malpighian tufts appear more prominent than normal, and present the appearance of gray translucent points, which reflect light better than the surrounding tissue. Usually both the cortical and medullary portions are simultaneously, but unequally, involved ; by the "iodine test" the amyloid change, however slight, will become very distinct, and a section under the microscope will show the change to be most marked in the vessels in the Malpighian tufts, the *vasa recta* and in the middle coats of the small arteries.

In a more advanced stage of the process the kidneys will be increased in size, their capsules be non-adherent, their surfaces smooth and of a pale color with stellate vascularity.

On section the increase in the size will be found to be due to an increase in the cortical substance, which is *denser* than normal. The medullary substance is but slightly increased. The normal anatomical outline of the cortical and medullary portion is lost, the Malpighian tufts are indistinct, looking like little grains of boiled sago, and the whole cortical substance has a peculiar waxy appearance. Under the microscope an entire section will present a shining yellow appearance, as if all the tissues of the organ were infiltrated

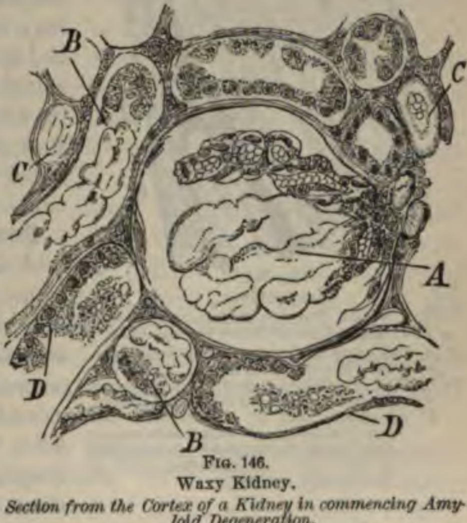

FIG. 146.
Waxy Kidney.

Section from the Cortex of a Kidney in commencing Amyloid Degeneration.

A. *Malpighian body. The lower part of the vascular tuft is the seat of the amyloid change.*
B, B. *Convoluted tubes containing hyaline and granulo-fatty matter.*
C. C. *Arteries, with coats showing waxy degeneration.*
D, D. *Epithelium of convoluted tubes containing granular and fatty matter.* × 300.

with amyloid material. The glomeruli, most of the arterioles, the small veins and the basement membrane of the tubules will be infiltrated. The epithelial cells of the convoluted tubes are not infrequently flattened. The contents of the tubes may be made up of broken-down epithelium and fatty granules, mingled with a material which is fibrinous in its nature; this material will not, however, give the characteristic reaction of amyloid matter. Fatty, granulo-fatty, and hyaline materials are found in all cases in addition to the above. Usually the kidneys atrophy and become very much diminished in size, sometimes less than one-half their normal size; their capsules are adherent, their surfaces uneven, granular, and of a pale color.

On *microscopical* examination it will be seen that the diminution in size is due to decrease of both the medullary and cortical portions. The Malpighian tufts are large and prominent, and are grouped together; the small arteries are enlarged and at points are impervious. On examination of sections from different portions of the kidney, the tubules will be found at all points more or less atrophied and their walls collapsed; some are obliterated; the blood-vessels will appear thickened, and their outline will be more or less irregular. Iodine upon the degenerated Malpighian tufts will give the characteristic amyloid reaction. The degree of atrophy may vary, but however extensive it may be, by dipping a section in the iodine solution, and microscopically examining it with a low power, one will always find abundant evidence of amyloid material in the degenerated vessels and tubes.

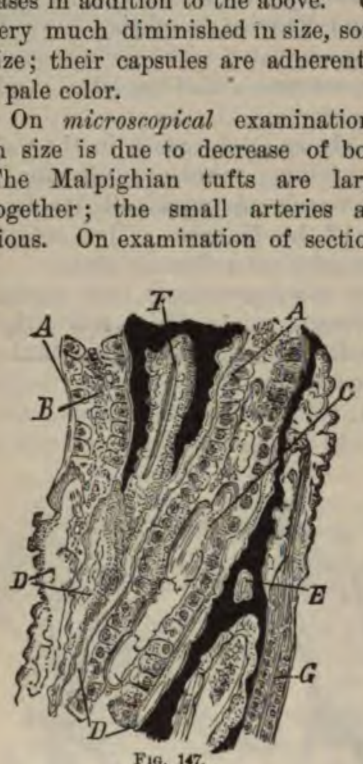

Fig. 147.
Waxy Kidney.
Vertical Section from the Medullary Portion of a Kidney in advanced Waxy Degeneration.
A, A. *Collecting tubules containing fatty granules, B, and colloid matter, C.*
D, D. *Wall of tubules showing thickening and irregularity, the result of waxy change.*
E. *Transverse and F longitudinal section of blood-vessels with lumen nearly obliterated by amyloid degeneration of the coats. G. Ascending limb of Henle's loop.* × 350.

Etiology.—The primary cause of amyloid degeneration is still a vexed question. It never occurs in those who are in perfect health, and the circumstances under which it almost uniformly occurs determine to a certain extent its causation. It is most frequently met with in syphilitic subjects. Another frequent cause is prolonged suppuration, especially when associated with diseases of bone. A long-continued empyema may give as a result an amyloid kidney. It is not infrequently met with in those who die of pulmonary phthisis, consequently chronic suppurative diseases of the lungs must be ranked among its causes. Caries of bone, ulcers of the intestines, cancer, and chronic rheumatism may induce it.

Symptoms.—The symptoms which attend the development of amyloid degeneration of the kidney are never well marked. The usual manner of its development is as follows: an individual who is suffering from tertiary syphilis or some exhausting form of disease, notices that he is losing strength, that he is becoming more feeble than usual, and that he has less mental and physical vigor than he is accustomed to have; that he is troubled with shortness of breath on exertion; that he has an unusually pallid countenance, and that there is a great increase in the quantity of urine passed. He is obliged to rise two or three times during the night to pass urine, and at times he passes large quantities. He also notices a fulness of the abdomen which he has never before observed, and sometimes there is a sense of weight in its upper portion. He may have detected a tumor in the right and perhaps in the left hypochondrium. When he assumes the recumbent posture, he must have the upper portion of the body elevated to prevent dyspnœa. Doubtless the dyspnœa is partially due to the anæmia and partially to the pressure caused by an enlarged liver and spleen. Perhaps there is slight œdema about the ankles, especially at night. The patient does not perspire readily, but when he does the perspiration has a urinous odor. Certain articles of food, especially fatty substances, which never before have disagreed with him, now give rise to dyspeptic symptoms and he may have occasional vomitings.

This train of symptoms coming on in one who has been the subject of any of the forms of disease to which I have referred, leads to the suspicion that amyloid degeneration of the kidney is taking place. If, upon further examination, a marked enlargement of the liver and spleen is found, and the surface of the liver is smooth and its edges sharp, it is almost certain that the amyloid form of Bright's disease exists. With these symptoms there will also be more or less fluid found in the abdominal cavity, but its presence will be due to changes which have occurred in the liver and not to changes in the kidneys. The blood is slightly altered; the white corpuscles are somewhat increased in number, and the red are diminished and ill-defined; in a large proportion of cases there is a peculiar cachexia present which is almost characteristic. The patient has a pale, waxy complexion, with little pigmentary deposits in the skin, particularly about the eyelids. This cachexia is usually most marked in syphilitic subjects.

As in the other forms of kidney disease, there are three important symptoms to be considered. First, abnormal changes in the urine; second, dropsy; third, nervous phenomena.

The *urine* is increased in quantity, the patient perhaps passing as much as one hundred ounces in twenty-four hours. It is light colored, looking very much like clear water, or it may have a slight amber color. It is of low specific gravity, sometimes as low as 1.005. When tested for albumen it will be found always to contain an appreciable quantity, never a large quantity. The amount of urea excreted is but little if at all diminished; the urine will always contain casts, either large hyaline or fine granular, or both, but the hyaline predominate. Casts of either variety usually are not abundant, and several examinations may be required before their presence or absence can be positively determined. Epithelial and fatty casts are sometimes found.

Dropsy is never very marked in this form of Bright's disease. The general anasarca which is so frequently met with in connection with parenchymatous nephritis, is never present. There may be slight œdema of the feet, especially at night, and there may be fluid in the abdominal cavity. The *nervous symptoms* are never very prominent. This class of patients do not usually suffer very much from headache, and rarely have convulsions or pass into coma. They usually die from exhaustion, or from some complication, or, in other words, die from amyloid degeneration of other organs, diarrhœa, the result of amyloid changes in the mucous membrane of the intestine, or ascites.

Differential Diagnosis.—The diagnosis of this form of Bright's disease is not difficult when it occurs as a late manifestation of syphilis. A copious secretion of urine of low specific gravity containing little albumen and few casts, in one who has a syphilitic history with an enlarged liver and spleen, leaves little doubt as to the character of the kidney change. It is hardly possible to confound the cachexia which attends this form of Bright's disease with that of any other chronic disease, for a urinary examination will give positive evidence of the renal disease, and it only remains to determine its character, which is usually readily reached by the history of the case. The large quantity of urine passed often causes the patient to consult the physician with the idea that he has *diabetes*, but the urinary examination soon settles this question.

Prognosis.—The duration of this form of Bright's disease is uncertain; it undoubtedly takes many years for the anatomical changes in the kidney to reach the stage of atrophy, yet when waxy changes are once established recovery is impossible. Resulting as it does from a grave constitutional cachexia, the causes which produce it are so often continuous that they are only in a slight degree influenced by treatment.

The progress of the disease may sometimes be temporarily arrested, but its usual course is steadily progressive to a fatal termination. Amyloid degeneration of the kidneys may exist for many years, and yet the patient enjoy a comparatively good degree of health. I now have the care of a medical gentleman in whom the disease has existed certainly eight years, yet he is in such good health as to be able to discharge the duties incumbent upon a large country practice.[1] An exhausting diarrhœa or an abdominal dropsy is often the direct cause of death. Most of the complications which occur are degenerative in character. Patients are not especially liable to have pneumonia, bronchitis or pericarditis, or any of the acute inflammations which occur in connection with other forms of kidney disease. Cardiac hypertrophy is rarely present in any stage of the amyloid kidney. Its early symptoms are so obscure that it is difficult to determine its average duration.

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[1] Bartholow records a case where there was complete recovery from the waxy kidney.

Treatment.—This is an incurable disease; there are no known means for arresting it or preventing its development. The same general principles are to govern its treatment as govern the treatment of waxy degeneration in other organs. First, if possible remove its cause, as diseased bones, prolonged suppuration, or purulent accumulations. If it occurs with syphilis anti-syphilitic remedies are indicated, always remembering that waxy degeneration occurs only as a tertiary manifestation of syphilis, and that all measures which have a tendency to debilitate the patient must be avoided. Iodide of potassium and mercury are the most reliable remedial agents. Both of these agents have gained some favor as remedies in the treatment of Bright's disease, and there are those who employ indiscriminately one or the other or both of them. The benefit derived in certain cases from their use is undoubtedly due to their power over syphilitic manifestations. In such cases, the long-continued use of small doses of mercurials will generally be followed by marked improvement, but care should be exercised that their use be not continued until the specific effect of the drug is produced.

When these patients are in a debilitated condition iodide of potassium with cod-liver oil will be of greater service. The form of iodine which I have found most serviceable to this class of patients is pil. ferri iodidi. One of these pills given three times a day, at the time of taking food, is often followed by the most beneficial results. Diuretics and hydragogue cathartics will rarely be required. The tincture of the perchloride of iron, quinine, nux vomica, and sirups of the phosphates are often beneficial.

PYELITIS.

Pyelitis is an inflammation of the mucous membrane of the pelvis and calices of the kidney, and may run an acute or chronic course. It may involve the pelvis and infundibula of one or of both kidneys. Some describe an acute catarrhal, pseudo-membranous, and calculous pyelitis and a chronic purulent pyelitis that may or may not result in pyonephrosis.

Morbid Anatomy.—In acute pyelitis the mucous membrane of the pelvis of the kidney is at first more or less reddened. When very hyperæmic the surface will be dotted here and there with little dark-red spots which are minute ecchymoses; the epithelium of the mucous surface is more or less removed; sometimes it is entirely removed, at others it is removed in patches. The peculiar "tailed" cells of the pelvis are thrown off in great quantity. As the inflammation progresses the mucous surface becomes covered with more or less muco-pus. The urine in the pelvis will contain numerous desquamated epithelial and lymph cells.

In some cases a membranous exudation may be developed upon the mucous membrane of the pelvis, called "pseudo-membranous" pyelitis. It is a diphtheritic exudation occurring in connection with diphtheritic exudations in other parts of the body, and should be called diphtheritic pyelitis. This diphtheritic membranous exudation is liable to become detached and block up the ureter. Sloughs may form, and after their removal an ulcerated surface may be left.

In *chronic pyelitis* the mucous membrane of the pelvis of the kidney is congested and thickened, and its surface presents small vascular granulations. It assumes a grayish white or slate color and is traversed by dilated veins ; the pelvis and infundibula and ureter are dilated and more or less thickened. Pus is more or less abundantly formed, and if there is no obstruction it passes off with the urine. Calculi or fragments of calculi may be found mingled with the pus.

Should there be an impediment to its escape it accumulates in the pelvis, which it distends more and more, and at last gives rise to a condition known as *pyonephrosis*. This dilatation as it progresses encroaches first upon the papillæ, which become flattened and obliterated, next on the pyramids, and finally, by the pressure it causes, the cortical portion of the kidney disappears. The apices of the pyramids may suppurate and ulcerate. In such cases only a sacculated pouch remains containing from one to several ounces of fluid, which may be mixed with inspissated pus, broken down calcareous matter, ammoniacal products and calculi.

If a renal calculus is present, and the cause of the pyelitis, more or less extensive ulcerations may be established. These ulcerations may cause perforation of the pelvis, and give rise to extravasation of urine into the adjacent tissues. In this (so-called) calculous pyelitis the kidneys are always the seat of interstitial nephritis, cysts, marked atrophy, etc. The ureter of the kidney which is the seat of the pyelitis may be completely obstructed, and pus, blood, and urinous material may accumulate behind the obstruction. If these obstructions are permanent an opening may be made through the dilated ureter and the contents of the sac discharged into the adjacent tissues, into some hollow viscus or into the abdominal cavity, or by an adhesive inflammation reach the surface and be discharged externally.

When the obstructions are temporary the contents of the sac are discharged into the bladder through the ureter when they give way, and such obstructions or accumulations may occur repeatedly. Sometimes these retained accumulations undergo entire absorption, and there remains a thick cicatricial tissue, with the normal kidney tissue entirely obliterated, and the ureter becomes transformed into a tendinous cord. Under such circumstances if the fellow kidney is healthy it becomes increased in size and performs in a very satisfactory manner the function of both kidneys, and the patient may live for many years. Again, in certain cases, the accumulation in the kidney is changed into a cheesy material, and presents an appearance resembling what is known as tubercular kidney. Mingled with this cheesy mass may be found the urine-salts which cause it to have a sandy feel. The kidney may be changed into a fibrous shell containing pus and débris.

Etiology.—Pyelitis is seldom, if ever, a primary disease. Its most frequent cause is the presence of calculi or some foreign substance in the pelvis of the kidney, and the pyelitis is then secondary to mechanical irritation. Pyelitis may result from extension of inflammation from the bladder or ureter, or from acute interstitial nephritis, rarely from perinephritic abscess. It may result from the irritation produced by the decomposition of urine retained in the pelvis of the kidney, as a consequence of some obstruction to its normal outlet. For instance, an enlarged prostate gland, a tumor pressing on the ureter, paralysis of the bladder, or an urethral stricture which causes obstruction to the passage of urine from the bladder. As a result of retention of urine in the bladder, cystitis is developed, and the inflammation of the mucous surface of the bladder may extend to the ureters, and from the thickening of their mucous lining and the diminution of their calibre, the passage of urine from the kidneys to the bladder is obstructed, and there is not only retention of urine in the bladder but also in the pelvis of the kidneys. As a result of such retention the urine undergoes decomposition, the urea is changed into carbonate of ammonia and water, the carbonate of ammonia acts as an irritant and excites inflammation of the lining membrane of the pelvis, and thus pyelitis is developed.

The absorption of the ammonia resulting from the decomposition of the urea may be sufficient to give rise to a condition which has received the name of *ammonæmia*. This condition is not infrequently mistaken for uræmia, yet they differ widely in their manifestations and the dangers which attend their development. In ammonæmia the urine when voided is ammoniacal, as are also the breath and perspiration. The mucous membrane of the mouth is dry and shining; the complexion is sallow and there is increasing emaciation; no dropsical accumulations are present. Convulsions and vomiting are rare; chills are frequent. Death is usually preceded by coma. The development of the train of symptoms indicative of ammonæmia, accompanied by the evidence of obstruction to the normal outlet of the urine, should cause one to hesitate before performing any operation, especially an operation for relief of stricture of the urethra.

Pyelitis not infrequently occurs in connection with that class of diseases which depend upon blood poisoning—pyæmia, diphtheria, and typhus fever. In this connection it is generally a complication of acute Bright's disease, which is not severe in character, but which causes bloody urine. It is an almost diagnostic complicating symptom of myelitis. Pyelitis occasionally occurs in consequence of over-doses, or the prolonged use, of certain irritating drugs, as turpentine, cantharides, and other stimulating diuretics. In very rare instances it seems to come on idiopathically from exposure to cold and wet, or from some unknown cause.

Symptoms.—In the majority of cases the development of pyelitis is preceded or accompanied by symptoms due to the causes which produce it, such as renal calculi, diseases of the bladder, etc.[1] Prominent among those symptoms which directly attend its development is pain in the back. This is present in the mild as well as in the severe cases. This pain may have its point of maximum intensity over one or both lumbar regions. It is often of an aching character, and shoots down along the course of the ureters. This pain is usually accompanied by frequent micturition, and when it is very intense the voiding of urine is almost incessant and is attended by severe pain. The commencement of acute pyelitis is usually marked by rigors, and in that chronic form in which temporary obstruction of the ureter occurs, rigors are frequent.

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[1] It is said (Klebs) that bacteria may be carried into the bladder on unclean catheters or other instruments, and that these, making their way into the pelvis, cause pyelitis.

Symptoms of hectic fever may also mark the occurrence of permanent obstruction of the ureter and the development of that condition termed *pyonephrosis*. There is usually considerable lassitude attending the progress of pyelitis, and when the disease is due to the presence of a calculus, the patient ordinarily suffers more or less pain on motion.

All of these symptoms are accompanied by changes in the *urine*, and these changes are its most reliable signs ;—in its early stage the urine contains blood mixed with mucus and epithelial cells from the pelvis and infundibula : the presence of these epithelial cells, which are readily distinguished from epithelium of any other portion of the urinary tract by their characteristic shape and appearance, is its most certain diagnostic indication. The specific gravity of the urine ranges from 1.025 to 1.030, and it usually retains its acid reaction. In the more advanced stages the characteristic epithelium is to a great extent replaced by an abundance of pus cells, but the urine retains its acid character. If sacculation of the kidneys is developed, the urine will become ammoniacal. More pain and hemorrhage attend calculous pyelitis than the other forms. Albumen is present in proportion to the amount of pus and blood. In the advanced stage of pyelitis, if the urinary channels remain free the discharge of pus is constant. If the ureter becomes blocked, for a time the urine may be quite normal, but the removal of the obstruction is followed by a copious flow of purulent urine. This may be repeated from time to time, at intervals varying from a few days to a few months. If the pelves of both kidneys are affected, and there is partial or complete obstruction of one side, the accumulation of pus in the urine is diminished, but not entirely prevented. If the obstruction is long continued or becomes permanent, a tumor develops in the lumbar region.

The development of a *pyonephrotic tumor* indicates complete obstruction of the ureter. The existence of the tumor is determined by the presence of bulging between the crest of the ilium and the false ribs on the right or left side, according as the right or left kidney is involved. As a consequence the outline of the abdomen is rendered unsymmetrical. On palpation, deep-seated fluctuation is felt over the tumor, which usually is tender on pressure. The area of percussion dulness will correspond to the outline of the tumor, except where it is crossed by the colon. With these physical signs present, and a history of pyelitis, one will be justified in resorting to the exploring trocar to complete the diagnosis.

Differential Diagnosis.—The diagnosis of pyelitis in its first or acute stage rests almost exclusively on the presence in the urine of the characteristic epithelium of the pelvis and infundibula mixed with blood globules and mucus. If the urine contains pus cells mixed with these epithelial cells, it indicates a more advanced stage of the disease. The presence of pus and acid urine, with pain in the lumbar region, accompanied by the development of a tumor at the seat of pain, which tumor gradually increases in size and suddenly disappears at the same time that a copious discharge of pus takes place from the bladder, which discharge is attended by a sense of great relief to the patient, renders the diagnosis of pyonephrosis very certain. If the ureter of the affected kidney is permanently obstructed, the lumbar tumor is liable to be mistaken for *hydronephrosis*, an *hydatid cyst*, or a *perinephritic abscess*.

In *perinephritic abscess* neither pus, blood, mucus, epithelia nor albumen will be found in the urine ; in pyonephrosis they are common and constant. Pain on *motion*, the occurrence of slight œdema over the tumor, the delayed appearance of fluctuation—these are in contrast to the symptoms of pyonephrosis. The mass of tumor in perinephritic abscess may be *tilted forwards* by pressure in the renal region, which is *never* the case with pyonephrosis.[1] Fever is a marked symptom in abscess, and slight or absent in pyonephrosis. In women a pyonephrotic tumor has been confounded with an *ovarian cyst*. The exploring trocar will very quickly remove all doubts.

Pyelitis is distinguished from *cystitis* by absence of vesical pain and frequent micturition, by *lumbar* pain, and by the intimate admixture of foreign materials in the urine. Pelvic epithelial cells are not found in the urine of uncomplicated cystitis. In pyelitis the urine is acid ; in cystitis it is alkaline. When pyelitis occurs as a complication of chronic cystitis, an enlarged prostate gland, or urethral stricture, it is often impossible to diagnosticate its existence if there is no tumor in the lumbar région. Under these circumstances the character of the urinary constituents is not of much assistance. If, however, the quantity of pus is large, the urine slightly acid, the loins painful on pressure, and the febrile movement constant, with rapid loss of flesh and strength, there is good reason to believe that chronic pyelitis has been added to disease of the bladder and urethra.

Prognosis.—The prognosis in pyelitis depends upon the nature of its exciting cause. In simple catarrhal pyelitis, not connected with extensive disease of other portions of the urinary apparatus, the prognosis is good, unless the disease affects both kidneys and has reached the purulent stage; then, whatever may have been its cause, the prognosis is bad. When the disease is confined to one side, recovery is possible, although one kidney may be completely destroyed. We suspect unilateral pyelitis with calculi and with tumors that compress an ureter ; but following a cystitis, urethritis, prostatitis, etc., the affection is usually *bilateral*, and the prognosis is unfavorable.

Pyelitis may be regarded as a hopeless disease when it is secondary to an enlarged prostate gland, extensive chronic cystitis, urethral stricture, or cancer of the kidney. It is exceedingly grave when it depends upon renal calculi or hydatids, although it is not necessarily fatal. The issues of a pyonephrosis are uncertain ; the various directions in which a sac may burst determine to a great extent its termination. Rupture into the peritoneal or thoracic cavity is speedily fatal. Recovery is possible if the rupture takes place externally or into the intestine. Sometimes, when the sac does not rupture, patients die from the exhaustion caused by the long-continued discharge. Recovery may be reached by a gradual diminution of the discharge and a final contraction and obliteration of the sac, provided the other kidney is unaffected. Death may occur from uræmia or ammonæmia.

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[1] London *Lancet*, January, February, March, 1879.

Treatment.—The first thing in the treatment of pyelitis is, if possible, to remove its cause. If the attack is an acute one, and at the onset of the disease the fever is considerable, the pain in the lumbar region severe, and the urine bloody, wet cups should be freely applied to the loins, followed by a hot bath, and a sufficiently large hypodermic of morphine to entirely relieve pain. The patient should drink freely of alkaline fluids and should be kept in bed.

In chronic pyelitis, when the secretion of pus is abundant, astringents may be employed to diminish the purulent secretion. Balsams are here indicated. Attention should be paid to the general health of the patient. Cod-liver oil and quinine should be administered with a nutritious and non-stimulating diet. A residence at, and prolonged use of the waters of some alkaline spring will often be found of great service. Diluent alkaline drinks and milk should be the sole articles of diet in the acute stage.

When a tumor exists and can readily be reached through the integument, aspiration may be performed, *after which* the question of a free permanent external opening will present itself, and must be decided by the peculiarities of the case.

HYDRONEPHROSIS.

Hydronephrosis is a chronic, non-inflammatory affection of the pelvis of the kidneys. Whenever the flow of urine through the ureters into the bladder is permanently obstructed, the urine collects in the pelvis and infundibula, compressing the renal substance, which becomes partially or completely atrophied, so that after a time the kidney is converted into a sac or pouch. This condition has received the name of hydronephrosis, or *dropsy of the kidney.* The dilatation may affect the ureter and pelvis, or only the pelvis.

Morbid Anatomy.—In a kidney that is the seat of moderate hydronephrosis following simple dilatation of the pelvis, the papillæ will become flattened, hardened and shrunken, and gradually disappear. The remaining portion of the renal substance gradually diminishes from the pressure and becomes more or less tough and resistant. In extreme cases the kidney substance finally entirely disappears and the kidney is converted into a large multilocular cyst; sometimes it is unilocular. At times such a cyst attains a size as large as a child's head; there is a case recorded where the whole abdominal cavity was occupied by an enormous tumor containing sixty pounds of fluid. Some healthy kidney substance will nearly always be found in its walls. That portion of the ureter which is the seat of dilatation may reach the size of a small intestine, has a blue-white color, its walls become greatly thickened, and it may become convoluted.

The fluid contained in hydronephrotic cysts is generally altered urine. It is much more watery than normal urine, containing more or less of the urinary salts; it may also contain blood, pus, epithelium and some albumen. Sometimes it is perfectly clear; it is usually alkaline. Adhesions frequently form between the enlarged kidney and neighboring organs.

Etiology.—Closure of a ureter which gives rise to hydronephrosis may be due to compression by a tumor external to its walls, especially rectal or uterine, or to the impaction of a calculus, blood-clot, or mass of echinococci within it, or to inflammation which has caused adhesion of its walls and complete obliteration of its lumen. A moderate degree of dilatation of the ureter sometimes results from obstruction to the free discharge of urine from the bladder; when this is the case the pelvic dilatation is bilateral, and can never become very extensive without destroying life, for when the pressure becomes equal to that within the blood-vessels the urinary secretion is entirely suppressed. Congenital defects often cause it.

Symptoms.—The symptoms of hydronephrosis depend upon the nature of its cause and the extent of dilatation. If the sac is small and the opposite kidney healthy, there may be no symptoms to indicate its existence; there will be no diminution in the urinary secretion, as the healthy (usually hypertrophied) kidney performs the work of its diseased fellow. There may be pain in the lumbar region.

As soon, however, as the tumor attains sufficient size to be readily felt, the existence of hydronephrosis may be determined by it. This tumor causes no pain or inconvenience except by its pressure. With double hydronephrosis uræmic symptoms may develop suddenly. The nephritic tumor is fluctuating, usually lobulated, and gives a tympanitic resonance in front on percussion unless the colon has been pushed aside. If the obstruction to the escape of urine from the kidney is temporarily removed, its removal will be followed by a sudden diminution and disappearance of the tumor, coincident with a sudden discharge of a large quantity of pale urine. Such an occurrence is almost pathognomonic of hydronephrosis. Constipation, from pressure of the tumor on some portion of the intestine, is not infrequent.

Differential Diagnosis.—Hydronephrotic tumors may be confounded with *ovarian cysts*, *ascites*, *hydatid cysts*, and *pyonephrosis*.

They are distinguished from *ovarian cysts* by the presence of the colon in front of the tumors, by the absence of tympanitic percussion in the lumbar region, and by a vaginal and rectal examination.

Single hydronephrosis is distinguished from *ascites* by the non-existence of dulness in both lumbar regions. In ascites, when the position of the patient is changed, there is a change in the level of dulness, which never occurs in hydronephrosis.

It is quite impossible to distinguish hydronephrosis from an *hydatid cyst*, unless the hydatid vesicles are found in the urine, or the hydatid fremitus is present.

It is distinguished from *pyonephrosis* by the non-purulent character of the urine, and by the absence of constitutional symptoms. An aspirating needle will generally decide the diagnosis, for the watery urine withdrawn differs, chemically and microscopically, from the fluid obtained from hydatid or ovarian cysts or the pus of a pyonephrosis or a perinephritic abscess.

Prognosis.—The prognosis is more favorable in this than in any other form of renal tumor; yet it is always serious. When only one kidney is involved, life may be indefinitely prolonged, and there is always a possibility that spontaneous evacuation of the sac may occur. But cases are recorded where—one kidney only being involved—it caused death by pressure on neighboring parts. If the healthy kidney becomes the seat of any form of nephritic degeneration, the prognosis becomes unfavorable; complete suppression of the urine may then occur at any moment; or if the impediment which has obstructed one ureter extends so as to prevent the flow of urine from both kidneys, uræmic symptoms will be developed, and death speedily follow.

Treatment.—In hydronephrosis the principal thing to be accomplished is the evacuation of the tumor. To accomplish this result it should be carefully manipulated. This can readily be done, as the tumor generally causes no pain. If this does not cause its evacuation, aspiration should be resorted to. I now have a case under observation in which aspiration has twice been performed with complete relief to the patient, and the aspiration has not been followed by any unpleasant symptoms; nothing is to be expected from medicinal treatment.

CYSTIC KIDNEYS.

Cysts of the kidneys are very frequently met with at autopsies, but they are of very little clinical importance, for if the cysts are of small size they give no symptoms during life. Cystic degeneration may have a congenital origin, and both kidneys may be converted into a mass of cysts of sufficient size to entirely fill the abdominal cavity; such conditions are usually associated with other congenital malformations. It is claimed that cysts originate in the epithelia or even in the fibrous stroma of the kidney. They are often found scattered through kidneys that are otherwise healthy. It is difficult to make any practical distinction between the cysts of a true cystic kidney and those occurring with cirrhotic kidney. They are usually situated in the cortical substance near the surface. Colloid cysts of the glomeruli are frequently surrounded by laminæ of fibrin from hemorrhages within the capsule.

The contents of kidney-cysts vary in character even in the true cyst. They may contain a clear albuminous fluid; sometimes it is gelatinous, containing phosphates, carbonates, cholesterin, and very rarely urea and uric acid. The vascular tuft in a glomerulus that is transformed into a cyst, is flattened against the wall of the thickened capsule, and the cyst may be lined with pavement epithelia.

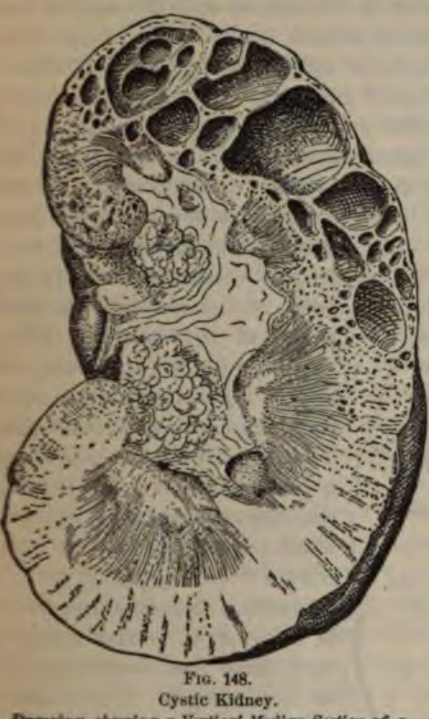

Fig. 148.
Cystic Kidney.
Drawing showing a Vertical Median Section of a Kidney containing Cysts.

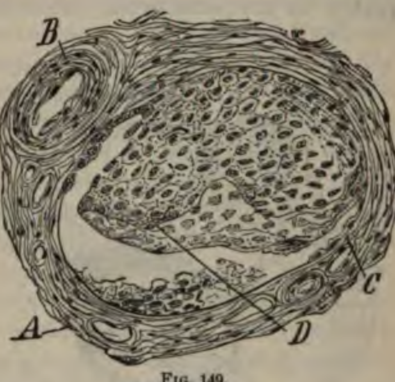

Fig. 149.
Cystic Kidney.
Section from the Cortex of a Cirrhotic Kidney, showing the Epithelial lining of a small Cyst.

- *A. Cirrhotic intertubular tissue.*
- *B. A small artery in transverse section showing the thickened coats.*
- *C. Cavity of a small cyst.*
- *D. Epithelial lining of the last, partly detached. (The extreme tenuity of this lining cannot be well shown in a wood-cut.)* × 300.

The origin of these cysts is obscure, although there is reason to believe that they are the results of dilatation of the kidney tubules and Malpighian bodies. Congenital cysts have their origin, as a rule, in the Malpighian bodies. Serous cysts are often found in kidneys that have undergone senile atrophy. They may be developed in the connective-tissue by an enlargement of a lacunar lymph-space.

RENAL CALCULI.

Renal concretions vary greatly in shape and differ in their composition. They may be deposited in the tubes of the pyramids, in the cortical substance, or in the pelvis of the kidney. Their development occurs at any age; they are met with in the kidney of the fœtus *in utero* and in the kidneys of the very aged.

Morbid Anatomy.—In the kidneys of infants dying within forty-eight hours after birth, brownish striæ of amorphous urates will invariably be found running from the papillæ to the base of the pyramids. In adults, urate of soda, in the form of crystals, may be found deposited in the white lines in the pyramids and cortical substance, both in the tubular and intertubular structure; this is always associated with a gouty diathesis. Carbonate and phosphate of lime may be found deposited in the tubes and pyramids of the kidneys of old people, or in connection with diseases of the bones. By far the most frequent variety is uric acid. Some think that oxalate of lime forms the starting-point of uric acid deposits. Cystine, ammonio-magnesian phosphate, or urate of ammonia and the mixed urates may form nuclei of renal calculi. Mixed calculi are not uncommon.

These different varieties of urinary concretions may be permanently impacted in the uriniferous tubes, and render them impervious and cause cysts to be developed, or they may be washed down the tubes by the urine, and finally deposit in the infundibula and pelvis of the kidney. They vary in number and size. A kidney may contain one or a large number of concretions. They usually vary in size from a pin's head to a hazel nut; the larger ones may fill the whole pelvis; the smallest constitute "*kidney gravel.*" If a concretion becomes impacted in the pelvis, it may attain a very large size, weighing one or two ounces. The smaller calculi pass through the ureters into the bladder and are discharged; the larger ones may permanently obstruct the ureters and become the cause of pyo- or hydronephrosis.

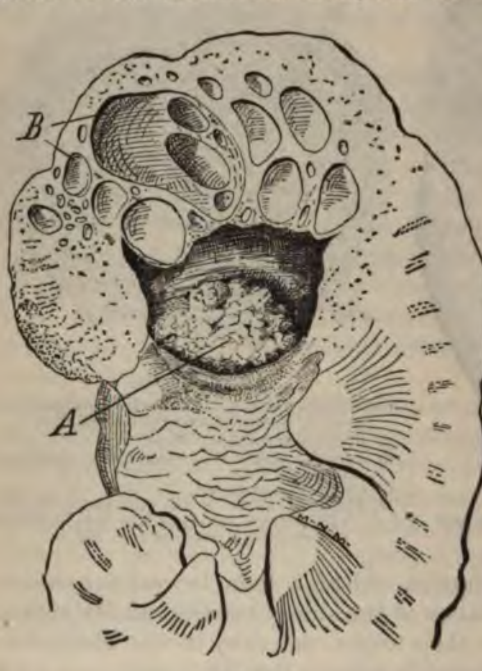

FIG. 150.
Renal Calculi.
Drawing showing an Impacted Renal (Mulberry) Calculus, A. B. Cysts.

The anatomical changes produced by renal concretions vary: they may cause pyelitis, pyonephrosis, hydronephrosis, or abscess, or they may excite parenchymatous nephritis.

Etiology.—The causes of the different concretions found in the kidneys are very obscure. Uric acid is most frequently met with in infants. The deposits of lime and triple phosphates are most frequently met with in adults. They are caused by the precipitation (in the nascent state) of uric acid or oxalate of lime due to renal excess of insoluble uric acid, or to deficiency in water of the urine. A colloid material composed of mucus or blood globules or other animal base exists in all. They increase by accretion. Certain constitutional conditions are supposed to be favorable to their development, but the exact nature of the urinary changes has not as yet been determined. In most cases, calculi that develop in the pelvis of the kidney have some foreign substance as a nucleus. These nuclei may be pus, blood, epithelium, or grains of pigment. The composition of the remaining portions of the calculi depends upon the varying conditions of the urine which attend their development.

Symptoms.—The symptoms which indicate the presence of renal calculi vary. In some instances they are well marked, in others very obscure. Usually, the existence of renal calculi is indicated by an aching pain in the lumbar region and loins, which frequently shoots into the testicles or labia and down the thighs,—by an itching at the end of the penis, and by a frequent desire to urinate. The urine often contains pus, blood and "tailed" epithelium from the pelvis of the kidney. These symptoms are usually aggravated by anything that disturbs the position of the calculus, especially by violent exercise, or by jolting in driving or horseback riding.

The symptoms often assume the characteristics of "*renal colic*," due to the passage of the calculus along the ureter to the bladder; this may occur after violent exercise or without any assignable cause. The attack may be sudden, or there may have been uneasiness in the loins for some time. The passage of a calculus along the ureter into the bladder is marked by sudden and intense pain in the region of the affected kidney. This pain radiates in various directions, but mainly toward the hypogastrium, testis, inside of the thigh and end of the penis. There is a constant desire to micturate,—"tenesmus of the bladder,"—but the urine is scanty or suppressed, and what is passed is of a smoky, high color, often bloody, and is discharged in drops, the individual at the time experiencing a painful burning sensation. When hemorrhage is profuse, elongated blood clots are not infrequently found in the urine. *The testicle of the affected side is retracted.* As the pain increases in severity the patient rolls from side to side and shrieks with pain. His countenance becomes pale and the surface of the body is covered with a cold perspiration. The pulse is small and the hands and feet are cool. The severe paroxysms of pain are often attended by violent and frequent vomitings. There is great anxiety, and if the patient is of a very nervous temperament convulsions may occur. If the attack is prolonged there is a slight rise in the temperature. Syncope is common during the attack.

The duration of these attacks varies. Sometimes they are only of a few hours' duration, at other times they may be prolonged for days; again, temporary remissions may occur, followed by violent exacerbations.

As the calculus reaches the bladder the pain suddenly subsides, with a sense of relief, and the patient is often conscious of its passage into the bladder. After the passage of a calculus into the bladder it will soon be found in the urine voided. Occasionally calculi become impacted in some portion of the ureter. In such cases the subsidence of the pain is more gradual and less complete, and signs of hydronephrosis follow, and a tumor may be felt in the region of the kidney. By placing the patient on his back with his knees drawn up, the enlarged kidney may be pressed forward, and with the other hand in front it may be pressed backward and below the margin of the ribs. In the young and in those who are thin this method will aid very much in the diagnosis. Renal calculi may attain a large size and destroy extensive portions of the kidney, and yet not a single symptom may be present to indicate their existence. Again, the signs of renal calculi may exist for a long time, and finally atrophy of the kidney occurs, or they may become encysted and cease to give any indication of their presence.

Differential Diagnosis.—Renal calculi may be confounded with *neuralgia;* the seat of pain is the same, and the neuralgic pains are often severe and paroxysmal; but the urinary symptoms and an examination of the urine will make the differential diagnosis.

The passage of *blood-clots* or *hydatids* through the ureter, causing renal colic, cannot be distinguished from the passage of renal calculi, unless the antecedent history is known and appreciated, and a subsequent urinary analysis is made.

The irritation produced by an impacted calculus on the right side will not long be mistaken for *perityphlitis,* if careful and repeated examinations of the urine are made. Frequently, the abnormal conditions of the urine which indicate the presence of renal concretions are present only after violent exercise.

Prognosis.—The prognosis in renal calculus is good, unless the calculi become impacted and obstruct the ureter, or are of too large size to pass through the ureter to the bladder. In these conditions the prognosis is the same as in similar conditions in pyelitis, pyonephrosis and hydronephrosis. Should both kidneys be involved, the prognosis is exceedingly unfavorable.

Treatment.—Since the tendency to the formation of renal calculi is usually persistent and develops in patients of a gouty or rheumatic diathesis, the most important indication for treatment is found in the constitutional condition. All the directions given for the constitutional treatment of gout and the uric acid diathesis apply with equal force here, and there is reason to believe that the faithful use of alkaline waters not only delays, but often arrests, the formation of renal concretions. It is claimed that some of the natural waters exert within the body something of that solvent action upon calculi which they are known to possess under experimental conditions. Solutions of carbonate of soda, or, better, lithia or the Carlsbad, Vichy, or Ems natural waters, are among the more valuable agents. The American lithia waters are also efficacious.

The paroxysms which attend the passage of renal calculi, or so-called *nephritic colic,* must be relieved by the careful administration of morphine hypodermically, warm baths, and the application of hot poultices to the loins and abdomen. The danger of opium poisoning developing upon the sudden cessation of pain attending the passage of a calculus from the ureter into the bladder must be borne in mind. In most instances, when the pain is intense and the vomiting constant, inhalation of chloroform will be found to give the most speedy and sometimes permanent relief. Change in the position of the patient, and manipulation of the abdomen along the course of the ureters, may sometimes dislodge a calculus and facilitate its passage into the bladder. The treatment during the interval between the paroxysms which mark the passage of renal calculi will depend upon the changes which have occurred in the kidneys; the surgical treatment alone is applicable to cases of impacted calculus associated with pyelitis or pyonephrosis. The means to be employed for the relief of these conditions when not associated with obstruction of the ureter have been considered under Pyelitis.

NEW GROWTHS IN THE KIDNEY.

(*Renal Cancer.*)

Renal Cancer may occur as a primary or secondary affection. When secondary, its developments usually are of small size, and may occur in both kidneys. When primary, it is limited to one kidney, which soon forms an enormous tumor.

Morbid Anatomy.—Both primary and secondary cancer of the kidney are generally of the medullary variety, and develop in the form of circumscribed nodules in the cortical substance, or occur as a diffuse infiltration. The medullary cancer, however, may be nearly as hard as scirrhus. Colloid cancer is rare. It develops from the fibrous stroma of the cortical substance. Sometimes a whole kidney is transformed into a cancerous mass, which attains an enormous size, filling up a large portion of the abdominal cavity. The average weight of a cancerous kidney is over eight pounds; it has weighed thirty-one pounds in children. Secondary cancer (bilateral) never reaches a very large size. The kidney tissue is always intensely congested; it is often associated with cancer of the testicle.

The pelvis, ureters, the veins, the peritoneum, colon, and even the skin adjacent to the neoplasm may be involved. The lymphatics and adjacent glands are always enlarged. With the growth of the cancer all traces of renal structure become obliterated and the diseased organ becomes adherent to the adjacent tissue. Hemorrhages occurring in the mass at varying points give an appearance called "fungus hæmatodes." Sometimes a cancerous kidney is movable, no adhesions taking place with surrounding parts. The minute anatomical changes that take place in cancerous developments in the kidney, are similar to those which occur in cancerous developments in the other organs of the body.

Etiology.—The etiology of renal cancer is as obscure as the general etiology of cancer. In a large proportion of cases it depends either upon hereditary taint or local infection. Primary cancer occurs oftenest *before* the tenth and *after* the fiftieth year of life.[1] Secondary cancer may occur by continuity or from metastasis; *e. g.*, mamma, uterus, liver, stomach, testis or supra-renal capsules. Males suffer oftener than females; the right kidney oftener than the left.

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[1] Out of Rohre's 107 cases of primary renal carcinoma, 37 were under ten; 30 were over fifty years of age.

Symptoms.—Cancer of the kidney often remains latent for a long time. Its development is marked by gradual emaciation, for which no cause can be assigned. It may not be attended by pain in the lumbar region; if pain is present it is not characteristic. There may be no change in the renal secretion; but as the disease advances more or less profuse hemorrhages occur; sometimes the blood appears in the urine in clots, in which elements of the neoplasm may be found.

As the disease advances, and the cancerous mass reaches a large size, it can be felt through the abdominal walls. The form of the tumor and its immobility will enable one to distinguish it from enlargements of the liver or spleen. Very large cancers of the right kidney may displace the liver upward. The tumor is usually nodulated and firm, gives a dull or tympanitic note on percussion, and can be tilted forward. The colon lies in front of it. Aortic impulse may cause it to pulsate. When hæmaturia is present it is constant. In its advanced stage the countenance assumes the characteristic cancerous cachexia.

Differential Diagnosis.—Cancer of the *left* kidney is distinguished from *splenic tumors* by its lower site, absence of splenic notch, absence of blood changes, by its nodulated outline, and by hæmaturia.

From *perinephritic abscess* it is distinguished by absence of febrile symptoms, by its slow growth, and absence of fluctuation.

Cancer of the *right* kidney may be distinguished from *hepatic tumors* by an area of tympanitic percussion between the liver and the tumor. Reliance is also to be placed on signs peculiar either to hepatic or renal lesions.

Tumors of the *liver* or *spleen* are carried down *on full inspiration*; renal cancer is not. Fæcal and ovarian tumors have peculiar characteristics.[1]

Abscess and *hydatid* tumors are distinguished by introducing an aspirating needle, which withdraws either pus or a saline fluid containing portions of the echinococci.

An *ovarian* tumor when tapped is found to contain a peculiar ovarian fluid; fluid from a *hydronephrosis* contains some urinary elements.

Prognosis.—The prognosis is always bad. But cancer of the kidney is tolerated longer than that of any other organ. Death is reached either by the exhaustion produced by repeated and profuse hemorrhages, or as a consequence of some intercurrent disease, as parenchymatous nephritis in the unaffected organ. The lungs, retro-peritoneal glands, and liver may be secondarily invaded. A year in children and two years in adults is its average duration. Intestinal fistulæ may be formed, and the skin may be ulcerated, as sequelæ to cancer of the kidney. Dropsy may result from compression of the vena cava. The vertebræ may be eroded.

Treatment.—Its treatment is palliative. The principal things to be accomplished are to relieve pain by hypodermatics of morphine and to sustain the patient.

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[1] See "*Intestinal Obstruction.*"

Of the new growths met with in the kidney, cancer is the only one which has any special clinical significance.

Leukæmic tumors are occasionally met with as small whitish masses, developed in the intertubular tissue. They are composed of lymphoid cells, and are always associated with similar growths in the other viscera. These "*lymphadenomata*" are developed in connective-tissue; the liver is usually simultaneously involved.

Syphilitic gummata are also met with in the kidneys in the form of small nodules, in connection with similar developments in the other organs; cicatrices may be left, usually in the cortex, but sometimes in the medulla of the organ. Gummata destroy the tubules. Patches of "*fibrous tissue*" independent of gummata occur in kidneys of those who are syphilitic.

Fibromata may appear in the pyramids of kidneys in the form of small, white, fibrous nodules. The remaining portion of the kidney will be normal, or the seat of parenchymatous nephritis.

Lipomata include those accumulations of fatty tissue which are sometimes developed around the capsule of the kidney and in the pelvis of atrophied kidneys; sometimes in the cortical substance beneath the capsule small, rounded, fatty tumors are found. Growths of *bony, muscular*, and *glandular* tissue have also been met with in a few instances.

Sarcomata have been found in young children.

TUBERCULAR DISEASE OF THE KIDNEY.

Tubercles are developed in the kidneys as an advanced lesion of general tuberculosis. Primary tuberculosis of the kidneys is occasionally met with in young subjects.

Morbid Anatomy.—At first, gray miliary tubercles are found throughout the affected kidney, principally in the pyramids. The tubercles may originate in the stroma or in the cortex, in the arterioles separating the pyramids of Ferrein, or on the surface of the kidney. Later, solid, cheesy, yellow masses are found in the pyramids and in the cortex. The organ is enlarged and lobulated. The mucous membrane of the pelvis and ureters is thickened, infiltrated, and often ulcerated. When the ureter is involved diminution of its lumen may result in hydro- or pyonephrosis. The larger yellow masses are found at the junction of the cortex and medulla; they are usually softened at their centres, containing a puriform débris; the pelvis and calices are also dilated and filled with caseous pus or with a semifluid pulp rich in cholesterin. The tubules are compressed, and their epithelium undergoes granular and fatty change. An inflammatory process may coexist (*strumous nephritis*, of English authors), and the entire mucous membrane of the genito-urinary tract may be involved. Calcareous nodules and incrustations are found mingled with tubercle granules. Every portion of the genito-urinary tract, especially in the male, may show tubercle granulations.

Etiology.—Renal tuberculosis generally occurs in the young. Men are far oftener affected than women, the right kidney oftener than the left. It may occur as a primary tuberculosis, as part of acute miliary tuberculosis, or it may complicate chronic pulmonary phthisis.

Symptoms.—The symptoms are essentially those of pyelitis ; such as pain and tenderness in the loins, an irritable bladder, and scalding urine, which contains mucus, pus, and blood. As the disease advances, hectic fever develops, with the coexistent symptoms of intestinal or pulmonary tuberculosis. The urine will contain albumen (*no casts*), and under the microscope it is found loaded with fatty granules, lymph cells, blood corpuscles, and débris of connective-tissue infiltrated with small and fatty granular cells. A flaky, cloudy deposit always occurs in this urine, unless the ureter from the affected side is impermeable. If masses of cheesy material or bacilli are found, they establish the diagnosis. A renal tumor may sometimes be detected.

Differential Diagnosis.—The diagnosis rests upon the hereditary history, the presence of tubercles in lungs or prostate, on lymphatic enlargements, cheesy, puriform urinary débris, and the presence of a painful renal tumor.

Prognosis.—The prognosis is very unfavorable ; the complications are tubercle in any or all of the other organs of the body, cystitis, pyelitis, pyelo-nephritis, abscess, hydro- and pyo-nephrosis, waxy kidney, peritonitis, and urinary suppression.

The **Treatment** is altogether palliative.

PARASITES IN THE KIDNEY.

Renal parasites are occasionally met with ; the most frequent is the echinococcus. The cysticercus cellulosus, strongylus gigas, pentastoma denticulatum, distoma hæmatobium, spiroptera hominis, and dactylus aculeatus are parasites of rare occurrence. They are sometimes found embedded in the kidney. The symptoms which attend their development, and the manner in which they gain entrance into the kidney, are obscure.

HYDATIDS OF THE KIDNEY.

While hydatids of the kidney are less common than hydatids of the liver, the affection occurs under similar conditions.

Morbid Anatomy.—A kidney the seat of hydatids is sometimes enormously enlarged ; as a rule, a spherical cyst projects from the surface whose fibrous wall is derived from the kidney. The inner cyst wall may or may not be covered with daughter vesicles containing scolices, but a clear saline fluid always distends it ; the pressure of the cyst causes atrophy of the kidney structure. These cysts may suppurate and be changed into a shrivelled cyst with caseous contents in which are embedded echinococci hooklets. They may rupture into the perinephritic tissue and give rise to a lumbar abscess, or into the lungs, intestine, stomach, peritoneum, or pelvis of the kidney.

Symptoms.—A nephritic tumor is the first noticeable sign. A vesicle passing from the pelvis to the bladder gives rise to the symptoms of renal colic. An examination of the urine may reveal echinococci hooklets. In all cases the exploring trocar will withdraw a clear saline fluid containing hooklets.

Percussion may elicit the hydatid fremitus. If pus or blood appears in the urine it results from complicating inflammation or suppuration set up by the cyst or its contents.

Prognosis.—This is always uncertain. It is possible for a cyst to grow so rapidly as to cause death of the echinococci by pressure, or the fluid necessary to their life may be insufficient, or it may become so altered that calcareous changes will occur and then a calcareous mass may remain for life and cause no further harm. An echinococcus may be the nucleus of a stone in the bladder or in the pelvis of the kidney.

Treatment.—Aspiration should always be practised, and if it is not followed by adhesive inflammation, iodine should be injected into the cyst.

PERINEPHRITIS.

(*Perinephritic Abscess.*)

This is an inflammation of the connective-tissue surrounding the kidney: it may terminate in *suppuration*, or in the formation of fibroid tissue.

Morbid Anatomy.—The cellular tissue about the kidney becomes œdematous and the seat of inflammatory exudation, causing the cellular, adipose and adjacent retro-peritoneal tissues to become solid and firm. Suppuration may commence at the centre of the mass, leading to the formation of one large abscess; or, if it commences at numerous points and gradually extends, a number of circumscribed abscesses are formed. The tumor formed may become so large as to reach from the level of the liver or spleen to the iliac fossa, and may project forward and cause bulging of the abdominal wall. The pus contained in the abscess may be odorless, or thin, fetid and ichorous, especially if mixed with urine. The pus may have an odor of fæces *independent of perforation from the bowel into the abscess cavity.* This process may end in gangrene. The peritoneum over the tumor is thickened.

A perinephritic abscess may open into the lung, pleural cavity, or bronchi, by extending into the retro-peritoneal tissue and then through the diaphragm. The pus may burrow along the psoas muscle and appear as a psoas abscess on the thigh, or abdomen. Spontaneous opening usually occurs, externally, in the lumbar region. The bladder, ureter, pelvis of the kidney, peritoneum, and colon have all been perforated by perinephritic abscesses. Sometimes inflammation of the perinephritic tissue is not followed by suppuration, but at the autopsy a thick, tough, fibrous mass is found occupying the place of the (so-called) adipose capsule of the kidney. The same result may follow discharge of the abscess and cicatrization.

Etiology.—Perinephritis may be caused by pyelitis, suppurative nephritis, blows, falls, strains, parasites, or wounds of the kidney or the tissue about it. It may occur in pyæmia or in the course of any of the exanthems or specific fevers. It may also complicate pelvic cellulitis, psoas abscess, and perityphlitis. It occurs more frequently in men than in women.

Symptoms.—Recurring rigors are among the first symptoms, followed or accompanied by pain in the lumbar region—which is increased by movement and firm pressure—shooting down toward the testicle. The pulse is rapid and feeble. The temperature rises to 100°–105° F. The skin at first is dry, but later it is covered with a profuse perspiration. There is anorexia, great thirst, and constipation. The urine is usually slightly diminished in quantity; otherwise it is normal, unless pyelitis or nephritis should coexist.

Physical Signs.—A tumor forms in, or a little below, the lumbar region; it rapidly increases in size; at first it is hard; later it gives signs of deep fluctuation. The skin over it is œdematous and pale. The tumor is immovable and cannot be separated from the kidney, but can readily be distinguished from the spleen or liver enlargements. An exploring trocar will establish the diagnosis.

Differential Diagnosis.—The differential diagnosis between perinephritic abscess and *pyonephrosis* and *hydronephrosis* has already been given.

It is distinguished from *suppurative nephritis* by the presence of a tumor, and by the absence of casts, albumen, blood or mucus in the urine.

From extravasation of blood due to rupture of an *aneurism*, it is distinguished by fever, rigors, a *fluctuating* tumor, and the absence of the causes and physical signs of aneurism.

Prognosis.—A perinephritic abscess is always serious. Its duration is usually from two to four weeks; in some cases several months have elapsed before the tumor has subsided. Its discharge into the intestine or bladder, or the establishment of an external opening, may be regarded as favorable. With an early diagnosis and prompt surgical interference the prognosis is good. Some regard many "cures" of hip-joint disease without deformity, as in reality cases of suppurative perinephritis.[1]

Treatment.—A free opening should be made as soon as the diagnosis is established. Grainger Stewart states that early counter-irritation by blistering is useful, and that iodide of potassium internally and iodine externally may prevent suppuration; my experience does not sustain this statement. Yet incision is safer than aspiration; after an opening is made the finger should be introduced into the abscess-cavity and any adhesions that may be present should be broken down. Then a drainage tube should be introduced. Antisepsis should be practised during the operation and with subsequent dressings. Stimulants and concentrated fluid nutrition should be freely administered.

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[1] *Amer. Jour. Med. Sciences*, April, 1877, and Oct., 1878. V. P. Gibney, M.D.

FLOATING OR MOVABLE KIDNEY.

As a congenital peculiarity one or both kidneys may be movable, and instead of occupying their normal position may lie upon the brim of the pelvis, or be freely movable in the loose retro-peritoneal connective-tissue which surrounds them, and the peritoneum may be so reflected in front and behind them as to allow their free motion. The displacement of the kidney under any one of these conditions may follow parturition or a severe shock from a fall. It is met with more frequently in females than in males.

Morbid Anatomy.—A congenital displacement is distinguished from an acquired displacement by the abnormal arrangement of the vessels of the kidney and its peritoneal coverings. The extent of the mobility in any case is determined by the length of the vessels which form the pedicle. Movable kidneys are almost always surrounded by connective-tissue formations, and after having been once movable they may become firmly fixed again in their normal or in an abnormal position.

Symptoms.—A displaced kidney is usually felt midway between the free border of the ribs and the umbilicus. If the right kidney is displaced it is apt to make its appearance just below the liver; it may be pushed upward and backward into its normal position, but it will return as soon as the support is withdrawn.

If a displaced kidney can be grasped its pressure causes a sickening sensation. If it gets compressed or otherwise injured, it may become painful, tender, and swollen. Otherwise it may give rise to no symptoms and be recognized only by accident.

Differential Diagnosis.—Its diagnosis rests—1st, on the shape and size of a tumor corresponding to that of a normal kidney; 2d, when the tumor can be felt in front there will be an abnormal tympanitic resonance over the normal position of the kidney; 3d, the tumor can be pressed back into the normal kidney region; 4th, the peculiar sickening sensation produced by its manipulation.

Prognosis.—Such kidneys are never a cause of death. Many observers have doubts in regard to the probable occurrence of a floating kidney. There is little *post-mortem* evidence in its favor. I have never made but one diagnosis of this condition during life that was sustained by a *post-mortem* examination.

Treatment.—When a movable kidney is painful, rest is indicated, and a concave abdominal pad so adjusted as to fit the form and position of the kidney tumor should be worn.

HÆMATURIA.

Hæmaturia is the passage of urine containing blood. The blood may have its origin at any point from the Malpighian tuft to the orifice of the urethra. As it is a symptom, it has no morbid anatomy; its *causes* constitute its pathology.

Etiology.—*Local causes.*—(1) In the kidney the conditions which induce hæmaturia are active and passive hyperæmia, acute (rarely, if ever, chronic) suppurative nephritis, or surgical kidney, infarctions (including embolism and thrombosis), tuberculosis, a single or multiple pyæmic abscess, pyelitis (especially when the pyelitis is calculous), stone in the kidney, or in the pelvis of the kidney, and, in a few cases, hydro- and pyonephrosis. Crystals in the tubules may induce it. Among kidney causes may be included the drugs which cause hæmaturia, *e. g.*, turpentine, cubebs, copaiba, cantharides, etc.

(2) The causes that have their seat in the *ureters* are ureteritis, cancer, polypi, ulcers, and calculi.

(3) The *bladder* causes are cystitis (but only when very acute and accompanied by erosion and ulceration), cancer, abscesses in the vesical walls, polypus of the bladder, stone in the bladder, rupture of the bladder, tuberculosis, specific or non-specific ulcers. Dilatation and varicosity of the vesical veins may cause it, called oftentimes "hemorrhoids of the bladder."

(4) The *urethral* causes are many: urethritis (non-specific and specific), peri-urethral abscess, chordee, cancer, fracture of penis, rupture of prostatic abscess, an enlarged prostate, urethral polypi (especially in females), caustic injections, chancre and chancroids, phimosis, impacted stone, and new growths in the prostate.

The *general causes* of hæmaturia are acute infectious diseases, fevers, especially malarial, scurvy, purpura, the condition known as hæmophilia (the bleeders), and certain central nervous diseases (see Myelitis).[1]

Symptoms.—The urine may be almost black and loaded with clots, or it may be only slightly smoky or pinkish in color. It is albuminous; under the microscope swollen or shrunken corpuscles are found, the degree of alteration depending on the time they have remained in the urine. If equal parts of tincture of guiacum and oil of turpentine are shaken together to form an emulsion, an intense blue color will arise when bloody urine is slowly added to it.

To determine the source of the hemorrhages the following rules may be observed:—urethral hemorrhages are independent of micturition, as only a residue of blood is washed out at the beginning of the flow of urine. The history will aid and inspection will probably reveal the true state of affairs; albumen, casts and epithelial cells are not often found in urine when it becomes bloody in the urethra. The *bladder* may be suspected as the seat of the hemorrhage when blood flows only at the time of micturition, and follows the discharge of urine; should the stream suddenly cease, a stone or blood-clot blocks up the opening of 'the urethra into the bladder, and this will be well-nigh diagnostic. Clots following the flow of urine indicate cystic disease. When they precede the flow or occur with it, urethral disease is indicated. Should blood globules, albumen, casts, and blood moulded in the form of renal tubules be found in the urine, renal disease may be regarded as the cause of the hæmaturia. In renal hemorrhage blood is mingled with the urine, and is commonly as profuse at the commencement as at the end of micturition.

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[1] There is a variety of hæmaturia which occurs in tropical countries (Egypt, Brazil and Cape of Good Hope especially) caused by a fluke called Bilharzia hæmatobia, a parasite (a trematode hæmatozōon), which is endemic. Dr. John Harley discovered this parasite in the blood of a patient in South America. It is one-half to three-quarters in. long, and is found chiefly in the vessels of the portal system and of the bladder. The eggs are found in the urine; they are 1-100 to 1-180 in. in length, and are peculiarly pointed at one end, the whole contour, however, being ovoid. This parasite causes thickening, ulceration, ecchymoses and large blood extravasations in the mucous membrane in whose vessels it is lodged.

Should hæmaturia be combined with the symptoms of stone in the bladder, of pyelitis, or of cystitis, the source of the hemorrhage is then no longer a matter of doubt. Sir Thomas Watson states "that slender cylinders of fibrin in the ureter indicate renal disease or commencing inflammation of the ureter."

In "endemic" hæmaturia the diagnosis rests on discovering the trematode or its ova in the urine or fæces; it causes pain along and over the genito-urinary tract.

In the so-called false hæmaturia the urine contains only hæmoglobin, the microscope failing to discover any corpuscular elements in the urine. It is also called *hæmoglobinuria*, *hæmatinuria*, and (when occurring periodically), *intermittent* or paroxysmal *hæmaturia*. The hæmoglobin of the blood is set free in one of two ways: either the extravasated corpuscles disintegrate or the hæmoglobin escapes without rupture of the capillary walls. Once free in the blood the kidneys eliminate the hæmoglobin. Fevers, poisons, gases, and cold are said to cause this condition.

When intermittent it is usually dependent on malaria, but a malarial cause need not necessarily exist for the paroxysm to occur. Chills, sweatings, and, at times, a rise in temperature attend the discharge of the reddish urine, which soon shows a granular, brownish sediment. Albumen, and hyaline and granular casts are very often present, independent of renal disease. In severe cases the patient becomes anæmic and cachectic.

Quite recently a disease has been described called "melænic fever," resembling somewhat in its constitutional symptoms acute yellow atrophy of the liver and yellow fever. The urine is brown-black and contains albumen, casts, and a large quantity of blood corpuscles (not hæmoglobin alone). Suppression often occurs, and the case ends fatally.[1]

Differential Diagnosis.—The points of differential diagnosis have been sufficiently considered in its etiology. First, care must be taken by microscopical examination and spectrum analysis to positively determine that blood corpuscles or hæmoglobin are actually present in the urine. Then a study of its causes and accompanying symptoms renders the diagnosis comparatively easy.

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[1] *Virginia Med. Monthly*, February, 1880.

Prognosis.—The prognosis in hæmaturia depends on its cause. Endemic hæmaturia is never the direct cause of death, but it may lead to extreme anæmia. Paroxysmal hæmoglobinuria is rarely fatal.

Treatment.—When the hæmaturia is slight and of short duration no special treatment is required; if profuse or persistent the patient should be placed in a recumbent position, ice-bags applied over the seat of the hemorrhage, and hæmostatic remedies used, such as gallic or tannic acid, ergot, acetate of lead, and astringent ferric preparations. If the hæmaturia is of parasitic origin prophylaxis demands that the drinking water be filtered and boiled; to expel the parasites male-fern or chloroform may be given internally. Harley advises belladonna and henbane. Quinine is indicated in *all* forms of paroxysmal hæmaturia or hæmoglobinuria. If the hemorrhage is from the bladder persistent weak astringent injections may be employed.

CHYLURIA.

Chyluria is characterized by the occasional or continuous discharge of urine which resembles milk when passed and coagulates into a jelly mass on standing.

Morbid Anatomy and Etiology.—The kidneys are usually found free from disease, and the affection is attended by no known constant pathological lesions. At one time it was regarded as a disease of defective assimilation which permitted the chyle to mingle with the blood; at another, a fault of the kidneys which allowed the unchanged chyle to be transuded with the urine. Neither of these explanations has been sustained by observation.

There are at present many theories in regard to its causation: first, that there is a direct communication between the chyle-carrying vessels and the urinary tract; second, that it is a symptom of piarrhæmia due to a deranged liver function; third, that it is caused by an eczema along the urinary tract; fourth, that it is due to hypertrophy of the lymph channels and their subsequent assumption of glandular functions; fifth, that it is due to a parasite, but whether the action of the entozöon is on the function of the liver or causes irritation and rupture of the lymph and chyle channels is not determined.

Symptoms.—No disease pursues a more irregular course: no two cases exactly resemble each other. There may be pain in the loins and along the genito-urinary tract, depression of spirits, and debility, before the urine becomes chylous; or, the first sign may be a sudden flow of milky urine, having a whey-like or milky odor, made more perceptible by warmth. It soon coagulates on standing, but the trembling, jelly-like clot breaks down and the urine decomposes in a few hours. Bloody coagula, usually shreddy, may also form. White and red blood discs are found in varying quantity. Clots may form in the bladder, and during micturition the flow may suddenly *stop* from blocking of the urethra. The sp. gr. of the urine varies, (1.007–1.020). Heat and nitric acid cause a precipitate. Shaken with ether the urine loses its milkiness. Fat, albumen, and fibrin are all present.

Blood analyses vary; but when the filaria sanguinis hominis is not found in chylous urine it is found in a drop of blood taken from the finger, and *vice versâ*. Hoppe-Seyler says blood in this disease resembles human lymph in its composition. Chylo-serous discharges take place also from axilla, groin, scrotum, and surface of the abdomen or inner corner of the eye. Chyluria is an intermittent disease, but there is no periodicity or regularity to it.

Prognosis.—The disease runs a chronic course. Men have suffered on and off for fifty years. Change of climate does not seem to improve the outlook when the disease is once established. Sudden death may occur at any moment, even in those with fair health. Elephantiasis, phlebitis, hæmaturia, "lymph-scrotum," craw-craw, leprosy, and furuncles are not infrequent complications.

Treatment.—This has been unsatisfactory. Turpentine and gallic acid are recommended. Iodide of potash and perchloride of iron are claimed to be highly beneficial. Mangrove and nigella sativa are used by the natives in places where chyluria prevails; sometimes they effect a cure, oftener not, however. Prophylaxis demands care in drinking water in a tropical region, and first boiling or filtering it.

CYSTITIS.

Cystitis is an inflammation of the mucous membrane lining the urinary bladder. It is *acute* or *chronic*; and it may be either *catarrhal*, *croupous*, or *diphtheritic*. The whole or part of the bladder may be involved; when "partial," it is limited to the neck and *bas-fond*.

Morbid Anatomy.—In acute catarrhal cystitis the appearances are in no wise different from those observed when any mucous surface is inflamed. The small glands at the base of the bladder are enlarged and filled with a pearly secretion. The interior portion of the trigone is also studded with these pearly masses. They may form a circle about the neck of the bladder. Intense (acute) cystitis may end in suppuration of the submucous connective-tissue, and ulceration of the mucous membrane may allow these submucous abscesses to empty into the bladder.

When cystitis results in paralysis of the bladder, gangrene of the mucous membrane may occur; then brownish-black, irregular patches are seen mingled with débris and phosphatic incrustations on the surface of the bladder. When the mucous layer is thus destroyed by gangrene, the urine infiltrates the neighboring tissue, and local or general peritonitis may result. An acute cystitis may lead to a pyelo-nephritis. Ulcerating cystitis occurs in typhoid and low eruptive fevers, in diphtheria, pyæmia, etc. It is called by some diphtheritic. The lesions in this form and in croupous cystitis are similar to those which take place in diphtheritic exudations on other mucous surfaces. (*See Inflammation.*)

In *chronic cystitis* the mucous membrane is thick, blue-gray in color, and very tough. Muco-pus and viscid mucus are formed in large quantities upon its surface. As the disease progresses a peri-cystitis consolidates the bladder with the neighboring organs and parts. Chronic catarrhal ulcers may form, and perforation of the bladder may result, and the vagina, rectum, or abdominal cavity may be entered, or an external opening may be formed through which pus is discharged. The muscular wall of the bladder may sometimes be half an inch thick, and the fasciculi give a ribbed appearance to the internal surface, called the "columnar bladder." The hypertrophy of chronic cystitis may be eccentric or concentric. In some cases diverticuli are formed, in whose walls are dilated and tortuous veins. Some of these cysts are in the form of hernial protrusions. In nearly all cases bacteria are found in abundance.

Etiology.—Acute cystitis is rarely idiopathic.

It may result from the presence of foreign bodies, especially calculi. Blows may cause it. Protracted retention of urine has set up a rapidly fatal cystitis.

It may be caused by some unknown blood condition, such as occurs in scarlet, typhus, and typhoid fevers, pyæmia, septicæmia, small-pox, and diphtheria; it is a frequent complication of certain grave lesions of the nervous system, especially myelitis.

Cystitis may result from the extension of an urethritis, a pyelitis, or a pelvic cellulitis.

Chronic cystitis may be the sequela of acute cystitis or result from the retention of urine caused by an enlarged prostate or urethral stricture. Over-distention, atony or paralysis of the bladder, calculi, polypi, and neoplasms of all kinds cause it. Gout and some forms of kidney disease are accompanied by chronic cystitis.

Symptoms.—Acute cystitis is always accompanied by frequent micturition, only a few drops being voided at each attempt. After its passage the patient strains (as in the *tenesmus* of dysentery) to pass what he imagines is still retained in the bladder. There are dull, aching pains over the pubis; sometimes the pains in the vesical region are agonizing, and there is a constant burning sensation along the urethra. These local symptoms are not infrequently accompanied by rigors, and the temperature rises to 100°-101° F., with loss of appetite, sleeplessness, and a feeling of great anxiety or depression.

The urine is cloudy, deposits mucus on standing, is alkaline, and *sometimes* fetid. *Microscopically*, epithelium, pus and red blood-corpuscles are found. Membranous exudations may be found, especially in females. Niemeyer states that in the "croupous cystitis following cantharides poisoning and forcible forceps deliveries large tenacious false membranes are discharged" with the urine.

Chronic cystitis is often only indicated by a frequent desire to pass urine.

Usually there is a constant, dull, aching pain, or a sense of weakness over the bladder. The bladder is nearly always intolerant of its contents, no matter how long the catarrh has persisted. Hence only a small amount of urine will be passed with each act. Distention and muscular hypertrophy of the bladder often give rise to an abdominal tumor reaching as high as the umbilicus ; it may contain from two to eight pints of urine ; as large a quantity as this, in some cases, may constantly remain in the bladder, only so much urine being passed as exceeds this amount, and then a patient will be passing very nearly a normal quantity, and the introduction of the catheter may remove a quart of stinking, alkaline urine, which, when it stands, divides into two parts, a lower thick, turbid, gelatinous, coherent and opaque mass—the supernatant layer being clear. The "glairy mucus" so frequently described in this connection is only met with when the urine is ammoniacal and also contains pus ; it is formed by the reaction of the alkali upon the pus. Chronic cystitis accompanied by enlargement and atony of the bladder often eventuates in ammonæmia, and then typhoid symptoms are developed. Great local pain, emaciation and occasional bloody urine indicate ulceration. Acute suppurative inflammation of the bladder, accompanied by hectic, rigors, and extreme exhaustion, may accompany acute suppurative nephritis.

Differential Diagnosis.—*Pyelitis* often resembles cystitis closely in its subjective symptoms ; there may be the same pain referred to the bladder, and the same frequent desire to micturate. In pyelitis the lumbar pain, the "tailed" cells in the urine, the even admixture of pus with the urine, the acid reaction, and the absence of ropy, gelatinous mucus, are symptoms in marked contrast to those of cystitis.

Prognosis.—The prognosis depends upon the cause ; in general it is good. Chronic cystitis may continue for years ; the longer it continues the less chance there is of recovery. Acute cystitis is usually recovered from in about a week.

Treatment.—In *acute* cystitis the patient must have perfect rest. Warm hip-baths give relief. Leeching or cupping over the bladder is often of service. Suppositories of opium and belladonna, or rectal injections of the same, are always indicated, with large poultices and very hot fomentations over the bladder. The bowels should be kept free with the *mildest* cathartics. An anodyne internally may be demanded for the relief of pain ; I have found chlorodyne the best. Twenty minims of liquor potassæ in mucilage may be given three times in the twenty-four hours. Half-drachm doses of fluid extract of Indian hemp are highly recommended. The diet should be nutritious ; milk is to be preferred. No form of alcohol should be allowed ; the patient may drink freely of flaxseed or linseed tea, barley water, or decoction of *triticum repens*. In all cases the cause should be sought for and if possible removed.

In *chronic* cystitis the catheter is to be regularly and persistently used. The bladder should be washed out with weak solutions of borax. Very weak solutions of salicylic acid, carbolic acid, permanganate of potash, and chloride of sodium are also recommended. The daily use of a mineral water, like Vichy, is beneficial in many cases of chronic cystitis. I have found great benefit from the daily use (drachm doses after each meal) of the "Lafayette mixture."[1] All stimulating drinks are forbidden. The injection of quinine into the bladder has recently been very successfully practised for the cure of chronic cystitis.

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[1] ℞ Bals. copab.
Spts. eth. nitros. āā .... ℥ss.
Liq. potass.. ʒi.
Mucilag. acaciæ ....ad ℥iv.
M.

SECTION IV.

ACUTE GENERAL DISEASES.

Modern pathology has demonstrated the specific nature of some pathological processes and their dependence upon the entrance and development within the system of micro-organisms.

The pathological and clinical resemblances which many diseases bear to conditions known to be of bacterial origin are so numerous and striking that we are compelled to believe that they also depend upon a similar cause. All these conditions are classed under the general term of Infectious Diseases. In the present state of bacteriological study the proper classification of some diseases is still unsettled. I shall include in this section only such as are generally accepted as of undoubted bacterial origin. A few, on the other hand—as acute hydrocephalus and pneumonia—whose specific nature is established, are better described in connection with other diseases of the organs which they principally affect. While not scientifically accurate, such a classification is the one generally adopted.

Although the specific bacteria of comparatively few of the infectious diseases have been isolated and recognized, they all present, clinically, certain well-defined class characteristics. *First:* Their pathogenic action is specific and invariable, so that, under similar circumstances, the pathological processes which any particular germ incites are always identical in kind, although several germs may have the power to produce a given morbid condition. *Second:* They possess the power of indefinite reproduction when placed under favorable circumstances, and their resulting diseases are therefore often *endemic* where permanent sources of infection have become established, or *epidemic* when the poison affects large numbers at the same time, rather than sporadic. The infective element in all infectious diseases, whether it be the bacterium itself or its ptomaïne, is termed a virus, and is always developed, so far as we know, either in the bodies of diseased living beings or in connection with vegetable organic matter.

Every virus is more or less diffusible and may be conveyed by air, fluids, or solids; while in some diseases it becomes so localized that it can be transmitted by inoculation. These morbific agents give rise to distinctive diseases either by changes which they produce in the blood or by their direct action upon the cellular elements of the different organs and tissues.

When a virus originates and attains its full development only in a living animal, and is excreted in an *active* state, it is called a *contagion,* and the disease which it produces is contagious.

of ganglionic centres in the medulla, and recent experiments have determined with almost equal certainty that heat production, as it depends upon oxidation in the tissues and glands, is regulated by a thermogenic centre. The exact location of this centre has not been definitely determined, but all investigators agree in placing it above the medulla, thus differentiating it from the vasomotor centre.

It is believed that this centre acts both by stimulation and inhibition of oxidation.

Etiology.—The demonstration of thermogenic centres seems sufficiently conclusive to warrant the assumption that fever is always primarily dependent upon some form of irritation of several correlated nervous centres, the thermogenic, vasomotor, and perspiratory being the most important. Setting aside physiological conditions, such as functional activity of glands and organs, which are attended by rise of temperature, we find that clinically the causes of fever may be classed as :

I. *Nervous:* under which are classed conditions mechanically affecting some portion of the nervous system unassociated with inflammatory changes.

II. *Hæmic:* including all those conditions in which irritating elements are known or supposed to be present in the blood.

Nervous Causes.—Both direct and reflex irritation of the nervous centres are known to produce rise of temperature without coincident inflammation. A temperature of 120° F. has been reported in a case of fracture of the spine which ended in recovery. Cerebral tumors affecting the pons, optic thalami, or crura cerebri, or hemorrhages in the same locality, are often attended by temperatures reaching 105°–106° F. The marked fall of temperature which is at times associated with these conditions gives equally strong evidence of the relations which these centres bear to the body temperature. The variations of temperature attendant upon irritation of peripheral nerves, even when they form part of a state of *shock*, will bear a similar interpretation.

Hæmic Causes.—Those pyrogenic elements which are found in the blood are at present divided into four classes :

First: Elements developed by perversion in the nutritive processes. This perversion may be in the primary digestion ; in secondary metabolism ; in the retrograde changes or in excretory functions, and results both in the production of new and the accumulation of normal poisonous elements in the blood.

Second: Leucomaïnes ; poisons developed from inflammatory and exudative products as already described.

Third: Ptomaïnes ; or the bacterial elements with which they are associated.

Fourth: Chemical, mineral, or vegetable poisons, as alcohol, opium, etc.

The infectious fevers are believed to depend upon the third class of hæmic poisons, whose action, therefore, require special consideration.

The development of ptomaïnes by bacterial growth has already been considered, but the exact relations which bacteria and their poisonous products bear to pathological change are not fully determined. It seems probable, however, that in most cases the ptomaïne is the active poison, although many bacteria do penetrate the tissues and are found in the blood and substance of organs. The question is an important one as regards prognosis and treatment, since destruction of living germs in the body can hardly be hoped for without severe injury of the tissues, while neutralization of chemical elements in the blood is known to be possible.

At present we are ignorant of the exact manner in which these poison elements excite those metabolic processes which are productive of fever. The existence of a thermogenic centre does not preclude the possibility that bacteria or their ptomaïnes may act directly upon parenchymatous elements to increase metabolism, unless we assume at the same time that all protoplasmic action is under the control of the nervous system, a supposition which I am inclined to accept, although it is as yet unproven. Many clinical facts, and the action of some drugs as antipyretics, also lend support to such a supposition. The term *thermogenic*, however, must not be considered in this connection as implying anything more than a nerve centre which controls, either by way of stimulation or inhibition, those metabolic processes in the tissues which result in heat production.

The general drift of opinion at present is toward the belief that pyrogenic elements act primarily upon such a centre, to produce fever; and secondarily upon the tissues, through perversion of the nutritive elements of the blood, to cause degenerative changes.

Results of Fever.—All the infectious diseases are characterized by more or less extensive changes in the blood and tissues. The fibrine factors and albuminous elements are decreased; the white cells are increased; the corpuscles swollen, crenated, or even broken down, and their pigment thrown into the serum, while parenchymatous elements throughout the body suffer varying degrees of cloudy swelling, granular or fatty degeneration.

Formerly all these changes were supposed to be the direct result of the high temperature, and other causes to have little if any connection with their development. Although our knowledge upon this point is still unsatisfactory, it is now more generally believed that the hæmic changes are the direct result of the action of the poison element upon the blood, while the parenchymatous degeneration depends upon decreased or perverted nutrition and the excessive metabolism which causes the increase of heat. Those cases, however, in which extreme degenerative changes are associated with low temperature or are developed with extreme rapidity, suggest a more direct action of the poison factors upon the tissues themselves.

Such a view does not entirely exclude the increase of body temperature as a factor in the causation of these degenerative changes. The effects of high temperatures upon the nervous system also appear to have been overestimated. It seems determined, both by clinical observation and physiological experiment, that there is a point at which increase of body temperature causes collapse of nervous force and speedy death ; but such temperatures are seldom attained in the specific diseases, notwithstanding the decreased resisting power which we must assume the nerve centres possess, in common with other tissues, under the influence of the systemic poison.

Direct irritation of the nervous tissues, by the specific poison or its products in the blood, is more generally regarded as the important element in the causation of those sensorial disturbances so characteristic of the specific diseases.

Febrile temperatures of moderate intensity, not exceeding, say, 105° F., have thus come to occupy a position of secondary importance with most clinicians, among the unfavorable symptoms of disease, while many careful observers even regard fever as purely a conservative process.

Treatment.—Our treatment of fever, *per se*, will vary with our views as to its dangers. To my mind there is little if any evidence that a temperature of 106° or 107° F. causes any serious injury to the body, nor do I recognize any necessary relation between the other febrile symptoms and the temperature range. Those who follow the teachings of Liebermeister, and believe that all the grave symptoms of febrile conditions are referable to the high temperature, will employ antipyretics early and persistently in most diseases, and endeavor to maintain a temperature range below 103° F.

A temperature of 105° F., however, has come to be quite as generally regarded as the indication for the use of antipyretic measures as was 103° F. formerly. The temperature range, however, is not to be the sole guide to the use of antipyretics. If a temperature of 105° or 106° F., in a case of fever or pneumonia, is rapidly reduced by mild antipyretic measures, the patient at the same time being made more comfortable, with decrease in the nervous symptoms, I should most certainly recommend their employment. But when powerful measures have but a slight effect upon the temperature, their use were better omitted.

Many authorities still insist, however, upon the employment of extreme antipyretic methods in certain diseases, who would not extend this treatment to febrile processes in general. They must believe either that the effects of high temperature are not constant, or that the methods of temperature reduction have some other effect than simply to decrease the body temperature.

Antipyretics are of two classes : 1st. External applications, which are known to abstract heat from the surface, and thus decrease the actual body temperature. Clinical facts suggest that such applications also have a reflex action upon the nervous system, not only acting as a sedative to nervous irritation, but also tending to restore nervous control of the thermogenetic processes in the tissues. It is certainly true that a cold bath will often produce a greater and more prolonged reduction of temperature than can be accounted for simply by heat abstraction. This sedative nervous effect is a more valuable guide in their use than the temperature range alone. When external abstraction of heat is followed by decrease of nervous irritability, as evidenced by decrease of muscular twitchings, cessation of delirium, clearing of the mind, or the advent of sleep after prolonged wakefulness, one may be assured that the applications are of value, whatever has been the effect upon the temperature. When, on the contrary, the opposite condition follows, with evidences of nervous shock or irritation, no reduction of temperature which they may accomplish can justify the continuance of such measures. Unfortunately, only trial can show what patients will be benefited and what injured.

The means employed for abstracting heat are baths, packs, spongings, ice-bags, and the coil. Of these, the sponging may be done with ice-water in extreme cases; the pack wrung from water at 60° or even 40° F.; while the bath is best started at a temperature of 65°–70° F., which may be gradually or rapidly reduced to 50° or 40° F., according to the effects obtained. Cold spongings may be repeated every twenty minutes to half an hour until the desired reduction is obtained. Cold packs should be renewed as soon as they cease to give a sensation of coolness to the patient, and this repetition kept up until the same result is gained. When a patient is put in a bath, its duration is determined by its effects. If the skin becomes blanched, the face pinched, the pulse disturbed in force or rhythm, and the respiration impeded, he must be removed at once, and reaction established by the usual means. If, however, the bath is grateful, if the pulse is quieter and the circulation well maintained, the bath is continued until the patient's temperature is 101° F. if it falls rapidly, or 100° F. if it comes down very slowly. On removal, reaction in the cutaneous circulation should be obtained as before. When successful, such baths may be repeated as often as necessary to maintain the desired temperature. Ice-bags are solely for local abstraction of heat, and are now entirely superseded by the coil when it can be obtained. While intended more particularly for local use, the persistent application of a large cold coil will exert a decided effect upon the general temperature, and in many cases will render the more difficult applications unnecessary.

2d. Internal antipyretics include all those substances which have been found capable of reducing body temperature when taken into the system. Unfortunately we have no rules to govern their use except those gained by clinical observation. In the light of our present knowledge of fever, it seems probable that these remedies may be of two classes: those which act primarily on the thermogenic centres; and those which neutralize or in some manner render inert pyrogenic substances in the blood. At present, however, we have no definite knowledge upon this point. Clinically, we know that in different conditions different antipyretics have unequal therapeutic values, and the above suggestion simply offers an explanation n accord with our present ideas regarding fever. Another decade may prove them all wrong. The more important antipyretics are quinine, antipyrin, antifebrin, salol, salicylic acid, phenacetin, opium, and other sedatives. Other remedies have been employed, but are all represented by these given. Substances which stimulate the flow of bile are also regarded as antipyretic in diseases where intestinal absorption is an element in the production of fever.

There can be no question as to the superiority of quinine in all malarial fevers, whether the malarial poison be the sole cause or only one factor in the etiology. Quinine is also generally considered preferable in surgical fever attended by suppuration, and in septic conditions, or simple inflammatory fever. At present antipyrin and antifebrin are the favorite antipyretics in the specific fevers and conditions where nervous irritation is prominent. Personally I prefer antifebrin. Salol and the salicylates are employed rather for their primary antiseptic action upon the intestinal contents, and only secondarily as antipyretics. Phenacetin is not so favorably regarded as the others of this class. Opium and sedatives probably act as antipyretics simply through their effects upon nervous irritation. They are used more in combination with other more active remedies than alone. Finally, it must be repeated that the above statements are to be taken in the most general manner, and that experience alone can determine which antipyretic is most suitable to any given condition.

TYPHOID FEVER.

This is the most prevalent of all fevers except malarial. So far as we know, there is no place where it may not be developed and spread. It more frequently prevails in the temperate zones than in the torrid or frigid, but it is possible for it to be developed in all latitudes and in all countries. This disease, which is essentially the same in all countries, is designated by different names. American writers describe it under the name of *typhoid fever.* The French call it the *typhoid affection,* or *dothinenteria.* English writers describe the same form of disease under the head of *enteric fever.* The Germans call it *abdominal typhus,* or *gastric fever.* I prefer the name typhoid fever.

Morbid Anatomy.—As soon as the disease is fully established a change in the *blood* occurs. It becomes darker in color, coagulating imperfectly, and the serum is of an unnaturally yellow color. The question arises :—did these changes take place in the blood prior to the occurrence of the fever, between the exposure and the period of attack? It is certain that as soon as the characteristic symptoms of the disease are present, the diminution in the fibrin of the blood is in exact proportion to the severity of the fever, and the number of white globules is increased in a similar ratio.

In connection with these blood changes, a series of changes take place in those organs and tissues of the body in which the process of waste and repair are most rapidly going on. They are of the nature of parenchymatous degeneration, the essential constituents of the affected organs and tissues being involved. Similar parenchymatous changes are met with to a greater or less extent in other acute infectious diseases.

Spleen.—The organ in which parenchymatous degeneration occurs earliest and most extensively is the spleen. We find this organ undergoing three distinct changes :—

First. It is increased in size, sometimes enormously. The enlargement commences soon after the beginning of the disease, and goes on rapidly until the third week, after which it ceases, and within a few days begins to diminish. If recovery takes place, by the time it is reached, the spleen will have returned to its normal size. The splenic enlargement is apparently due to congestion and to an increase of normal elements.

Second. As soon as the spleen reaches its maximum size, its consistency diminishes, and this softening is sometimes so marked that, if a post-mortem be made at the end of the third week, it will present the appearance of a dark, jelly-like mass, which is easily broken down.

Third. The organ becomes almost black in color, owing to the intense congestion which attends its enlargement, and to the deposit of a brown pigment in its substance. These changes in the spleen take place, in a greater or less degree, in ninety-eight cases out of every hundred. At the post-mortem of those who have died of typhoid fever infarctions are sometimes found, although there is nothing peculiar about them. In rare instances, rupture of the spleen occurs without infarctions.

Liver.—Changes in the liver are by no means as common as those in the spleen. The liver may be found presenting its normal appearance, or it may be soft and flabby. When soft and flabby, a microscopic examination shows the liver cells more or less granular and fatty; the nuclei of the cells can no longer be seen, and the degeneration may become so extensive that the outline of the hepatic cells is lost, and nothing but a mass of granules remains. Occasionally there will be found in the liver small grayish nodules situated along the course of the small veins; these bodies consist of lymphoid cells. The lining membrane of the gall-bladder sometimes presents evidences of catarrhal or diphtheritic inflammation, when there has been no evidence of its existence during life; cases are recorded where it has been found ulcerated.

Kidneys.—Degenerative changes in the kidneys are of not infrequent occurrence in the course of typhoid fever; they vary in extent with the duration and severity of the fever. When present, they are more marked in the cortical than in the medullary portion of the organ. In some cases they are confined to the epithelial elements, while in other cases degeneration of all the anatomical elements of the organs can be found. Such extensive changes are less liable to occur in typhoid than in typhus fever. Small gray nodules, similar to those referred to as occurring in the liver, are sometimes found. If the epithelial degeneration of the cortical substance is extensive, the cells finally break down into a granular detritus, and the cut surface assumes a yellow color and is softer than normal. Infarctions are sometimes met with in the kidneys of those dying of typhoid fever.

Heart.—The parenchymatous changes which take place in the heart are more marked than those in any other organ except the spleen. In a large proportion of cases it becomes soft and flabby, and is of a grayish or brown color. Sometimes it is so much changed that its tissue is easily broken down by moderate pressure; it loses its normal outline, and when removed from the body the walls of its cavities readily fall together. When its muscular tissue is examined microscopically, in many instances it will be found that granular changes, affecting the ultimate muscular fibres, have occurred; this granular muscular degeneration may be general or local. Occasionally the muscular fibres are infiltrated with brown pigment. If, as is sometimes the case, the heart retains its normal outline, is friable, and its cut surface glistens, the muscular fibres will be found to have undergone a change which closely resembles amyloid degeneration; they will be filled with a material which presents the same shining appearance as the amyloid substance, but on applying the iodine test the amyloid reaction does not take place. It is a form of degeneration which is not confined to the muscular tissue of the heart, but is found to a greater or less extent in the voluntary muscles throughout the body. Thrombi are sometimes found in the heart, and vegetations adhering to the valves and chordæ tendineæ. These may give rise to infarctions in the different organs. The existence of these degenerative changes in the heart may be recognized during the life of the patient, for the heart-sounds become feeble according to the extent of the degeneration, and in some cases the first sound of the heart will be absent.

Lungs.—The lungs undergo changes which have received the name of splenization, from the close resemblance which the affected portion of lung then bears to the spleen. The affected tissue is of a darker color than normal, and scattered through its substance will be seen minute red or yellowish-white points; these points are scanty blood extravasations. It is of a reddish-blue, brown, or black color; its consistence is firmer than normal, it crepitates less freely, has a more homogeneous appearance upon its cut surface, and is less moist than normal lung-tissue; a dark fluid will sometimes ooze from its cut surface, but not as freely as in hyperæmia, and the fluid is more watery in appearance. A microscopical examination of lung-tissue in this condition shows the capillary vessels filled with blood, and the alveoli containing a variable number of cells. It is a condition closely resembling that condition known as static pneumonia, but no inflammatory process exists; it is simply a stasis in the capillary circulation, accompanied by a slight increase in the cell elements in the alveoli.

So constantly is catarrhal bronchitis present in this fever, that Dr. Stokes proposed to call typhoid fever *bronchial typhus.* In most cases this catarrh is not extensive, but affects only the larger bronchi; it may, however, extend to the smaller tubes and give rise to capillary bronchitis and broncho-pneumonia. Pulmonary infarctions are frequently found in the lungs of those who have died of typhoid fever. They are sometimes quite numerous, are usually of small size, and vary in appearance according to the stage of their development. When recent they are of dark color, and feel like consolidated lung-tissue; later, the color changes to yellow; they may soften and break down.

Larynx.—The larynx, as well as the bronchial tubes, is frequently the seat of catarrhal inflammation; less frequently it is the seat of diphtheritic inflammation. In connection with these laryngeal inflammations, ulcers appear in the larynx; these have received the name of "typhoid ulcers of the larynx:" sometimes they give rise to quite extensive hemorrhages. In connection with, or independent of, these laryngeal ulcers, ulceration of the mucous membrane of the mouth and pharynx may occur; at times it involves the epiglottis in such a manner as to clip off its edges. These ulcers may develop on the mucous membrane of the Eustachian tubes. In those cases where permanent deafness follows an attack of typhoid fever, it will usually be due to ulceration of the mucous membrane of the Eustachian tube.

Brain and Nervous System.—As yet we have not been able to determine whether there are any structural changes in the brain or nervous system so constant that they may be regarded as lesions of typhoid fever, although it is reasonable to infer that in a disease where such severe functional disturbances of the cerebro-spinal system exist there must be constant and definite parenchymatous changes. Œdema of the pia mater and of the brain substance, with occasionally quite extensive adhesions of the dura mater to the cranium, not infrequently exists. Punctate extravasations into the brain substance are found in a certain number of cases, but even in severe cases they are not always present.

Stomach.—The changes which occur in the stomach are equally important with those which occur in the other internal organs, and are degenerative in their nature. Softening and degeneration of its glandular structure are sometimes so extensive that, if recovery from the fever takes place, a very long time must elapse before the organ can perform its normal function. It is the existence of these degenerative changes that gives rise to the disturbance in digestion which is present in so many cases, not only during the continuance of the fever, but during convalescence.

Muscles.—Muscular degeneration is of two varieties :—*first*, a granular degeneration, which corresponds to ordinary fatty degeneration. *Secondly*, a waxy or vitreous degeneration, which consists in the conversion of the contractile substance of the primitive bundles into a homogeneous, waxy shining mass. ·Often both forms of degeneration occur together, one or the other predominating. In both forms of degeneration the muscular fibres become thicker and more brittle than normal. In the highest degree of degeneration the muscular fibres are entirely lost, and the muscle may present a yellowish or whitish appearance, so that hardly any traces of its normal color remain. This muscular degeneration, however, is not peculiar to typhoid fever, but is met with in all severe infectious diseases. The want of muscular power, which is so prominent a symptom during the height of the fever, may depend on the disturbances of the nervous system, but the excessive loss of muscular power which is so often present during convalescence is due almost entirely to the muscular changes. The physical strength returns gradually during convalescence as the muscles are regenerated, and it may be months before it is fully re-established. The muscles of the tongue undergo degeneration in the same way as the other voluntary muscles, which accounts in some degree for the interference with the function of that organ, so often a prominent phenomenon of the disease.

The *salivary glands* enlarge, become firm and tense, and assume a more or less brown-yellow color. They have the consistence of cartilage. Late in the disease the hardness diminishes, and they assume a red color. These changes are due to a parenchymatous degeneration, which has been preceded by a cellular hyperplasia. It accounts to a certain extent for the diminution of the salivary secretion, which is so marked and constant an attendant of the fever. Similar cellular and parenchymatous changes take place in the pancreas. Changes similar to these occur in other febrile diseases, so that they cannot be regarded as characteristic of typhoid fever.

Intestines.—The essential and characteristic lesions of typhoid fever are found in the lymph structures of the intestines. They vary only in degree and not in character with the duration of the fever and their proximity to the ileo-cæcal valve. Although changes closely resembling them may be present in other diseases, there is no other disease in which they follow a regular course of development, with stages limited by days and weeks. These changes in typhoid fever correspond very closely in their different stages with the four weeks of the disease. During the first week they are confined to a catarrhal inflammation of the intestinal mucous membrane, most marked about the Peyerian patches, with a medullary infiltration of these and the solitary glands, which extends in some cases into the adjacent tissues. The infiltrated cells are mostly lymphoid cells, though large, round and polygonal cells with multiple nuclei are also present. These latter are swollen epithelial cells from the reticulated tissue of the mucous membrane and lymph follicles. As a result of these processes, there is hyperæmia and swelling of the mucous membrane, and the affected glands become enlarged and elevated from one to two lines above the mucous surface. They assume a dark red or reddish-gray color marked with fine white striations, and present the so-called "shaven beard" appearance. Their consistence varies with the severity of the process. When moderately swollen, they are soft and present a spongy appearance, but in the severer types the entire gland becomes hard and smooth. These changes begin and are most extensive in the glands nearest the ileocæcal valve; they are generally well marked within forty-eight hours after the commencement of the disease, but are not fully developed until the end of the first week, when all the glands are involved which are likely to undergo change. The number of patches involved varies from four to five near the valve to twenty or thirty throughout the whole intestine. The solitary follicles do not participate in the infiltration and swelling to the same extent as the agminated glands.

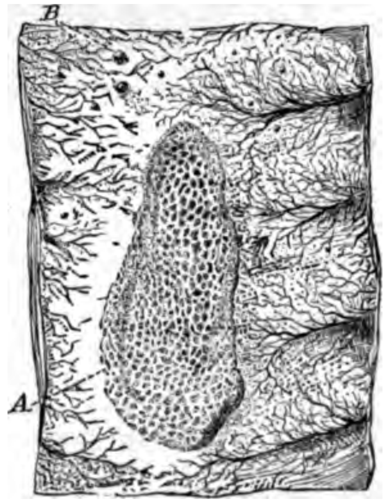


FIG. 151.

Mucous surface of a portion of the Ileum during the first week of Typhoid Fever.

A. Peyer's patch, showing the reticulated or shaven-beard appearance.
The mucous membrane is hyperæmic, and the solitary follicles, B, are only slightly involved.

In the *second week* the hyperæmia and catarrh of the mucous membrane subside, leaving the agminated and solitary glands more elevated; the white lines upon their surface disappear, and they assume a uniformly red color. An unusually rapid cellular hyperplasia takes place in the follicles, by which they become swollen in all directions. Usually the new cell growth extends beyond the limit of the follicles, so that the adjoining mucous membrane is also infiltrated with cells. The new cells distend the glands, and a vertical section of a patch in this stage shows the villi increased in length and width, and fused together at their bases or throughout their entire length by the embryonic tissue. These newly formed cells may wander through the muscular coat or penetrate the sub-serous tissue. Thus hyperplasia of the adenoid tissue is the essential pathological change in the second week. By the middle or latter part of the second week the process passes into its third stage, and necrotic changes are established in the newly formed tissue.

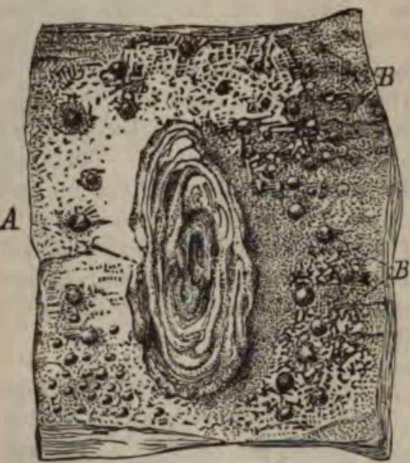


FIG. 152.
Mucous surface of the Ileum during the second week of Typhoid Fever.
A. Peyer's patch thickened and raised—from hyperplasia.
B. Solitary follicles much enlarged.

These morbid changes may terminate in three ways: first, the new elements in these ductless glands may become disintegrated and undergo absorption, and in this way they may gradually undergo resolution; second, either the tissue between the follicles remains infiltrated and elevated while their contents are absorbed, or individual follicles of the agminated glands rupture and discharge their contents into the intestine, leaving depressions which give the gland a reticulated appearance; third, the most frequent and characteristic termination of the typhoid process is the separation of the dead tissue as a slough, or by ulceration and the formation of the typhoid ulcer. Ulceration which begins at the most elevated portion of a patch, already stained yellow or yellowish green by intestinal fluids or darker by sanguineous infiltration advances gradually until a small irregular ulcer has enlarged to one covering the whole gland, or the entire gland may slough uniformly, and at once form the complete ulcer.

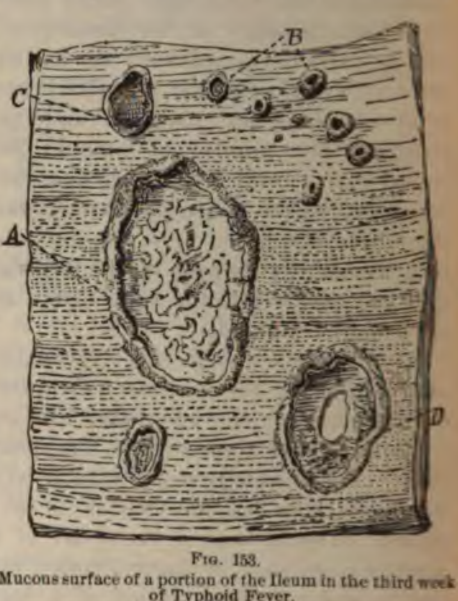


FIG. 153.
Mucous surface of a portion of the Ileum in the third week of Typhoid Fever.
A. Peyer's patch, ulcerated, showing the overhanging edges and the roughened base.
B. Solitary follicles ulcerated at their apices.
C. A small oval ulcer.
D. Perforation of the intestine.

Usually the sloughing and removal of the necrotic tissue does not take place until the *third week* of the disease. As the sloughs gradually loosen and fall off, there is a loss of substance which extends to the deeper layer of the mucous membrane, removing the entire gland and the mucous tissue surrounding it, laying bare the muscular coat of the intestine. The necrotic process may extend and involve the muscular tissue, and end in perforation of the peritoneal covering. These ulcers may be developed in the jejunum, the ileum, the stomach, and the large intestine. In the lower part of the ileum, at the ileo-cæcal valve, they are usually of large size—so large that only small portions of healthy mucous membrane are left between them; in the jejunum, stomach, and large intestine they are usually round and of small size. The form of the ulceration corresponds to that of the necrotic tissue; if an entire Peyerian patch is necrotic, an elliptical ulcer is formed, with its long axis corresponding to that of the intestine. In the jejunum and large intestine, the ulcers are usually small and round. The edges of the ulcer are sharp, tumid, and overhang the floor of the ulcer. Sometimes the ulcers are hemorrhagic.

In the *fourth week* the process of cicatrization is commenced. Gradually the swollen edges of the ulcers subside, granulation-tissue springs up from their base, connective-tissue membrane is formed, and the edges of the ulcer become united by a cicatrix which is covered with a layer of epithelium. The gland structure is never regenerated. The cicatrix which is formed by the healing of these ulcers is slightly depressed, and less vascular than the surrounding mucous membrane. During the healing process the cicatrix becomes more or less pigmented, and these pigmented scars may be recognized years after cicatrization has taken place. They seldom cause any puckering or diminution in the calibre of the intestine. In many cases the process does not pursue this regular course; while one portion of the ulcer is cicatrizing, ulceration in another part may be extending; such long-continued ulceration prolongs convalescence, and may even cause death from exhaustion.

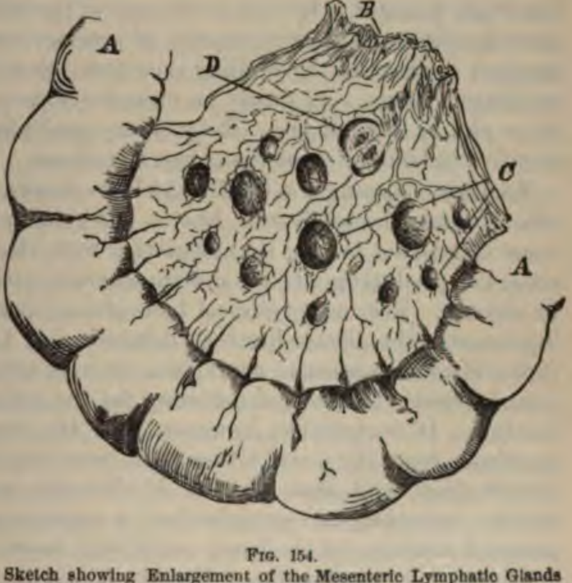


Fig. 154.
Sketch showing Enlargement of the Mesenteric Lymphatic Glands in Typhoid Fever.

A, A. Portion of Small Intestine.
B. Mesentery.
C. Glands enlarged. At D a gland is shown in section.

Mesenteric Glands.—Associated with these intestinal changes, analogous processes take place in the mesenteric glands. These mesenteric changes are also most marked in the glands situated nearest the ileo-cæcal valve; they are secondary to the changes in the intestinal glands, and usually in a degree corresponding to the extent of the intestinal lesions. The glands are first congested, then there is a production of lymphoid and large cells similar to those which are found in the enlarged intestinal follicles; the glands become enlarged, and are the seat of an acute cellular hyperplasia. When the enlargement has attained its full size, the hyperæmia diminishes, and the cellular elements begin to disintegrate and are absorbed. In about one-half the cases the enlargement reaches its maximum size by the middle of the second or at the commencement of the third week. The enlarged glands vary in size from that of a hazel-nut to a small hen's egg. In the stage of retrogression some of the glands simply shrink and return to their normal condition; in others, partial softening takes place and afterward absorption, leaving a fibrous cicatrix. If the glands reach a very large size, absorption is incomplete, and dry, yellow, cheesy masses are left, which after a time become calcareous and enclosed in a fibrous capsule. In rare instances the glands become fluid, their capsules are destroyed, and the softened masses escape into the peritoneal cavity and cause peritonitis. A calcareous condition of the mesenteric glands, like the pigmented cicatrices of the solitary and agminated glands, gives evidence of a previous severe attack of typhoid fever.

Another lesion of typhoid fever occurring during convalescence, is suppurative inflammation in the subcutaneous *cellular tissue.* The inflammation is not of an active type, but is accompanied by some redness and pain. Gradually a tumor is formed at the seat of the inflammation, usually where there has been the greatest amount of pressure, and after a time fluctuation becomes distinct as the swelling increases; sometimes two or more of these swellings coalesce, and finally an immense abscess may be formed containing a pint or more of pus. Retro-pharyngeal ulcers are the result of suppurative inflammation of the connective-tissue.

Etiology.—Among the bacilli which are known to be connected with specific diseases, the bacillus of Eberth is generally recognized as the specific cause of typhoid fever. By inoculation with this bacillus, Gaffky has succeeded in producing a fever, and the characteristic lesions of typhoid fever, in animals. Very many clinical facts also support the claim that the exciting cause of the disease has been determined. I have included this fever in the class of miasmatic contagious diseases because a period of extra-corporeal growth has seemed necessary for the complete development of the bacillus. It is possible, however, that the circumstances attending its expulsion from the body, rather than inactivity of the virus, determine its classification, and that while it is clinically miasmatic-contagious, it is strictly, according to our definition, a contagious disease. The generally accepted meaning of the word *contagious*, however, makes it desirable to continue this classification. Usually it has been regarded as an endemic form of disease. There seems to be no connection between its development and destitution. It may occur as an isolated case, or whole households and neighborhoods may be stricken down with the disease. It is possible for it to prevail as an epidemic, but it must first have been endemic.

Persons sick with this fever are now admitted into our general hospitals, and are placed by the side of patients with pneumonia or any form of chronic disease, without endangering the lives of such patients. This fact shows how generally the profession regard this disease as non-contagious. Typhoid-fever patients are no longer restricted by quarantine regulations. The history of epidemics of typhoid fever leads to the conclusion that the poison is contained in the *fæcal discharges* of the sick. When such excrement is in fresh condition the poison is not active; it must go through a stage of development outside of the body. This may take place in the excrement itself, but it goes on more rapidly and abundantly if the excrement is collected in privies, or in earth that is already saturated with organic matter. In this way we can readily explain how a typhoid-fever patient coming into a locality previously free from the disease can establish there a focus of infection, from which many persons may become diseased. It is supposed that this poison is not active in its fresh state, from the fact that the disease is not carried directly from one individual to another; attendants, nurses, and physicians are no more liable to the disease than those who are not thus directly exposed to the disease.

It is difficult to determine the period of *incubation*, or length of time the poison must remain in the body before symptoms of the disease are manifest. The histories of isolated cases would lead to the conclusion that the period varies from fourteen to twenty days. Undoubtedly there are two principal channels of infection, namely, the air we breathe and the water we drink. This poison may be contained in gases which emanate from privies, sewers, etc., which have been the receptacle of excrement from typhoid patients, and also in drinking-water from springs and wells which have become contaminated by matters from adjoining privies and cess-pools. It is an established fact that water remains contaminated, though far remote from the point where it came in contact with a defective sewer or water-closet. Soil pipes and sewerage may be defective for a long time and no case of typhoid fever occur, when suddenly an endemic of typhoid fever breaks out, and careful investigation shows that its development was preceded by the introduction of the excrement of a single individual sick with the disease. It is the belief of some that milk may convey the typhoid poison, and there is evidence in favor of this opinion; but there is stronger evidence that the water used to dilute the milk, and not the milk itself, is the medium through which the poison is transmitted.

This poison has great vitality. Typhoid fever frequently occurs in the same locality year after year, when the surrounding conditions are favorable to its development. Those conditions are more frequently present in the autumn than in any other season of the year, and for this reason it has been called *autumnal fever*. Usually it makes its appearance in a locality, each year, at about the same time; case after case is developed until entire households and neighborhoods become its victims. Individuals who come to care for the sick may contract the disease, and even persons who visit houses in which the disease is prevailing may afterward develop the fever, contracting it, not from the sick, but from the infected atmosphere of the locality.

Age must be regarded as a predisposing cause of typhoid fever. It is much more likely to occur in young than in old persons; it occurs most frequently between the ages of fifteen and twenty-five, and is rarely met with in persons over fifty. There are also individual idiosyncrasies which seem to predispose to this fever. Some contract it upon the slightest exposure to the influence of the poison, while others, frequently brought in contact with it for a long time, escape. Again, an individual may have repeated attacks of typhoid fever.

Symptoms.—I shall first consider the prominent symptoms of a typical case, and discuss in detail these symptoms, without special regard to the time of their occurrence.

This fever is usually insidious in its approach, and comes on with a certain degree of uneasiness throughout the system; the patient feels uncomfortable, has no pain, but feels that he is about to be sick. He complains of a grumbling headache, more or less aching of the limbs, "a tired feeling all over," chilly sensations, alternating with flashes of heat and loss of appetite;—not infrequently nausea and vomiting are present. The premonitory symptoms gradually increasing in severity, by the fifth or sixth day the patient is compelled to take to his bed. At this early period there may be a slight diarrhœa. In very mild cases the disease comes on so insidiously, and with symptoms so mild, that the patient is often able to pursue his ordinary avocations, complaining only of an undefined indisposition. In very many severe cases it is impossible for the patient to accurately fix upon the time when the fever commenced; and in no case will an early positive diagnosis be possible. Typhoid fever may be suspected, but that is as far as one can safely go.

In all cases variation in *temperature* is one of the most important early symptoms. Such variations in temperature in a typical case may be divided into four periods, of one week each, which correspond to the four weeks of the disease. In the first week there is a gradual and steady rise in temperature, with regular morning and evening variations, each evening temperature being about 2° F. higher than that of the morning, and 1° F. higher than the previous night, so that at the end of the first week it is at its maximum,—104° or 105° F. This is one of the most characteristic features of the disease. This gradual rise of, and these variations in, temperature are not present in every case, but when they are present they will greatly assist in making an early diagnosis. It has been said that typhoid fever is the only disease, except double quotidian intermittent fever, that gives two full thermometrical curves within twenty-four hours; that is, two remissions and two exacerbations. If this is true, it helps to explain certain high temperatures in the morning, and affords valuable assistance in making a diagnosis.

During the second week the variations in temperature are slight, retaining, however, the same maximum as was reached at the end of the first week. The variations during the third week are remittent in character. During the fourth week they become intermittent, and the range of temperature in the exacerbations is lower. The variations in pulse correspond to those in temperature. During the first week the pulse gradually becomes more and more frequent, and remains at the height reached at the end of the first week; throughout the second and third weeks there are distinct morning and evening remissions; during the fourth week it falls to its normal standard. Diarrhœa generally comes on during the first week or is continued from the prodromal stage. In some cases it may have ceased by the second week.

On the seventh day, or between it and the twelfth day, the characteristic eruption appears. About this time the headache abates and more or less somnolence and delirium come on. The delirium at first is slight, and is only observed during the night. Day by day the patient loses flesh and strength, and becomes more and more unconscious, and all the phenomena of the typhoid state are developed, viz.: a dry brown tongue, feeble pulse, low muttering delirium, stupor, tremors, subsultus, involuntary evacuations, and the other phenomena of great prostration. If the disease is to terminate favorably, the amendment is usually gradual. The first sign of improvement is a decided remission of the fever.

Such, in brief, are the phenomena which attend the ushering-in and development of an ordinary case of typhoid fever; they are, however, subject to numerous modifications. Some cases are mild throughout their entire course; some are severe at first and mild afterward; some are mild at first and severe afterward; while others are severe throughout their entire course.

The Physiognomy.—As a rule, the countenance has nothing peculiar in its appearance; but if the disease is of a severe type, by the second week the countenance assumes a characteristic appearance—there is a pale, olive, leaden look, the eye becomes dull and the conjunctiva congested, and usually there is a small, rose-colored spot in the centre of each cheek. The face does not assume the dark mahogany color seen in typhus, but in the advanced stage of the fever it has more of the hectic flush of phthisis.

Tongue.—From the very outset, the tongue is covered with a light, white coat, but there is nothing special in its appearance before the end of the first week; then it may become red upon its sides and tip, and show a slight disposition to dryness in its centre. As the disease passes into its second and third weeks, the tongue becomes more heavily coated, the coating becomes brown and dry, and sordes collect upon the teeth and sides of the mouth in sufficient quantities to form crusts. These crusts may become thicker and more abundant as the disease progresses. At any period in the course of the disease the tongue may suddenly clear off, and present a shiny red, "beef-colored" appearance. The tongue and lips may become dry, cracked and fissured. As the sordes are removed from the lips, they will often bleed; and in certain cases, more especially in the severer forms of the disease, the entire mouth and tongue may be covered with dark-colored incrustations. Such incrustations are seen early in connection with those cases where there are extensive blood changes; when present they are of grave significance. One of the first indications of convalescence is a moist condition of the tongue about its edges; gradually its entire surface becomes moist, and by the time convalescence is fully established it is restored to its natural condition.

Gastric symptoms are always more or less prominent; loss of appetite is one of the earliest symptoms, and nausea and vomiting are quite common during the first week of the fever. The vomited matters usually consist of a greenish fluid. When vomiting comes on late in the fever, it is either due to sub-acute gastric catarrh, or is symptomatic of local or general peritonitis. In a large proportion of cases thirst is excessive. The lips are parched, and in severe cases crack and bleed. In some cases hemorrhage from the gums occurs.

Diarrhœa.—Although not invariably present, diarrhœa is so frequent an attendant of this fever that it is considered one of its characteristic symptoms. It varies with the severity of the attack, the date of its commencement, and its duration. The characteristic typhoid discharges are of a yellowish-green color, described as "pea-soup discharges." Sometimes they are of a dark color, resembling coffee-grounds; their reaction is alkaline. In some cases diarrhœa is present at the very outset of the disease, and continues throughout the entire course. In others, it does not appear until the third week. The second week is the ordinary time for its appearance. When the diarrhœa appears late in the course of the disease, the discharges are more copious than when it appears early. A mild diarrhœa throughout the entire course of the fever is a favorable rather than an unfavorable symptom. In mild cases diarrhœa is sometimes absent.

Intestinal Hemorrhage.—This occurs in about one in twenty cases, and varies in quantity from a mere trace of blood in the stools to a profuse discharge of from sixteen to eighteen ounces. The slight hemorrhages which occur early in the disease simply indicate a hemorrhagic tendency, the same as the epistaxis, which is very frequently among the early symptoms. In both instances the bleeding comes from the capillaries of the mucous membrane. The more profuse hemorrhages are due to the opening of an artery in some intestinal ulcer. Hemorrhages due to this cause may be sudden and profuse, and may destroy the life of the patient. The usual time for the occurrence of these profuse intestinal hemorrhages is the latter part of the second and during the third week. These hemorrhages are usually accompanied by a sudden fall in temperature, perhaps two or three degrees; if then in a patient severely ill of typhoid fever a sudden fall in temperature occurs during the second or third week, accompanied by extreme prostration, it is very conclusive evidence that intestinal hemorrhage has occurred, although externally the hemorrhage may not have made its appearance. The blood is usually fluid, rarely clotted; generally it is of a bright red color, owing to the alkaline condition of the intestinal contents. Copious intestinal hemorrhages are more frequent in severe cases that have been attended by profuse diarrhœa. Patients may die of intestinal hemorrhage before any blood has been voided externally. If the patient survive a profuse intestinal hemorrhage, there is great danger of his dying from peritonitis. He may die unexpectedly by syncope a number of hours after a profuse intestinal hemorrhage.

Abdominal pain and *tenderness* are not usually present at the very outset of typhoid fever, but generally, and almost without exception in the severer cases, by the sixth day of the disease some pain and tenderness will be present in the right iliac fossa. The pain and tenderness usually increase as the disease progresses, and in the advanced stages it is sometimes so marked that slight pressure over this region is unbearable. While examining this region in order to determine the presence or absence of pain and tenderness, the pressure should be made with the palm of the hand; the expression of the countenance will indicate the presence of tenderness, long before an audible complaint is made by the patient. It is important to bear in mind the possible occurrence of a more severe abdominal pain arising from intestinal perforation. The following are the characteristic symptoms of this lesion: if in the course of a slight or severe form of this fever, or even when the disease has been latent and the diagnosis of typhoid fever has not been clear, the patient should be suddenly seized with diarrhœa, pain in the abdomen, aggravated by pressure, perhaps at first localized in the right iliac fossa, but soon extending over the entire abdominal cavity, attended by rapid tympanitic distention of the abdomen, and symptoms of great prostration, a rapid, feeble pulse, a sunken, anxious expression of countenance, nausea and vomiting, quickly followed by coldness and blueness of the extremities, and the other signs of sudden collapse, it is almost certain that perforation of the intestine has occurred. I have known this accident to occur when convalescence was apparently progressing satisfactorily.

Tympanitis is another very common symptom of typhoid fever. Usually it is not present during the first week, but by the end of the first or the commencement of the second week a fulness of the abdomen will be noticed. As the fever advances, the distention often becomes extreme; this is due to a collection of gas in the large intestine, developed through some change in the mucous membrane, the exact nature of which we do not fully understand. When once it is developed it remains until convalescence is fully established, and is always an important diagnostic sign of this fever.

In connection with the development of the tympanitis, when firm pressure is made over the right iliac fossa, a gurgling sound is produced; but *gurgling* in the right iliac fossa cannot by any means be regarded as a positive symptom of typhoid fever, as it may occur in any disease where there is distention of the abdomen due to accumulation of gas in the intestines. In typhoid fever, so long as the abdomen remains tympanitic, no matter what the temperature and pulse of the patient may be, he is in more or less danger, for it shows that there are intestinal changes still in progress, and the reparative processes are not complete; this is more especially the case when the tympanitis has continued from the active period of the disease into the period of convalescence.

Urine.—Extended and very careful analyses of the urine of typhoid fever patients have been frequently made, without giving any very practical results. Usually during the first two weeks of the fever the urine is diminished in quantity, and its color is dark and its specific gravity high; after the second week it is increased, and when convalescence is established, it becomes pale, and its specific gravity is lowered. The amount of urine excreted daily throughout the active period of the fever is increased. The increase is in proportion to the intensity of the fever, subject in some degree to the quantity and quality of the food taken. It will be greater when large quantities of strong beef-tea are taken than when the diet consists of milk. So long as the kidneys are able to eliminate the excess of urea, no harm results; but if the quantity exceeds their power of elimination, or if their function is interfered with, uræmic symptoms will be developed, such as delirium, stupor and coma. *Albumen* in the urine is only of occasional occurrence in the course of typhoid fever. When present the quantity is usually small, and is only temporary. It rarely appears before the third week. Its appearance is often marked by the occurrence of cerebral symptoms. Renal epithelium and casts may or may not be present with the albumen.

Nervous Phenomena.—The symptoms referable to the nervous system are not so prominent in typhoid as in typhus fever; yet there are many cases in which these symptoms form an important part of the history. One of the most constant is headache. In the majority of cases it is one of the ushering-in symptoms of the disease. It is present in mild as well as in severe cases; sometimes it is confined to the forehead and temples; more often it extends over the whole head; it is not violent, but a dull, heavy pain. It usually increases in severity until the middle period of the disease, certainly until the close of the first week; and generally associated with it there is intolerance of light and conjunctival injection, pain in the back and limbs, and a general aching of the whole body.

Somnolence is present to a greater or less degree in all cases. In mild cases it does not appear until late, and usually is not long continued. In the severer cases it appears early and continues until convalescence begins; in fatal cases it passes into a state of coma. It is often interrupted by delirium. In children this symptom is especially prominent, and is very valuable as a means of diagnosis.

Delirium is more frequently present than absent in typhoid fever. The character of the delirium varies; the usual form is known as the "low-muttering" delirium, and is rather characteristic of this type of fever, although in very many cases the delirium is violent in character, and may become maniacal to such an extent as to require physical restraint. Not infrequently typhoid fever patients attempt to jump out of a window, or injure themselves or their attendants in their endeavors to escape from fancied dangers. It is very common for the minds of this class of patients to be occupied with those things which engaged their attention just prior to their illness. The delirium rarely comes on until the second week of the fever, and it commences and is most active at night. After it has once appeared, it usually continues until convalescence is established, and generally disappears during a sound sleep which attends the early stage of convalescence. The maniacal form of delirium in typhoid fever is usually most marked at night. During the low-muttering delirium, if the patient is asked questions he will generally answer correctly.

Muscular Prostration and Paralysis.—In all severe cases of typhoid fever, muscular prostration is noticeable in the early stages, and increases with the progress of the fever. It is generally most marked during the third week. Where there is marked muscular paralysis, the urine and fæces are passed involuntarily, there is inability to protrude the tongue, and more or less difficulty in deglutition, or inability to articulate distinctly. Retention of the urine may also occur from vesical paralysis.

Muscular Tremors.—Tremors of the hands, tongue, or lips are most often met with in young subjects and those who are addicted to the use of spirits. Severe tremors unaccompanied by much mental disturbance often attend extensive intestinal changes. Spasmodic movements, such as subsultus, hiccough, etc., and rigid contraction of the muscles of the neck or extremities are sometimes present in severe cases. General convulsions are of rare occurrence, except in very young children, and when they occur have no special significance.

Special Senses.—The symptoms referable to the special senses require little more than enumeration. As regards the *sense* of *sight*, there is nothing worthy of note, except that the eye assumes a dull expression and that the pupil is dilated ; some patients complain of haziness of vision, which is increased when they assume a sitting posture. The *sense* of *hearing* is always more or less impaired. This is most marked about the middle period of the fever ; then it is impossible for the patient to hear ordinary conversation. Ringing and buzzing sounds in the ears are often complained of in the early stage of the fever. When the loss of hearing is confined to one ear, it is generally caused by ulceration of the mucous lining of the Eustachian tube, or by suppuration of the middle ear. The *sense* of *taste* usually is altered or perverted ; articles of food are tasteless, or have an unnatural flavor. When the tongue and mouth are covered with a heavy coating of sordes, the patient is unable to distinguish between bitter and sweet, and swallows the most disgusting doses without complaint.

Hyperæsthesia is another disturbance of a special sense. The surface of the body of a typhoid fever patient may become so sensitive that he will cry out with pain from the slightest touch. This hyperæsthesia may be present during the first week, or may not be present until convalescence is established. It is most marked over the abdomen and lower extremities, and usually occurs in females of an hysterical tendency. It is of importance to discriminate between cutaneous tenderness in the abdominal region, and the tenderness of peritoneal inflammation.

Epistaxis.—When this occurs during the first week, in most cases it is of little importance except as a diagnostic sign of this type of fever ; when it occurs during the third week it becomes important as an element of prognosis, as it may be sufficiently profuse to destroy the life of the patient.

Occurring late in the disease, unless it can be promptly arrested, it always jeopardizes the life of the patient.

Emaciation is perhaps more marked and rapid in this than in any other form of fever. It commences early and is progressive. By the time a patient has reached the fourth week of a typhoid fever of even moderate severity he is usually in a condition of extreme emaciation. In this particular he markedly differs from a patient ill with typhus fever, for in the latter case emaciation to any great extent does not occur.

Temperature.—In making thermometrical observations in this as well as in all other forms of fever, the thermometer may be placed in the axilla, the mouth, or the rectum. I shall refer to axillary temperature wherever I speak of temperature without qualification. Usually in a typical case the temperature begins to rise about noon on the first day of the development of the fever, and continues so to do until between six and eight o'clock in the evening, when it reaches its maximum for that day; then there is no change until midnight, when it begins to decline, and by six or eight o'clock in the morning it has reached its minimum, which is a degree higher than on the morning of the preceding day. After six or eight o'clock in the morning the temperature does not vary much until noon; then it again begins to rise, and by six o'clock in the evening it has reached its maximum for that day, which is one degree higher than on the evening of the preceding day. Again, at midnight it begins to fall, and by morning it has fallen a degree, which leaves the minimum, and the average for the day a degree higher than on the preceding day. Thus it rises a degree each day, with regular morning and evening variations, until the eighth day of the fever, when, in most cases, it has reached its maximum for the whole course of the disease.

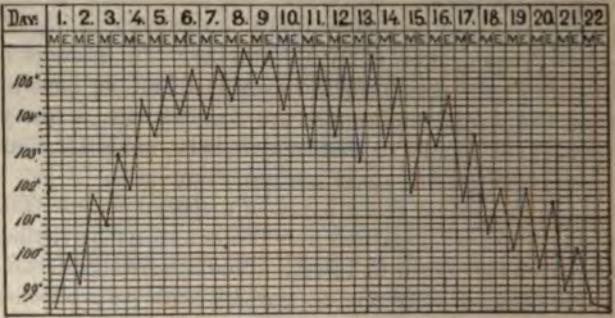


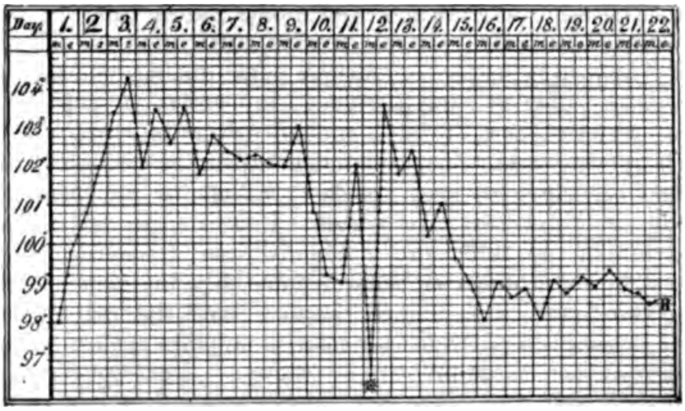
Fig. 155.
Temperature Record in a Typical Case of Mild Typhoid Fever. Recovery.

During the *second week* the temperature remains at about the same maximum degree which it has reached at the end of the first week. There are morning and evening variations of a degree or more, but the maximum of the evening exacerbation remains the same.

During the *third week* the remission becomes more and more marked, while during the exacerbation the temperature retains nearly the same standard as during the second week. By the end of the third week the morning temperature during the remission will be two or three degrees below the maximum of the second week.

By the time the *fourth week* is reached, or at least by the middle of that week, the temperature becomes intermittent, and with each exacerbation it falls lower and lower, until by the end of the week the normal standard of temperature has been reached, or it may fall a little below the normal standard. These are the typical thermometrical variations of typhoid fever, yet they are not always present, and there are many things which will materially modify them. The fever does not always follow this typical course. Marked deviations from the record may be produced by complications which would never have been discovered but for the irregular thermometrical variations. By treatment, the temperature can, for a time, be very much lowered ; but if the treatment be discontinued, it will rise again. In some cases it is not possible to detect the cause of the irregularity.

Pulse.—The pulse is also subject to variations, which correspond very nearly with the variations in temperature, and occur not only on different days, but at different hours on the same day. During the first week the pulse becomes more and more frequent ; in the second and third weeks it remains at its height, and during the fourth week sinks to its normal average. Throughout the whole course of the disease it is less frequent in the morning than in the evening. If, at the commencement of the fever, the pulse is 98, by the end of the first week it will have reached 100, or 110 per minute, and there it remains during the second week ; after that time it may become as frequent as 120, or 140. During the first and second weeks the rate of the pulse and the temperature range correspond, but after this time the parallelism ceases as the failure of heart-power begins



● *An intestinal hemorrhage occurred here.*

FIG. 156.

Temperature Record in a Non-typical Case of Typhoid Fever.

to manifest itself. This failure of heart-power is indicated by an increase in the frequency and feebleness of the pulse, which at this time may reach 140 per minute and yet the temperature show no alarming variation. Under these circumstances, the pulse may become irregular and intermitting. Should these irregularities and intermissions occur during the third week, in most cases they are followed by death. With an irregular and intermitting pulse, the first sound of the heart will usually be inaudible over the precordial space, and this indicates that prompt and judicious means must be employed to restore the heart's normal action and to avert a fatal issue.

The severity of the disease during the first and second weeks of its development is, to a great extent, indicated by the frequency of the pulse and the height of the temperature. Although delirium and extensive tympanitis are important symptoms they do not determine the result; but if a patient during the first, or at the commencement of the second week of the disease, has a pulse of 120 per minute, and a temperature of one hundred and six, it is very doubtful whether convalescence can ever be established. The pulse may increase in frequency from feeble heart-power alone while the temperature is steadily falling. On the other hand, the pulse sometimes falls almost to a normal standard, while the temperature remains high.

Eruption.—Some have claimed that this should be considered as a lesion of the disease, but I prefer to class it among the symptoms. It makes its appearance between the sixth and twelfth days, dating from the commencement of the fever, and it is not attended by any unusual sensation. It remains visible from eight to fourteen days, leaving no stain or mark on the surface after its disappearance. It consists of isolated, lenticular spots scattered more or less abundantly over the surface of any part of the body, but usually most abundant upon the chest and abdomen. There may be only a few spots visible at a time, or they may be so profuse as to cover the body like a rash. Two or three well-defined spots of the eruption are sufficient to establish the existence of the fever. Each spot varies in diameter from a point to a line and a half, rarely reaching two lines. It is slightly elevated above the surface of the surrounding cuticle, is of a bright *rose* color, disappears upon slight pressure, and returns as soon as the pressure is removed. Each spot remains visible for three days, and then disappears. Sometimes, as one crop of the eruption disappears another is developed, and this may go on for eight, twelve or fourteen days. There are many cases in which only one crop appears. As soon as one spot makes its appearance, it is well to mark it with tincture of iodine or nitrate of silver, so that observations will be always made upon the one point. If it is a spot of typhoid eruption, and one crop of eruption is to follow another, it will disappear within three days from the time at which it was first seen, and other spots will take its place. It is this feature which distinguishes the typhoid eruption from that of all other fevers.

The question may be asked:—is this eruption essential to the diagnosis of typhoid fever? Many observers mention that the eruption is not constant, and consequently not necessary for its diagnosis; while others, equally competent, maintain that, unless the eruption be present at some period during the progress of the disease, the diagnosis of typhoid fever cannot be made with certainty. Jenner states that he found the eruption present in one hundred and forty-eight out of one hundred and fifty-two cases. I would not say that it is possible for typhoid fever to occur without the eruption; neither would I affirm that scarlet fever ever exists without the characteristic rash of the disease; but as regards these respective fevers, if no eruption was present, I would make the diagnosis with equal hesitancy in the one case as in the other. The eruption is usually most marked in cases of typhoid fever which occur between the ages of ten and thirty. Before ten and after thirty years it is usually not as well marked, and may be readily overlooked unless careful search is made.

The typhoid poison, in its operation on the human body, does not always effect the series of changes and symptoms just described. On the contrary, there are cases which run so mild a course that they can scarcely be dignified by the name of fever, and, besides, there are imperfectly developed cases which show a great diversity in their course, but they all can be included under two heads.

First.—Mild typhoid fever, in which the symptoms are all mild.

Second.—Abortive typhoid fever, in which the duration of the disease is markedly shortened.

In the *mild type*, the fever runs its regular course, but is of low grade. The temperature rises regularly until its maximum is reached, which rarely exceeds 103° F., then it remains stationary for a time, generally about a week; the decline follows in the same manner as in a typical case. This is the regular course of these cases if left to themselves, and, as a rule, they should be left to themselves. Some of these cases are so mild that the patients are not confined to the bed, or even to their rooms, and perhaps throughout the entire course of the disease are able to transact a certain amount of business. Such cases have been called "walking cases" of typhoid fever. The eruption appears in these cases early, is of short duration and only a few spots appear; usually there is only one crop. Diarrhœa is also present in most cases of this class, but it is of a mild type and the discharges from the bowels apparently give relief to the patient. In some cases the diarrhœa alternates with constipation, or constipation may be present throughout the entire course of the disease, and the cases go on exhibiting a varying amount of fever for from twenty to thirty days, until gradual convalescence is established. This class of cases, if properly managed, rarely prove fatal; but if improperly managed, there is great danger. If a patient walks about while he is suffering from one of these so-called mild attacks of typhoid fever, he does it at great risk to life; there should be no "walking cases" of typhoid fever. A patient sick with typhoid fever, however mild the type, should take to his bed and remain there until convalescence is fully established, as it is impossible to say just how extensive the changes may be that have occurred in the intestinal tract, and in the mildest type of the disease they may be of such a nature that

Pneumonia.—Pneumonia, with typhoid symptoms, is sometimes mistaken for typhoid fever. The pneumonia which complicates typhoid fever does not come on until late in the fever, and is preceded by the regular history of typhoid fever. On the other hand, when the typhoid symptoms are present from the beginning, or come on at the end of the second stage of the pneumonia, the physical signs of the pneumonia will attend or precede the typhoid symptoms. There will be cough and the characteristic pneumonic expectoration; there will be no eruption, and no typical variation in temperature. If a patient who is over sixty years of age with this type of pneumonia is not seen until the second or third week of its progress, although evidences of lung consolidation may be present, it will frequently be very difficult to decide whether the pneumonia is or is not complicating a typhoid fever. The diagnosis must be based upon the history of the case.

Gastro-Enteritis.—In the adult this disease is quite readily distinguished from typhoid fever, as the diarrhœa and vomiting precede the febrile movement; the fever is irregular in its development and progress, and the temperature rarely rises higher than 103° F. In a child between two and six years of age it is very difficult to distinguish gastro-enteritis from typhoid fever. The typhoid eruption is not so prominent or constant a symptom in the child as in the adult, and with both diseases we have diarrhœa, tympanitis, and typhoid symptoms. When all the symptoms precede the fever, and there is a history of the case, and a thermometrical record from the beginning of the fever, in most cases the diagnosis can readily be made; but if the case is not seen until it has reached the second week of its progress, and there is no accurate or reliable history of its development, a positive diagnosis is impossible.

Trichinous Disease.—This condition is not infrequently attended by diarrhœa, vomiting, and the development of other typhoid symptoms; but with poisoning by trichinæ there are almost constantly present intense muscular pains and œdema of the eyelids, which will be sufficient to arrest attention. There will be wanting the typical temperature trace and the rose rash, and a microscopic examination of small portions of the muscular tissue will afford a positive diagnosis.

Prognosis.—Death may occur at any stage of this fever. A typhoid patient is not out of danger until all tympanitis, diarrhœa, and other abdominal symptoms which indicate that intestinal changes are still progressing, have disappeared. Independent of complications, the duration, type, and intensity of the febrile excitement have more to do than all the other elements in determining the prognosis in any case of typhoid fever. The height of the temperature on the eighth day determines the range of temperature that may be expected on each succeeding day. If upon that day it is not higher than 104° or 105° F., and has been regular in its development (independent of complications), the prognosis is good; in uncomplicated cases it very rarely rises higher than the degree it has reached at that time. A prolonged high temperature (above 105° F.) after the first week renders the prognosis unfavorable. In mild cases, during the second week, a marked morning remission occurs, which begins early and continues until midday; the evening exacerbation is late, and by the end of the second week there is a marked and permanent fall in the temperature. In severe cases, the opposite conditions are observed. A sudden rise in temperature, or a rapid and extreme fall at any period of the fever is a very bad omen; the latter often precedes the occurrence of a severe intestinal hemorrhage. Marked variation from the typical temperature of the disease indicates the existence of complications. Slight decline accompanied by great fluctuation of temperature, during the third week, is an unfavorable symptom. The natural power of an individual to resist disease, especially the effects of prolonged high temperature, is a very important element in prognosis. The organ which is the surest indicator of such power (especially in typhoid fever) is the heart.

If the pulse is full and regular, perhaps beating at the rate of 110 or 115 per minute, if the cardiac impulse is good, and a distinct first sound can be heard, even though at the end of the second week the temperature stands as high as 106° F., the prognosis is favorable. If, however, the pulse has risen to 120 or 130 per minute, if the apex-beat is feeble or imperceptible, and the first sound of the heart is indistinct or altogether obscured, with a tendency to cyanosis and pulmonary œdema, the indications are that the patient's powers of resistance are failing, and under such circumstances the prognosis must be unfavorable. It is not so much rapidity as irregularity, *a sudden falling and a sudden rising of the pulse*, that indicates impending danger. The rapid rising of the pulse upon the slightest excitement is the most unfavorable indication, as it shows extensive heart-failure and a rapid giving way of vital power. A sudden fall of the pulse from any cause must always be regarded as an unfavorable indication. The abundance or color of the eruption does not influence the prognosis. Excessive tympanitis and severe abdominal pains are unfavorable symptoms. Severe and protracted muscular tremors, with subsultus, indicate danger. Sudden collapse during the second and third weeks of the fever is always attended with danger, as it is very likely to be due to copious intestinal hemorrhages or intestinal perforation. It sometimes occurs independently of either of these causes, but nevertheless is very apt to be soon followed by a fatal result.

The prognosis is always bad in those who are very fat, and in those who are the subjects of gout, disease of the kidney, or any other severe form of chronic disease. In all such persons, during the second and third weeks of the disease, it is necessary to be constantly on the watch for the occurrence of sudden collapse. My own experience leads me to the belief that when intestinal hemorrhage is scanty it has little influence on the final result. When it occurs before the twelfth day of the fever, it is often of advantage by relieving the intestinal congestion. But when profuse, or even slight, after the twelfth day, it is an unfavorable symptom and renders the prognosis unfavorable. The occurrence of the hemorrhage renders it probable that ulceration has extended to the vessels beneath the transverse muscular fibres of the intestine, and such ulceration is very apt to go on to perforation and a fatal peritonitis.

The influence of age is very great in determining the prognosis in any case of typhoid fever. It is much better in children than in adults; and in persons over forty years of age the prognosis is decidedly unfavorable, even though the symptoms may not indicate a severe type of the disease. In the case of those individuals who habitually use alcoholic stimulants, whose power of resistance to high temperature is diminished, the rate of mortality is very great. The puerperal state renders the prognosis especially unfavorable. The danger to the patient is equally great, whether the fever comes on prior to delivery or during puerperal convalescence. The parenchymatous changes which take place in the different organs of the body during the progress of this fever, necessarily influence the prognosis. The muscular degenerations of the cardiac walls, and consequent loss of heart-power, which favor pulmonary and other hypostatic congestions, and the diminished quantity of blood sent to the various tissues of the body, interfere more or less with their nutrition. Necrotic and gangrenous processes, sometimes met with in the cellular tissue of the surface and along the line of the intestines, as also the venous thrombi which so frequently develop in a protracted case of this fever, are, to a certain extent, the result of this cardiac weakness, and render the prognosis unfavorable. Excessive cardiac weakness also favors the development of blood-clots in the heart-cavities; these may break up and cause embolism somewhere in the course of the general circulation, and thus lead to changes which may destroy life. *Intestinal perforations*, one of the results of the intestinal changes incident to the fever, render the prognosis most unfavorable.

Complications.—Slight bronchial catarrh can hardly be regarded as a complication, it is so much a part of the clinical history of the disease, but another much more serious bronchial complication is capillary bronchitis. This usually comes on during the second or third week of the disease, and, if extensive, greatly endangers the life of the patient. It is indicated by subcrepitant râles suddenly developed over the whole of both lungs, accompanied by great dyspnœa and an abundant expectoration of stringy mucus. Its advent renders the prognosis most unfavorable. Extensive œdema of the lungs, occurring with, or independent of, capillary bronchitis and pulmonary congestion, sometimes comes on suddenly during the third week of typhoid fever, and indicates great failing in heart-power. The slightest indication of its occurrence would always be regarded with suspicion. It is not infrequently accompanied by more or less extensive hemorrhagic infarctions of the lungs. These depend on embolism of some of the branches of the pulmonary artery due to fragments of clots which have formed in the right side of the heart, the result of the cardiac weakness, and often lead to gangrene of the lung. It is sometimes impossible to diagnosticate their existence during life.

Pneumonia, when it complicates typhoid fever, is generally latent. It comes on very insidiously, and will be recognized only by the most careful physical examination. It is more frequently developed during the third and fourth weeks of the fever, and usually is lobular rather than lobar in character. At first only single lobules are involved, but after a time an entire lobe becomes consolidated. When irregular variations in temperature occur during convalescence, or during the third or fourth week of the fever, there is reason to suspect the development of pneumonia. In the majority of cases the characteristic pneumonic cough and expectoration are absent. Whenever an extensive pneumonia complicates typhoid fever, the prognosis is especially unfavorable.

Pleurisy is not so frequently a complication of typhoid fever as is pneumonia or bronchitis. When it does occur, the almost invariable product of the inflammatory process is pus. Usually it comes on insidiously, late in the disease, and is quite likely to pass unrecognized unless frequent physical examinations are made. In many instances it is really a sequela of the fever, not developing until three or four weeks after the fever has run its course. Its occurrence must always be regarded as unfavorable, for a year, or even longer time, must elapse before recovery can take place, and even then complete recovery is doubtful.

Occasionally *laryngitis* is a serious complication of this fever. It generally occurs in those cases where the fever has been very protracted, and there is great prostration. Its presence is marked by sudden and very intense inflammation of the mucous membrane of the glottis, which is liable to become œdematous, when death may suddenly occur. It may lead to ulceration of the mucous membrane. *Pyæmia* may be met with as a complication during convalescence from typhoid fever, but it is not of as frequent occurrence as septicæmia. Whenever septic poisoning is developed, with extensive sloughs in the intestines, the prognosis is exceedingly unfavorable. *Acute gastric catarrh* is another complication of this fever. Disturbances of nerve-function have been considered under the head of symptoms, but not infrequently certain brain and nerve lesions are developed which cannot be classed under that head. Cerebral œdema may complicate a typhoid fever during its third week, and give rise to symptoms of a grave character. A decided enfeebling of the mental powers and a tendency to stupor announce its occurrence. Hemorrhagic extravasations on the surface and into the substance of the brain, the result of degeneration of the walls of the cerebral vessels, occasionally occur during the height of the fever. If the effusion is moderate, no marked symptoms are developed; but if a considerable extravasation takes place, it gives rise to symptoms of cerebral compression. Meningeal inflammation is a rare complication. The occurrence of any of these complications in any case renders the prognosis unfavorable.

During the second and third weeks of the fever certain cerebral disturbances may occur which seem to indicate the existence of some one of these complications, when no cerebral lesion exists. Usually they are present in patients who have had a continuously high temperature, and in favorable cases they disappear after a few days. Various other disturbances of the nervous system, such as hemiplegia, paraplegia, etc., which may simulate those due to lesions of the nerve-centres, or forms of local paralysis and anæsthesia which seem to be confined to individual nerves are met with, but as these functional disturbances do not depend upon any anatomical changes, the prognosis in such cases is good. Those changes in the kidney, due to parenchymatous degeneration, which usually attend this fever have already been noticed; but occasionally nephritis is developed as a sequela. The urine becomes scanty, is loaded with albumen, and contains blood and casts; the face and extremities become œdematous, and death may occur from uræmia. The occurrence of this complication necessarily renders the prognosis bad. In a few instances under my observation, severe catarrh of the bladder has developed during convalescence, greatly complicating the case; in one instance the cystitis was accompanied by pyelitis. Cellulitis, especially of the surface, often complicates convalescence, and in some cases causes death. Occasionally it is met with in the pharynx and along the line of the lymphatics. Accompanying this cellular inflammation, or independently of it, gangrenous inflammations of the integument not infrequently occur, giving rise to *bed-sores.* These gangrenous processes are most frequently developed at those points which have been subjected to the greatest pressure, on account of the position of the patient in bed, such as the sacrum, nates, heels, and shoulder-blades. In the simplest form of *bed-sores* there is only a superficial loss of substance; in more severe cases the subcutaneous cellular tissue is involved; and in the worst cases the muscles and fibrous tissues. I have met with cases where the slough had involved the connective-tissue and muscles, and laid bare the bone. A considerable number of typhoid patients who have lived through the fever die either from the exhausting effects of these bed-sores or from the resulting septic poisoning. The possible occurrence of these complications must enter into the prognosis in every severe case, and the earlier they make their appearance the greater the danger.

The average *duration* of typhoid fever is from three to four weeks. It may terminate in death or recovery at an earlier date. A typical case extends over a period of four weeks. The period of invasion lasts from one to five days. The period of glandular enlargement continues until about the fourteenth day. The period of ulceration extends from the twelfth or fourteenth day to between the twenty-first and twenty-eighth. When the fever is protracted beyond the middle of the fourth week, in most instances this is due to some complication, or to an extension of the intestinal ulceration. The period of greatest danger is at the close of the third week.

Death rarely occurs before the fourteenth day. The prominent direct causes of death are: *toxæmia; asthenia; suppression of the excretory function of the kidneys; hyperæmia and œdema of the lungs; intestinal hemorrhage; exhaustive diarrhœa; intestinal perforation; and peritonitis with or without intestinal perforation.* In nearly all cases the failure of heart-power is directly or indirectly the cause of death. In no case can convalescence be said to be fairly established until the temperature remains normal for two successive evenings. The termination, like the commencement, is gradual, and is not marked by any critical evacuation or day of crisis.

Relapses.—After typhoid fever has run its course, and the patient is entirely free from fever, quite frequently there is a new development of the fever; these new developments are called *relapses.* Their course corresponds with that of the primary attack, only they are of shorter duration. The temperature rises more rapidly, the eruption reappears, the spleen enlarges, the intestinal and abdominal symptoms return, and all the prominent symptoms of the primary fever are rapidly developed. As a rule, the relapse is milder than the primary attack. If it terminates fatally, the post-mortem examination shows, in addition to the cicatrizing intestinal ulcers of the primary attack, the recent intestinal changes of the relapse. The lesions of the relapse, although of the same character as those of the primary attack, are less extensive.

It is very difficult to give a satisfactory explanation of these relapses. Some claim that they are the result of certain plans of treatment, especially the cold-water plan. This assertion lacks proof. Others hold that all relapses depend upon a new infection. Perhaps this is possible if the patient remains in the same locality and has the same surroundings as when he had the primary attack; but it does not explain relapses in those who are removed from all the sources of the primary infection. Another explanation offered is that a part of the typhoid poison has remained in the system, undeveloped during the primary attack, and that some time after this has passed the poison reproduces itself and sets up a second fever. A more recent theory is that the typhoid poison thrown off in the fæces of the patient is reabsorbed and causes the relapse. Unquestionably, it is possible for healthy glands to become inoculated by sloughs thrown off from those first affected. In many cases it is impossible to account for the occurrence of the relapse, and all of these explanations as to the cause in any case are more or less unsatisfactory. In those cases which have come under my own observation, I have noticed that the splenic enlargement which has existed during the course of the fever does not subside with its decline; and that the tenderness along the line of the intestines, especially in the right iliac region, continues during the period between the original attack and the relapse. In some instances, apparently, the relapse has been brought on by indiscretion in diet, or by injudicious exercise on the part of the convalescent patient. Occasionally relapses have occurred when great care had been taken against any indiscretion or over-exertion. There is little doubt but that relapses are of much more frequent occurrence in those cases that are treated with cathartics during the first week of the fever, than in those where cathartics are not employed.

Treatment.—Since the specific poison of typhoid fever is contained in the excrements of typhoid patients, the *first* indication in *prophylaxis* is to destroy this poison as soon as it is discharged from the body. For this purpose the intestinal discharges should be received into a porcelain bed-pan, the bottom of which is covered with a thin layer of powdered sulphate of iron; immediately after the discharge, crude muriatic acid, equal in quantity to one-third of the fæcal mass, should be poured over it. The discharges of a typhoid patient (no matter how thoroughly they may have been disinfected) should never be emptied into a privy or water-closet. Trenches should be dug for their reception, and new trenches should be opened every few days; the greatest care must be taken that these trenches are not so situated that the drainage from them can contaminate wells or springs which furnish drinking-water.

All underclothing or bed-clothing that may have become soiled by the discharges from the bowels should be immediately immersed in chlorine water or other disinfectant, and thoroughly boiled within twenty-four hours. These procedures will certainly destroy the infective power of the typhoid poison contained in the intestinal discharges, and in the majority of instances will prevent the spread of the fever. Repeated observations show that when one member of a family has typhoid fever, not infrequently it is developed in every other member. This spread of the disease can be prevented, unless there is some local cause for its development which cannot be reached. When its origin is not apparent, the wells, springs, and all the sources from whence water is derived for drinking and cooking purposes should be carefully and thoroughly inspected. Care must be taken that the waste-pipes from wells and springs do not pass directly into cess-pools or sewers, and thus become a means for the conveyance of impure gases into the springs and wells. The greatest care must be exercised in regard to house drains and sewer pipes, that they shall be free from leakage and obstruction, and that all water-closets, sinks, and other openings into them be provided with suitable traps. When unpleasant odors are constantly present in dwellings, especially in sleeping apartments, disinfectants should be employed, and the house be thoroughly ventilated. When it is necessary to open drains and cess-pools in a dwelling for purposes of repairing or cleansing, the same precautions should be exercised; they are especially of importance during the summer and autumn.

The question naturally arises:—is it not possible to counteract or neutralize the effects of the fever-poison after it has gained admission into the system, and thus prevent the development of typhoid fever? To accomplish this, blood-letting, emetics and diaphoretics have all been employed; but there is not the slightest proof that typhoid or any fever-poison was ever removed from the system by these or any other agents. A patient with some of the premonitory symptoms of fever may perspire, be relieved, and at once recover, but such a patient had not received the typhoid poison into the system, and was not, as is sometimes said, "threatened with typhoid fever." Notwithstanding the bold affirmation of the author of the cold affusion plan of treatment, that if it were resorted to before the third day of the disease, it would invariably arrest its development, it has failed to stand the test of practical experience. More recently, sulphate of quinine, administered in large doses, has been thought to have the power of arresting the development of typhoid fever in the same way that it arrests malarial fever, by its anti-periodic power; but there is no evidence that it has any such power, and as a prophylactic remedy it has been abandoned.

After the poison has once gained entrance into the system, no means has as yet been discovered by which it can be counteracted or neutralized so as to prevent the development of the disease. The duty of the physician is to guide the disease, so far as he may be able, to a favorable issue, and prevent injury to organs essential to life, keeping in mind that a certain definite period must elapse before this result can be accomplished.

The arrangement of *the sick-room* of fever patients, though often overlooked, is a matter of no inconsiderable importance, not only as regards the comfort of the patient, but also the successful issue of the case. It is of the greatest importance that a properly qualified nurse be selected; one who has had experience in the care of fever-patients is to be preferred. The patient should be placed in a large and well-ventilated apartment. All furniture should be removed from the sick-room except those articles which are necessary for the comfort of the patient and the convenience of the attendants. The carpets should be removed from the floor and the patient placed in a bed of moderate size in the centre of the room. Free ventilation during both day and night, is of the utmost importance. The temperature of the apartment should be kept below 60° F. The bed and body linen of the patient should be changed daily, and at once removed from the sick-room and placed in a weak solution of chlorinated soda; especially is this important if the patient is having frequent discharges from the bowels. The apartment should be kept perfectly quiet, the light subdued, and only the attendants should be allowed in the room. Any medicinal interference in a mild type is unnecessary. The treatment resolves itself into the arrangement of the sick-room and proper diet; milk is the most suitable food, and *fruits are not to be allowed in any case.* Even in the *mildest* case this care in diet is important, and the patient should be kept in bed until convalescence is fully established. This should be insisted upon in the mild as well as in the severe cases.

The temperature in a mild type of this fever rarely rises above 103° F.; therefore there is no necessity for resorting to antipyretic measures; frequent sponging of the surface with cold or tepid water, as is most agreeable to the patient, will be found of service. By far the larger number of cases of this fever, however, are of a more severe type, and though the treatment must be regulated by the circumstances which attend each individual case, more decided measures will usually be necessary. Typhoid fever is a disease that has certain stages to pass through, and there is great doubt whether the physician can shorten its duration by a single day, but experience warrants the belief that many lives may be saved by remedial measures, used at the proper time, and combined with judicious hygienic management.

Unquestionably one of the most important things to be accomplished is the reduction of temperature, or rather the keeping of the temperature below a certain standard. The agents which have been employed more recently for this purpose act powerfully in reducing the temperature and lessening the severity of the disease. It is claimed by many distinguished observers of the present day that the parenchymatous degenerations of the different organs and tissues of the body which are found in those who die of typhoid fever are due to the prolonged high temperature which is present during the course of this disease; but as yet there are no facts to prove this assertion, for the same parenchymatous changes are found in the bodies of those who have died of diseases the course of which was not marked by high temperature, and did not extend over a period of more than forty-eight hours. So far as we are able to determine by analogy upon what these parenchymatous changes depend, we are led to believe that the specific poison of the disease has more to do with their development than the high rate of temperature. One thing must be apparent to every clinical observer: that the injurious effects of a prolonged high temperature are early and most markedly shown by disturbances of the cerebro-spinal system. It is still an unsettled question whether these disturbances are due to the primary changes in the constituents of the blood, which always accompany a high range of temperature, or to the direct effects of the high temperature or of the peculiar poison on the nerve centres. Whichever view we adopt, the employment of those means which have the power of safely reducing temperature is indicated, and when judiciously used they have much to do with the safety of the patient.

All those means which have been employed for the reduction of temperature are included under the general term of *antipyretics*, and the treatment of disease by the use of these agents has received the name of *antipyretic treatment*. Unquestionably one of the most efficient and reliable of the antipyretic agents is the external application of cold by means of baths, packs, and affusions.

At the present time the opinion prevails, to a great extent, that the application of cold to the surface is the great antipyretic in the treatment of fever. This is no new teaching. Long ago Dr. Currie recommended the application of cold to the surface of the body for the purpose of rapidly reducing temperature, and proved that it had such an effect; yet it was never very generally practised, and soon fell into disuse, as there were no reliable indications to guide one in its application. As we now have the thermometer as such a guide, it has been resorted to more recently with considerable success. It is employed in the following manner. As soon as the axillary temperature in the evening rises above 105° F., the patient is placed in a water-bath having a temperature of 70° F. or 80° F., which is gradually lowered, by the addition of cold water or ice, until the temperature of the patient begins to fall. It may be necessary to lower the temperature of the bath to 60° F. before the temperature of the patient is affected. When the temperature begins to fall, thermometrical observations should be made every two or three minutes, by placing the thermometer in the rectum. If it falls rapidly—that is, two or three degrees in five or six minutes—as soon as the temperature has reached 103° F. the patient is to be removed from the bath; if it falls slowly, as soon as it reaches 101° F. he should be removed and immediately placed in bed. It is never safe to keep the patient in the bath until the temperature shall have reached the normal standard; for he may pass into a state of collapse, since the temperature continues to fall for some time after his removal from the bath. While in the bath, cold should be applied to the head by means of a sponge wet in cold water, or by an ice-bag. The cold pack is much less effective than the bath; but if the patient is too feeble to be removed, it may be employed with benefit. The patient is wrapped in a sheet wrung out of tepid water, and over this one wrung out of cold water is applied. The latter may be removed as it becomes warmed, and its application and removal continued until the desired fall in temperature shall be obtained. In severe cases, during the first and second weeks, after the temperature has been reduced by the application of cold to the surface, it will soon begin to rise, and continue to do so until it reaches its former height. Usually one to three hours will elapse before it begins to rise, and from two to six before it reaches its former height. It will then be necessary to repeat the baths or packs, and to continue their use, both day and night, from three to six times during the twenty-four hours, in order to keep the temperature below 103° F., and accomplish anything by this plan of treatment.

My experience in the use of cold applications leads me to believe that unless it is possible to maintain a low range of temperature after four or five baths very little is gained by their continuance. I am also convinced that, after the second week of typhoid fever, cold baths should not be employed to reduce temperature. The condition of a typhoid patient during the first and second weeks of the fever is very different from that during the third and fourth weeks. During this latter period there is great danger of collapse after a cold bath, and in several instances I am confident that pulmonary complications have been the result. In some cases when the patient is placed in the cold bath, the temperature will immediately begin to fall; in other cases there will be a gradual reduction of temperature as the water is made cooler. In certain severe cases a patient may be kept in a bath of the temperature of 60° F. for the space of half an hour without the temperature falling a degree. These cases are exceedingly grave in character, and the bath should be used with great care.

There is *no remedial agent which requires greater care* and judgment in its use than the cold bath, yet, doubtless, when judiciously employed, the lives of many typhoid patients may be saved, and it is equally certain that when injudiciously employed many lives may be destroyed. The general condition of a patient and the stage of the fever must be considered; also the effects of the first few baths must be carefully noted. Should a patient's temperature range at 104° F. or 105° F., it is no positive indication for the resort to a cold bath, or that a cold bath is the best agent to be employed for its reduction. If the patient after the second or third bath s more quiet, has less delirium (if delirium previously existed), if his breathing becomes easy and natural, if the heart's action is more regular and forcible, and he falls asleep and perspires, there can be no question in regard to the beneficial effects of the bath. If, on the other hand, the bath is followed by feebler heart's action, by dusky cheeks, by rapid respiration, and by coldness of the extremities, from which condition the patient rallies slowly and imperfectly, it is certain that, however high the temperature may range, harm will be done by continuing the baths. When the extremities are cold, or there is profuse hemorrhage from the bowels, or when from any cause there is great feebleness of the heart's action, and especially in the case of aged persons, cold baths are contra-indicated.

In a few instances the temperature can be very rapidly lowered by the application of the cold coil to the abdomen, and it is my habit in all severe cases to apply the cold coil early and continue its use until the temperature curve indicates that it is no longer necessary. The use of cold baths will thus be avoided in many cases. In addition to its beneficial effect on the intestinal changes which constitute such an important element in the history of this fever, the cold coil often has great power in reducing the general heat of the body. I have also in some instances found the body temperature rapidly lowered by injections of ice-water into the rectum. Care must be exercised that the cold injections are not administered too rapidly or in too large quantities. Although this mode of abstracting heat and lowering the body temperature is never so effective as by baths and packs, still it has this advantage, that no such compensating increase in the production of heat follows the use of the cold injections as follows the cooling of the external surface by the baths. In many cases the extreme obstinacy of the fever, which resists the most systematic use of cold, as well as the fact that some patients cannot bear a sufficiently frequent repetition of the baths to effect the desired result, or that there may be contra-indications to their use, necessitates the employment of other means for the reduction of the body temperature.

The use of the internal antipyretics has undergone many changes within a short time. Formerly it was my practice to employ sulphate of quinine almost exclusively in the treatment of typhoid fever. Its antipyretic power is established beyond question, and I still regard it as a most valuable agent in many conditions. When used as an antipyretic it must be given in large doses. Its use may often be combined advantageously with that of cold baths, the quinine being given when the patient is removed from the bath. Thus used it will delay the return of the previous high temperature.

Antipyrin and antifebrin have recently been largely employed in the treatment of all forms of fever, and often with little discrimination as to their action.

Of the two I prefer antifebrin, and have found it so valuable that it has largely supplanted quinine in my treatment of typhoid fever. I prefer to give it in five to ten grain doses three or four times in the twenty-four hours rather than in one large dose. It may be employed in connection with the cold baths in the same manner as quinine. It has seemed to me not only to lower the temperature, but to exert a most favorable influence upon the cerebro-spinal disturbance. The same general rules are to be followed as govern the use of all antipyretics. If, during the third and fourth weeks, these means fail to reduce the temperature, from ten to twenty grains of powdered digitalis may be administered within twenty-four hours, unless the pulse is very frequent and irregular—when its use is contra-indicated.

Digitalis should be employed only when some other antipyretic is given. It seems to increase their power, but has little or no effect when administered alone. The use of all antipyretic remedies must be persisted in until the desired end—the reduction of temperature—is accomplished; but the peculiarities of each patient must be studied, and these agents must be so administered as to suit each individual case. The satisfactory results obtained by the systematic use of these remedies justify their employment; but the exact rules which are to govern one in their use, as to manner and time, can only be determined by experience.

If the temperature of a patient can be kept below 103° F., during the first two weeks of the fever, the *first* and perhaps the most important thing in the treatment of this disease will be accomplished.

Toward the end of the second, or during the third week, signs of failure of heart-power begin to manifest themselves; although the temperature may not rise higher than 101° F., the pulse frequently becomes extremely feeble and irregular and reaches 140 per minute, while the first sound of the heart becomes inaudible at times; the surface is cool and moist; the patient complains of a sense of exhaustion, and perhaps is unable to turn in bed; muscular tremors, dry, brown tongue, and all the symptoms which indicate failure of vital power are present.

Under these circumstances the use of stimulants seems to be urgently demanded.

A few simple rules govern their administration.

First. They should never be administered indiscriminately—that is, simply because the patient has typhoid fever.

Second. When there is reasonable doubt as to the propriety of giving or withholding stimulants, it is safer to withhold them, at least until the signs which indicate their use become more marked.

Third. In every case, but especially when stimulants are not clearly indicated, the effect of the first few doses should be carefully noted. There are few whose experience in the treatment of typhoid fever is such as to enable them to determine positively, from the appearance of the patient, when the administration of stimulants should be commenced.

If under their use the tongue becomes dry, the patient more restless, the delirium more active, the temperature higher, and the pulse more frequent, it is very certain that stimulants are contraindicated. If, on the other hand, the pulse becomes fuller and more regular, if the first sound f the heart is more distinctly heard, or if, having been absent, it returns, f the restlessness and delirium are less marked, the tongue more moist, nd the patient more intelligent, it is equally certain that the time for administering stimulants has arrived. When their use is once begun, it is of he greatest importance to administer them at stated intervals, especially during the night. In a severe case of typhoid fever, free stimulation, just at a critical period (which may not last more than twenty-four hours), will often be followed by a refreshing sleep, and the patient may rapidly pass from an apparently hopeless condition to one of convalescence.

The *third* important thing to be accomplished in the management of typhoid fever patients is the maintenance of nutrition. The principal effects of the typhoid poison are manifested in the changes which take place in the lymphatics of the gastro-intestinal tract. Experience has taught us that the enfeeblement of the digestive and assimilative powers, due to these glandular changes, which is manifested from the very commencement of the fever, renders the digestion of solid food impossible, and for a long time it has been the rule of the profession to allow typhoid fever patients only liquid food. There has been, and still is, great diversity of opinion in regard to the special articles of diet best suited to this class of patients. There is no disease in which a waste of all the tissues of the body goes on so rapidly as in typhoid fever.

Milk is an article of diet which furnishes the elements of nutrition necessary to repair this rapid waste, and there are not the objections to its use which are against animal broths and gruels. Although there have been, and still are, in some quarters, strong objections against its use as an article of diet in fevers, recently it has been regarded with more favor, and those who have had most extended opportunities for testing its nutritive qualities have come to regard it as the only article of diet required by typhoid patients. In it we not only find all the elements required for repairing the rapidly wasting tissues, but they are in a condition to be most readily assimilated by the enfeebled digestive apparatus. In order that it shall not become distasteful to the patient some variations must be made in its preparation. It may be simply curdled, boiled, frozen, slightly fermented, or mixed with lime-water, seltzer, or some other mineral water, and various palatable preparations can be made from milk which has been partially digested with pepsin or pancreatin. If agreeable, buttermilk may be substituted for a time. The quantity of milk is not limited; the patient may take all his stomach will digest—usually patients will take from four to six pints in the twenty-four hours. After the patient has passed into the fourth week of the disease it may be necessary to administer cream and the yolks of eggs in connection with the milk.

I now come to the treatment of the accidents of the disease.

Diarrhœa.—The poison which produces this fever unquestionably has a specific action upon the intestinal glands and lymphatics. It is here that we find the characteristic lesions of the disease, and it is scarcely questioned that the typhoid poison, to a great extent, gains entrance to the system through these glands and lymphatics, and here produces the primary irritation. Following the irritation and inflammation of the follicles, other portions of the mucous membrane become involved, and a catarrhal inflammation of the mucous membrane of the intestinal tract follows. The necessary consequence of this is a diarrhœal discharge, which is simply an indication that these intestinal changes are going on; it is not due to the elimination of the typhoid fever poison, but to the inflammation which the fever poison has excited in the intestinal glands. When the diarrhœa is present in the earlier period of the disease, it is better to let it alone, as during the first and second weeks the danger is very slight. It has been proposed to treat this diarrhœa, which makes its appearance early in the disease, with alkalies, bismuth, pepsin, etc. It is claimed that if these remedies be administered, diarrhœa can be prevented, or, if it already exists, that it can be controlled. Theoretically, I see no reason for employing alkaline remedies, for the diarrhœal discharges are always alkaline, and from clinical observation, I am convinced that bismuth, pepsin, etc., have little or no effect either in controlling the diarrhœa or in preventing the intestinal changes which produce it.

When diarrhœa commences late in the disease (during the latter part of the third, or during the fourth week of the fever), it is of a very different character from that which occurs during the first and second weeks. Ulceration of the intestinal glands, and perhaps sloughing has been established, and, in addition to the extensive local changes, there is a septic element which enters into the causation of the diarrhœa at this stage. Besides, the increased peristaltic action of the intestines, which attends the diarrhœa, favors an extension of the inflammatory processes to the peritoneum, especially that portion which covers Peyer's patches. In view of these facts, the diarrhœa should be arrested or held in check. For the accomplishment of this, there is but one remedy which can be relied upon —that is opium. My experience is against the use of astringents. If opium will not arrest it, one may expect little aid from astringents combined with opium as they are usually administered. The use of opium is objected to by some, who claim that it diminishes the power of the heart's action ; but in this disease, when administered in small doses, it seems to me to increase rather than diminish the heart-power. It is acknowledged that opium, more than any other drug, arrests the peristaltic action of the intestines ; and that is what we wish to accomplish when diarrhœa is present during the third and fourth week of typhoid fever.

When during convalescence diarrhœa is persistent, the patient should be kept in bed and some of the vegetable astringents, as catechu or hæmatoxylon, may be employed.

Tympanitis.—When this has proved a distressing symptom, I have usually found relief to be obtained by the application of turpentine stupes to the abdomen. Some claim that if turpentine be administered internally from the beginning to the end of typhoid fever, tympanitis and the intestinal changes which lead to it and to the diarrhœa are much less severe. I am confident that the turpentine treatment, as it is called, does not have the controlling influence over this fever which has been claimed for it ; but I am certain that it is our most reliable agent for the relief of the tympanitis.

Intestinal Hemorrhage.—When this occurs early in the fever, it usually requires no treatment ; but when it occurs during the third or fourth week, or after convalescence is apparently fully established, it must be arrested as promptly as possible. The occurrence of severe intestinal hemorrhages may sometimes be prevented by keeping the patient in bed. A typhoid fever patient should not be allowed to get out of bed from the beginning of the attack until convalescence is fully established. Especially is this of importance if the case is a severe one, and attended by symptoms that indicate extensive intestinal lesions. When hemorrhage from the intestines occurs during the third or fourth week of the fever, it is most surely controlled by the administration of opium in small doses at short intervals. Absolute rest of the body must be insisted on, the patient must not be turned on the side or moved in bed, and an ice-bag should be applied over the abdomen. I doubt if any good results can be accomplished by the use of astringents, either by enemata or by the mouth, as it is not known that they even reach the seat of the hemorrhage, although gallic acid and the persulphate of iron are usually recommended in cases of intestinal hemorrhage occurring in typhoid fever. If the hemorrhage is profuse, it may be necessary to keep the patient under the influence of opium for a week or ten days; in such cases the internal use of turpentine in connection with the opium will be found of service.

Peritonitis.—When perforation of the intestine occurs, the case may be regarded as hopeless; death takes place usually within twenty-four hours, as the result of general peritonitis; no plan of treatment avails anything. If the peritonitis occur without perforation, from extension of the inflammatory process from the intestinal ulcers, bringing the patient rapidly into a state of semi-narcotism and holding him there for five or six days may prevent the occurrence of the perforation, and thus save life. Such a case is to be treated in every respect as one of localized peritonitis. After recovery from an intestinal hemorrhage or a localized peritonitis in typhoid fever great care should be exercised in the administration of cathartics or enemata. The bowels will move spontaneously after a time, even though the use of opium be continued, and no harm will follow should two or three weeks pass before they do so. When the stomach is irritable, the hypodermic injection of morphine is preferable to opium administered by the mouth.

Bronchitis.—For the catarrh of the larger bronchial tubes no special treatment is required; but, if the bronchitis becomes capillary, great relief will be obtained from the application of dry cups to the chest and the internal administration of carbonate of ammonia. Vapor inhalations will also be found of service in severe cases.

Pneumonia.—The pneumonia which complicates typhoid fever in nearly every case is lobular in character. The signs which indicate its occurrence are sudden rise of temperature, increased frequency of respiration, and the physical signs of localized pulmonary consolidation; cough and expectoration are rarely present. Its occurrence is always an indication that stimulants should be administered. If they are being administered, they should be increased in quantity. To prevent or relieve the hypostatic congestion of other portions of the lung, which frequently accompanies pneumonic development, the heart-power must be increased, and the position of the patient changed.

Laryngitis.—For the relief of the laryngitis which occasionally complicates typhoid fever, a small blister may be applied on either side below the angle of the jaw, and the whole neck enveloped in a poultice. If these measures fail, and suffocation appears imminent, tracheotomy should be resorted to without delay.

Sub-acute gastric catarrh, occurring as a complication during convalescence from the fever, can only be managed successfully by giving the stomach rest as far as possible, restricting the diet to a single tablespoonful of milk at a time, and applying hot fomentations over the epigastrium.

Bed-sores.—The severer forms of bed-sores are the most intractable complications one has to combat. Fortunately the severer forms are much less frequently met with under the more recent plan of treatment. Scrupulous cleanliness is the principal means for preventing their development. So long as there are no erosions, the parts should be frequently bathed in spirits of camphor, and the points of attack should be relieved from all pressure. If the sores penetrate the integument, they should be frequently washed with a weak solution of carbolic acid, or brushed over with equal parts of balsam of Peru and balsam copaiba, and afterward covered with dry lint, or lint covered with vaseline. The most unfavorable cases are those in which the point of pressure caused by the weight of the body becomes gangrenous. In such cases, a continuous warm bath is recommended by some. As soon as sloughing takes place, and the parts separate, they should be dressed with lint saturated with balsam of Peru and carbolic acid.

Constipation.—As already stated, diarrhœa is usually present in the early period of this fever; but sometimes there is constipation. The question arises:—is the administration of cathartics ever admissible in typhoid fever? Quite diverse views are still held in regard to this question. Recently, certain observers of extended experience have claimed that there is sufficient reason for the belief that a portion of the typhoid poison lodged in the alimentary tract may be expelled by the timely administration of cathartics, and thus the severity of the fever be mitigated and its duration shortened. Recent German writers claim that *calomel* acts beneficially only as a cathartic. Those who favor the administration of cathartics recommend their use mainly during the first week of the disease. On the other hand, equally competent observers maintain that the intestinal changes are augmented and rendered more extensive by the action of cathartics, that the normal course of the fever is interfered with, and that in a large proportion of cases where intestinal and peritoneal complications occur, hypercatharsis has been induced at an early period of the fever by the administration of cathartics for the purpose of shortening its duration. My own experience leads me to exercise the greatest caution in the administration of cathartics in any stage of this fever.

I am confident that the routine practice of administering purgative medicines in the early stage of typhoid fever can only be followed by a threefold injury: *first*, the patient is weakened. *Secondly*, the local intestinal lesions are increased. *Thirdly*, perforation and peritonitis are more liable to occur.

Nervous Phenomena.—Should headache be severe, not readily relieved by fomenting the forehead and temples with warm water, or should it give place to active delirium and other severe nervous disturbances, the question presents itself:—shall anodynes be administered? If they are to be used the most reliable is opium, and usually the condition of the pupil of the eye will serve to indicate whether opium shall or shall not be administered. A contracted or "pin-hole" pupil may be considered to contra-indicate its use, though there are exceptional cases in which opium acts favorably, notwithstanding this condition of the pupil. Opium should be given with great caution whenever signs of cyanosis are present. In all cases of typhoid fever, it is safer to administer opium in small and repeated doses than to venture upon the administration of one large dose. There are other anodynes which will sometimes be of service, such as hyoscyamus, chloral and the bromides. Chloral is said to have a special value in quieting active delirium, which is sometimes so troublesome, but my own experience in its use has not been favorable.

When anodynes have failed to give relief to typhoid fever patients, who have been delirious and somnolent for days, they will sometimes become quiet and fall asleep immediately after the free administration of stimulants. Those cases in which the nervous symptoms are due to an anæmic condition of the brain, associated with a weak heart and a flagging circulation, are most likely to be benefited by the use of stimulants. In those cases in which subsultus becomes very marked, and there is general tremor, jactitation and restlessness, I have seen most happy effects produced by the use of hypodermic injections of sulphuric ether. I would use, as an average quantity, four drachms, given in injections of one drachm each, in different places. The same watchful care should be taken of a typhoid fever patient during convalescence as during the active period of the fever. The number of typhoid patients who die during convalescence is relatively large.

Death is often due to the fact that the physician has laid down no strict rules to be observed as to diet and exercise, and frequently from the non-observance of such rules when they have been given. The diet of fever patients during convalescence should be carefully watched. Only small quantities of food should be taken at a time, so that the gastric juice secreted by the enfeebled stomach may be sufficient for its complete digestion. All indigestible articles of food, and those which furnish a large amount of waste, should be strictly forbidden. An apparently insignificant disturbance of the stomach, a slight vomiting, or a moderate diarrhœa occurring during the period of convalescence, should be regarded as dangerous, for any one of these may induce a sub-acute gastritis, or lead to intestinal perforation and a fatal peritonitis. It is obvious that while the intestinal ulcers are healing much mischief may be done by improper diet. Notwithstanding the cravings of the patient's appetite, the diet must be restricted to such articles as milk, cream, gruels, jellies, and animal broths. Solid food must be strictly forbidden, especially meats, vegetables, and fruits. If diarrhœa is present during convalescence it is far safer to restrict the patient to milk and cream. All exercise, except simple walking around the sick-room, should be prohibited.

It is of the greatest importance that this class of patients should keep in the recumbent or semi-recumbent posture until the cicatrization of the intestinal ulcers is completed, which in some instances does not take place until two or three weeks after convalescence is well established. If convalescence is slow, small doses of quinine, iron, and cod-liver oil are of service. They should be given after the patient has taken food. In many cases it is important to take the evening temperature for at least two weeks after the commencement of convalescence, for by its range it will be possible to more accurately determine the exact condition of the patient. When convalescence is delayed, so that at the end of four or five weeks the patient has not regained strength, change of air is indicated.

YELLOW FEVER.

Yellow fever is an endemic *miasmatic contagious* disease, which usually appears as an epidemic. It prevails most in tropical regions, and is characterized by a yellowish discoloration of the skin. From some of its more prominent symptoms it has been called *typhus icterodes*, *black-vomit* or *hæmo-gastric* fever, *febris flava*, and also *mal de Siam*.

Morbid Anatomy.—The pathological changes of yellow fever have much that is common both to contagious and miasmatic diseases. Its most constant and characteristic lesion is to be found in the changes which take place in the liver.

The *liver* is usually slightly enlarged ; it may, however, be normal, or even slightly diminished in size. The most striking change is in its color, which has been described as butter-, cheese-, mustard-, or chamois-yellow. Sometimes it is of a chocolate or bright orange color. The change in color may be uniform throughout the entire organ, or it may occur in irregular patches of different hues. Slight extravasations of blood are sometimes found on its surface. In some few instances this change will be confined to a single lobe or a circumscribed portion of the organ. The liver-tissue breaks down readily on firm pressure, and *on section* is drier than normal, containing less blood. Small points of extravasation sometimes stud its substance.

Under the microscope the liver cells, while unaltered in shape, are seen to be filled with oil-globules, so large that at times one globule occupies an entire cell. Sometimes the change is a granular one, the nuclei of the hepatic cells being obscured ; or they have entirely disappeared.[1] This change is an acute fatty degeneration, and not an infiltration, as many suppose. The organ in its gross as well as in its minute anatomical changes resembles the fatty degeneration of the drinker's liver. Cornil and Ranvier[1] say this degeneration is secondary to a congested and ecchymosed state of the liver.

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[1] *Yellow Fever considered in its Historical, Pathological, Etiological, and Therapeutical Relations.* R. La Roche. Philadelphia, 1855. *Yellow Fever.* Fritz Haenisch. Ziemssen's Cyc. Prac. Med., vol. i.

The heart is lighter in color than normal, soft, friable and flabby. It breaks down readily under firm pressure, and resembles strongly in its gross and microscopical characters the heart of typhoid fever. The muscular elements undergo the same granular degeneration, which cannot be ascribed to prolonged high temperature, for in yellow fever the temperature is neither high nor does it persist above normal for a long time. As in typhoid, so here we are inclined to regard the degenerative changes as the result of the specific poison of the disease. The cavity of the pericardium usually contains one or two ounces of blood-stained serum. Long coagula or partly organized clots extend from the heart cavities quite a distance into the vessels. These coagula are the result of the heart-failure, and are formed during the few last hours of life. Sometimes the blood in the heart is fluid, varying in color and reaction.

The *blood-changes* are similar to those of typhoid and typhus fever, yet are more extensive than in either; the blood is of a darker color than normal, and coagulates very much more slowly and imperfectly than normal; a fact due either to a diminution in, or to a partial loss of the coagulating power of the fibrin-factors. The red blood globules are destroyed, or they are serrated and shrivelled, and in many instances broken down—this explains the yellow color of the surface which gave the name to the disease. A solution of part of the red corpuscles occurs, and the hæmatin is changed into pigment. This condition of the blood also explains to a certain extent the degenerations which are found in the different organs of the body. Very soon after withdrawal the blood undergoes ammoniacal decomposition, due in part to the altered relation of its salty constituents. Some affirm that the blood contains free ammonia. It contains no free pigment.

The *mucous membrane* of the œsophagus, stomach and small intestine is always the seat of a more or less acute catarrh. The veins are varicose and turgid, often giving rise to arborescent injection of the membrane; and ecchymotic spots of extravasation irregularly stud its surface. Hemorrhagic erosion of the stomach is sometimes present, and throughout the whole intestinal tract there is often a considerable quantity of dark-colored fluid blood, the stomach, however, containing matters similar to those vomited during life. The gastric mucous membrane is also not infrequently found thickened, softened, and reddened. The mucous membrane lining the larynx also suffers a catarrhal inflammation; and ecchymotic spots are found on the lining membrane of the bladder.

The *lungs* are almost constantly the seat of infarctions, and these are occasionally quite numerous. When diffuse, pulmonary apoplexy occurs, and when a large portion of a lobe is involved, the lung-tissue will be broken down and large blood-clots will occupy the space.

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[1] *Patholog. Histology.*

The *pleuræ* are sometimes covered with ecchymotic spots, and occasionally there is a blood-stained serous exudation into the pleural cavity.

The *brain* and *cord*, if at all altered, are only slightly hyperæmic. Punctate extravasations may occur in the meninges; and some affirm that an abundant serous exudation is often present in the lumbar and sacral regions, attended by an inflammation of the membranes of the cord at the same point, with more or less intense inflammation of the neurilemma of the nerves in the cœliac and hepatic plexuses.

The *kidneys* are the seat of parenchymatous inflammation, which rapidly passes to the stage of fatty metamorphosis. There are sometimes small abscesses in the parenchyma. On microscopical examination oil-globules are seen to fill the tubules, whose epithelium is sometimes desquamated, or the seat of fatty or granular change. Occasionally the tubules are filled with broken-down epithelium. The pelves of the kidneys and the ureters are frequently the seat of an acute catarrh.

The *spleen* may be slightly enlarged; but is usually softer, more friable, and darker than normal.

The *skin* varies in color from a bright golden-yellow to a dark orange. Petechiæ, ecchymoses, vesicles, pustules, and large patches of extravasation may be found upon the surface of the body. The mucous membranes are not infrequently of a distinctly yellowish tinge.

The *gall-bladder* may or may not be increased in size; it commonly contains a moderate quantity of dark-colored bile, and its mucous surface exhibits spots of punctate extravasation as well as arborescent vascularity.

The *ovaries* and *uterus* very frequently contain a considerable quantity of extravasated blood.[1]

Etiology.—As yet the specific microbe of yellow fever has not been discovered, although two or three have been claimed by different investigators as pathogenic of this disease. From its clinical history we are led to believe that it is to be included in the class of diseases whose microbic origin has been determined.

Yellow fever is rarely met with beyond the limits of 40° North and 20° South latitude; it prevails in the West Indies and eastern part of the Western Hemisphere far more frequently than any other region, and the *locus*, if we may say so, of the malady is the Antilles. In these places it is endemic, and to a comparatively slight extent it is so in certain portions of Europe and Africa. Commercial seaports are pre-eminently the starting-points of great epidemics; it is sometimes circumscribed within very narrow limits in the seaports. Crowding is one of the essentials to its development. The average temperature of the locality where it prevails must be at least 73° F.; there must be a certain amount of moisture; and animal and vegetable matter must undergo decomposition, either on the surface or in the substance of the soil. On ship-board there may be the greatest uncleanliness, yet the fever will not appear on the vessel till it has touched land in an affected port or been brought into communication with a ship already contaminated.

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[1] *Traité des Maladies Infectieuses: Maladie des Marais, Fièvre Jaune, Maladies Typhoïdes, Fièvre Typhus des Armées.* Wilhelm Griesinger, Paris, 1868.

The time of year during which the fever prevails varies with the climate and temperature; in the United States it usually appears in July and August, to disappear with the first frost. The epidemic in New York city in 1795 began in August and terminated in October. When the prevailing winds are southeasterly, the development and spread of an epidemic are favored; northwesterly winds check or arrest it. As has been mentioned, a severe frost or a "freeze" puts an end to the further progress of the disease when it prevails under the most propitious circumstances for its development.

There is much reason in the arguments of those who contend that yellow fever is an "acclimation" disease. First (and here, however, it should be remarked that the disease is indigenous in some regions), certain islands and seaport towns along our Southern coast always suffer from an epidemic whenever certain atmospheric conditions exist; a resident of one of these places where yellow fever is indigenous is far less liable to have the disease than a stranger, especially one from the North. One attack is usually, not absolutely, a protection against a second. The disease is especially liable to appear in those localities where a severe type of pernicious fever has prevailed, and after a warm, rainy season rather than after a dry, cool one. Whether the fever is epidemic or endemic, and whether the locality is one frequently visited or one in which the disease is indigenous, *sporadic* cases are of very rare occurrence.

The negro race has a marked immunity from this fever. Age and sex exercise no influence upon the etiology. Occupation seems to have some effect in its production, since those who work over, or near, hot fires are stricken much oftener than those who work in unhealthy, filthy surroundings. Exposure to cold and wet, alcoholismus, and venereal excesses here, as elsewhere, render individuals more liable to the fever.

In regard to the *nature* of yellow fever poison, some assert that it is a malarial miasm, modified by the person in whom it lodges. It is in many respects similar to the poison of typhoid, both in etiology and the manner of its conveyance. It is unquestionably a *specific* poison, which differs essentially from the poison of every other fever. Typhoid, malarial and yellow fever may all prevail at the same time in the same locality, but one will never merge into the other; each runs its own individual and peculiar course. All chemical and microscopical research has, as yet, failed to discover *what* the poison is; but we are led from its mode of conveyance and from the conditions of its development to believe that it has the elements both of a miasm and a contagion.

There are three leading doctrines in regard to the contagious character of yellow fever:—*first*, that it is contagious, like small-pox and scarlatina; *second*, that it is non-contagious, and never directly transmitted from the sick to the healthy; and *third*, that when yellow fever is prevailing in a locality, it may be carried from one person to another *in that* locality. The last is the doctrine of contingent contagion. One who has seen the fever in hospitals needs no argument to prove that it is not directly contagious. Some claim that yellow fever poison, though not *directly* transmissible from the sick to the healthy, becomes infectious when brought in contact with decomposing animal and vegetable matter. It is well established that epidemics of yellow fever only occur in those localities where decomposing animal and vegetable matter is present; and when men are crowded together in shops and around the docks and wharves of seaports, or in the filthy streets and dwellings of such localities. In some few instances evidences exist that yellow fever breaking out in the hold of vessels has been circumscribed to well-defined and very narrow limits by free ventilation. There are ample facts to sustain the belief that this fever is infectious only when the atmosphere has become loaded with the emanations of animal and vegetable decomposition to which has been added the specific yellow fever poison. Under such circumstances, the disease may be propagated from the sick to the healthy.

Whatever view is taken of the contagious or non-contagious character of yellow fever, all observers agree that it is *portable*, that it can be conveyed from one place to another by means of clothing and merchandise and in the holds of vessels. That whenever the poison is thus introduced into healthy localities which are suited by temperature to its reproduction, and where there is animal and vegetable decomposition, it rapidly reproduces itself, and thus epidemics of yellow fever occur in localities that otherwise would be free from the disease.

There is no doubt that the poison of yellow fever retains its vitality for a very long period; and with favoring conditions may cause an epidemic in places very remote from the origin of the poison. The poison is also capable of great concentration, for short exposure to the contaminated air that often fills the holds of ships on which yellow fever is prevailing will be followed by the fever in a few hours. Ordinarily there is little danger in visiting those sick of yellow fever if there is free ventilation, and one does not remain in the infected locality for a long time. The period of incubation varies in duration from twelve hours to four or five days; when the exposure is followed in a few hours by the fever, the fever poison must necessarily be very concentrated. The activity of yellow fever poison is destroyed by cold; one or two hard frosts will arrest a yellow fever epidemic. Some claim that epidemics of yellow fever are self-limiting, rarely exceeding sixty or seventy days in their duration. There is not, however, sufficient proof to establish this statement.[1]

Symptoms.—As in typhoid fever, there are mild and severe cases of yellow fever; but they differ only in degree, *not* in kind; the clinical history of both is the same.

Prodromata may occur; but headache, anorexia, lassitude and pains in the limbs cannot be reckoned as characteristic of the fever, and only when these occur during an epidemic are they especially significant. Whether premonitory signs have or have not been present, the disease commences with a chill, distinct and severe. In a few instances a series of rigors takes the place of distinct chills. Sometimes persons while appearing in perfect health are seized with a severe chill, and immediately become seriously ill and take to their beds in a most dejected manner. Following the chill there is nausea and vomiting, the face is flushed, the conjunctivæ are injected, there is circumorbital headache, and violent pains in the bones, back and limbs, especially in the calves of the legs. The eye has a peculiar lustre and a staring look. The course of the fever is the same in the severe and in the mild cases.

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[1] In this connection see: *The Cause and Prevention of Yellow Fever, in the Report of the Sanitary Commission of New Orleans.* Dr. E. H. Barton, New York, 1857. *Mémoire sur la Fièvre Jaune qui en 1857, a Décimé la Population de Montevideo.* A. Brunel, Paris, 1860. *Account of the Yellow Fever which occurred in the City of New York in the year* 1822. Dr. James Hardie, New York, 1822. *Remarks on the Epidemic Yellow Fever on the South Coasts of Spain.* Dr. R. Jackson, London, 1821.

The *temperature* rises rapidly after the chill to 103° or 104°F.; the limits vary, but yellow fever is not a disease of high temperature. In a few epidemics the initial rise in temperature has been as great as 110° F., but these are phenomenal occurrences. At the end, or beginning, of the third day the maximum fever will have been attained; in our country this is rarely more than 104° or 105° F. Between this period and the fourth day of the disease slight variations, hardly amounting to distinct remissions are present; on the fourth day there is a rapid defervescence; it is not an intermission but a *remission*, for the temperature only falls to 100° or 101°F. The period of remission lasts from a few hours to two or three days, after which a second rise begins, one that does not take place quite as rapidly as the first, and is not usually preceded by a chill or rigors; and a temperature of 104° or 105° is again reached. The temperature now remains stationary from one to two days, it then falls to normal and remains so. This last fall is, like the first, marked by a very sharp temperature curve. The range of temperature is important, for it divides this disease into three parts; *first*, the stage of invasion, the febrile stage or period of exacerbation; *second*, the stage of remission, calm or passive period; and, *third*, the stage of the second exacerbation or collapse.

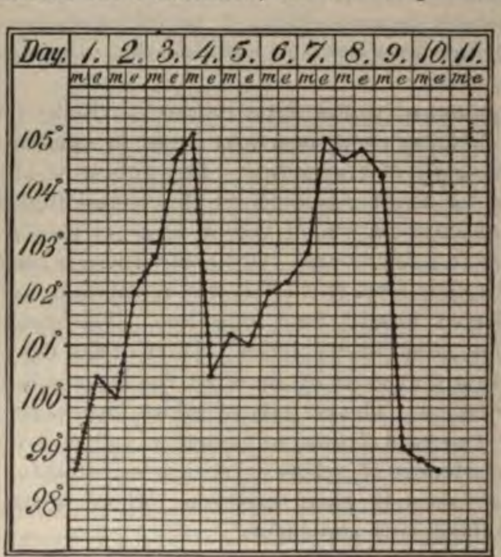

FIG. 157.
Temperature Record in a Case of Yellow Fever.

The *pulse* in yellow fever is peculiar. It rarely exceeds 110 beats per minute, thus differing from that of other fevers in which the rule is an increase of five beats for every one degree rise of temperature. Indeed, in mild cases, the pulse-rate may only be five or six beats above the normal. It has been observed to fall much below the normal, as low as 40 and sometimes 30 in a minute. The "feel" of the pulse is as if the arteries were distended with gas, and hence the name, "gaseous pulse," is not inappropriate. It is compressible and of an uncertain volume, offering no resistance, so to speak, to the touch.

The *skin*, as soon as the temperature begins to rise, may be either dry or bathed in a copious perspiration. Following the chill there is sometimes an abnormal coldness on the surface, while rectal thermometry shows a marked rise in the temperature. At the close of the first, or beginning of the second day, the body emits a peculiar corpse-like odor. About the third day the skin begins to assume a jaundiced hue, noticed first in the sclera and then spreading over the whole body. It is a dark jaundice, like that of pyæmia, and is to be regarded as hematogenous and not hepatogenous. Those who maintain that the jaundice is due to retention and reabsorption of bile have no proof to offer, since evidences of mechanical obstruction to the outflow of the bile are among the rarest *post-mortem* appearances. The true etiology is found in the change which takes place in the blood. The pigment thus formed is deposited in the tissues, and causes a true hematogenous icterus. The perspiration now stains the linen yellow. This jaundice is not always present in yellow fever, but when it becomes a symptom it does not run into the period of convalescence. In the third stage the jaundice assumes a mahogany hue.

Vomiting.—Immediately following the chill, nausea and vomiting are present. First the contents of the stomach are voided, then a yellowish green matter; when the latter color is present the vomiting becomes projectile in character, and the ejected matter has an alkaline reaction and is fluid. The alkalinity is due to ammoniacal decomposition. The vomiting is accompanied by burning pains at the xiphoid cartilage. If the vomiting continues without any other change in the matter vomited, it is an evidence that the fever is going on to recovery; in severe cases the characteristic "black vomit" is present, the result of hemorrhage into the stomach. This vomit is brownish black, semi-fluid, with a glistening reflection, and varies in amount from a mere trace to many pints. It may occur on the second or third day of the fever, but usually it does not come on until about forty-eight hours before death, or on the day of death; it occurs only in about one-third of the fatal cases. It undoubtedly occurs more frequently in yellow fever than in any other disease, but it differs in one of its constituents from a similar material which is sometimes vomited other diseases where small capillary hemorrhages occur in the stomach. Microscopically[1] it is seen to be made up of blood corpuscles, degenerated mphoid cells, fat cells, epithelial cells from the mucous membrane of e stomach, fine granules of pigment, aggregated non-granular masses, nd serous fluid. The action of the gastric juice is such that the coloring matter escapes from the corpuscles as small granular or rounded masses. It is claimed that the black vomit of yellow fever is specific, in that it contains a peculiar microscopic vegetable organism. This is yet lacking confirmation. The enfeeblement of the walls of the capillary vessels results from the pathological blood-conditions, and as qualitative alterations are likewise added, hemorrhagic extravasations occur in the stomach, and on other mucous surfaces. The hemorrhages from the nose and gums that so frequently occur, and fluid blood in the discharges from the bowels are caused by the same changes as cause the gastric hemorrhages. Very rarely hemorrhagic extravasations occur during life from the respiratory organs, the genitals, the skin, and the meatus auditorius externus.

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[1] *Microscopic Researches in the Black Vomit of Yellow Fever.* Dr. M. Michell. Charleston Medical ournal, 1853.

Urine.—Early in the disease the urine is scanty, acid, and slight traces of albumen may be found. Later, when the jaundice appears, its reaction is alkaline, and bile pigment is present; as the disease progresses it becomes more abundant; if not present before, it makes its appearance during the stage of remission; in all severe cases, leucin, tyrosin and fatty casts will likewise be found. Entire suppression of urine is of frequent occurrence in severe cases. Patients with black vomit may recover, but a fatal result almost certainly follows urinary suppression. In cases where the yellow fever poison is concentrated and the nervous symptoms are prominent, suppression of urine may exist from the onset, but it usually does not take place until the second exacerbation. Uræmic toxæmia is then added to the yellow fever poison, and the condition is almost necessarily hopeless. The perspiration in this condition has a urinous odor.

The *countenance* in some cases is almost diagnostic: the eyes are lustrous and staring, the face is flushed, the conjunctivæ are injected, the intense conjunctival congestion giving the eyes the appearance of two balls of fire set in a face of a dusky, deathly hue; this gives to the countenance a remarkable expression of dejection and dulness.

The tongue is covered at the outset of the fever with a thick, yellowish white coating, except at the tip and edges, which remain red. It is often indented by the teeth; and as the disease advances may become dry, brown, cracked and fissured, resembling the typhoid tongue. The buccal mucous membrane is bright red at first, subsequently becoming œdematous.

The bowels are usually constipated, but when diarrhœa does occur, fluid blood is apt to be mingled with the discharges. Sometimes when intense jaundice is present, the stools are clay colored, but this is an accidental circumstance.

The mind is usually clear to the last, but when delirium sets in it will be wild and accompanied by a desire to get out of bed. The patient lies in a state resembling collapse, his features shrunken, indifferent both to his own condition and to what is occurring about him.

Pain is quite severe over the lumbar and epigastric regions which are exquisitely sensitive to pressure; convulsive twitchings of the muscles, and diaphragmatic contractions are often present before death. In favorabl cases all the severe symptoms distinctly remit on the second day after the beginning of the stage of the second exacerbation, and then follows a protracted convalescence, and it is with the greatest difficulty that the stomach will retain the blandest food. When death is to follow, the vomiting persists, the urine becomes less and less in amount and richer in albumen, and uræmic coma, or wild delirium ends the scene. Just before death, in some epidemics, the temperature falls; hence the name *algid yellow fever*. But whether coma, algidity, delirium, suppression of urine, or black vomit is the predominant symptom in an epidemic, the disease is the *same specific* fever. The mortality varies as much in different epidemics as the prominent symptoms do.

Differential Diagnosis.—Yellow fever may be confounded with *acute yellow atrophy* of the liver, *relapsing, bilious remittent, continued malarial* fever, and the *icteric* variety of *pernicious* fever.

The diagnoses of acute yellow atrophy of the liver and yellow fever have already been considered.

Relapsing fever is an inland disease, as a rule, while yellow fever is essentially a coast disease. In relapsing fever the temperature rises to a high point, often 107° or 108°, the pulse keeping pace and running up to 140 or 150 beats per minute; in yellow fever a pulse of over 110 is very rare, and the temperature averages 104°, often lower. Jaundice and the peculiar-colored "yellow fever face" are early symptoms in this disease; while there is no change in the face in relapsing fever and jaundice is a very late symptom. Relapsing fever has a true intermission, while yellow fever has only a remission. The spleen is markedly enlarged and tender in relapsing fever; in yellow fever it is normal. During the pyrexial period spirilli are found in the blood in all cases of relapsing fever, and are absent from yellow fever. Bronchitis is a *very* common complication of relapsing fever, while pulmonary complications are very rare in yellow fever. Finally, relapsing fever is propagated by contagion, and yellow is not.

Yellow fever is a portable disease, and usually prevails in cities and along the coast; *bilious remittent* fever is not portable, and is a disease of the country and inland towns. The pulse-rate is 120 or 130 in bilious remittent; in yellow fever it is rarely over 110; the temperature is 105° or 106° in bilious remittent, and rarely exceeds 104° in yellow fever. The liver is enlarged in yellow fever, and normal in size in bilious remittent; the spleen is invariably enlarged in remittent and unchanged in yellow fever. There is projectile vomiting in yellow fever, while in bilious remittent it is retching in character. In twenty-four hours a remission occurs in bilious remittent, while in yellow fever the remission does not occur until the fourth day. The urine is rarely albuminous in remittent fever, while even in mild cases of yellow fever albumen is rarely absent. The mind is clear in yellow fever, while a patient with bilious remittent is dull and delirious. The difference in the invasion of the two diseases, the countenance, the existence of the hemorrhagic tendency, and the history of the epidemic are sufficient to distinguish yellow fever from the so-called yellow type of remittent fever.

In *continued malarial* (so-called typho-malarial) fever the temperature is higher than in yellow; there is diarrhœa, which is absent from yellow fever, and the spleen undergoes marked enlargement. Yellow fever, on the other hand, is attended by albuminuria and a peculiar facial aspect that are both absent from all cases of continued malarial fever. There is periodicity in the variations in temperature in continued malarial fever, and the disease is continuous over two or three weeks; while in yellow fever there are slight and irregular variations in the fever, and a distinct *remission* on the fourth day, which removes all doubt. Pain in the right iliac fossa is much more marked in continued malarial than in yellow fever. The history of the epidemic, the portability, and other etiological points will also often greatly aid in making the diagnosis.

Prognosis.—The mortality-rate differs in different epidemics; the highest mortality is given as one out of every three; while in mild epidemics only one out of fifteen or twenty dies. The average duration is six days, but in cases where a concentration of the poison overwhelms the system at the very onset, death may occur within twenty-four hours, and between this time and six days there is a varying number of fatal cases. The conditions that render the prognosis *unfavorable* are early high temperature, a severe period of invasion, deep jaundice, scanty urine containing albumen and *casts*, black vomit, intense pains over and irritability of the stomach, a gaseous pulse, delirium, and, worst of all, suppression of urine.

Among the *favorable* signs are diminution in the quantity of albumen, a quiet stomach, slight and late jaundice, a moderate degree of fever, and fewer attacks of black vomit. A positive prognosis is best withheld; but "black vomit" and complete suppression render a case hopeless. Yellow fever, in some epidemics, is complicated by numerous boils and abscesses, and by cellulitis and inflammation of the parotid gland, perhaps terminating in suppuration. Regarding convalescence, it may be said, however quickly it may be established, it is longer than in any other disease in proportion to the length of the fever. Indeed it is often two weeks after the final fall in temperature before the patient begins to mend, and five or six months may have to elapse before he is entirely well. Death may result from rapid overwhelming of the system with the poison, *i.e.*, from the effects of the blood change, from uræmia, black vomit, suppression, exhaustion or asthenia.[1]

Treatment.—*Prophylaxis* is, in a great measure, summed up in the word *quarantine*. A strict quarantine, that should include not only individuals but also all articles that have been near the infected person or spot, would be very desirable. This does no harm to the sick; they may be removed to a hospital at once, after disinfection, for the disease is not contagious. To go into the details of quarantine, of ship and hospital disinfection, would be out of the domain of this work. A person who is in the yellow-fever region can take the best prophylactic measure—removal from the neighborhood. When this is impracticable, sulphate of quinine may be taken, and all predisposing causes avoided as far as possible. Mercury is by some regarded as an efficient means of prophylaxis.[1] The variability of the mortality-rate has been referred to. Blood-letting, mercurials, stimulants, and quinine,—these are the four chief methods that have been tried.[2] Blood-letting, even to the extent of 180 ounces at a time, was formerly practised, but has been abandoned, as not only wrong in theory but harmful in practice. Mercurials are exhibited to-day only for catharsis at the commencement. Stimulation is bad in excess; and quinine is of no avail for any but prophylactic measures, if even here it possesses as much efficacy as theory attributes to it. Recently carbolic acid has been added to this list, but it has had so slight a trial that nothing can be said *pro* or *con*, except that it is likely to go the way of all specifics.

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[1] "*Relation de la Fièvre Jaune survenue a Saint-Nazaire en* 1861." M. F. Melier, Paris.

The plan of treatment which seems, at the present state of our knowledge, most reasonable, may be called a *diaphoretic and expectant* plan, the diaphoresis looking toward the relief of the grave kidney trouble, and hence tiding over the most serious point in the fever. When a patient is stricken with the fever, apply counter-irritation over the kidneys, and at the same time administer ten grains of quinine along with fifteen or twenty grains of calomel. The body should be covered with flannel and slightly heated, moderate diaphoresis being continually kept up by these methods. At the same time the air must always be fresh; close quarters are always contraindicated. The nausea and vomiting may be controlled by eating cracked ice, drinking milk and lime-water, or by small hypodermic doses of morphia. The restlessness, tossing, and jactitation which are so exhausting in some cases, and which probably arise from the action of the urea in the circulation on the nerve centres, are best controlled by hypodermic injections of morphine. Full doses of opium, producing as they do free diaphoresis, may also be administered, unless the kidney lesions are very grave. Suppression is treated by the usual methods, large doses of turpentine being given. In the last epidemic ʒj of turpentine in sugared water was given every four hours in the case of a negro, and recovery followed.

In copious hæmatemesis styptics can be given cautiously, and cold compresses may be applied over the epigastrium. When the various discharges have caused much exhaustion the judicious use of stimulants is often beneficial. When the opportunity offers, it might be well to try hypodermic injections of the sulpho-carbolate of quinine. Yellow fever runs its course in five or six days; hence the vital powers must be sustained until the defervescence, and this is found to be extremely difficult on account of the extreme gastric irritability. A bland and highly nourishing diet is to be prescribed as soon as convalescence occurs, and tonics form an essential part of treatment at this period.[1]

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[1] *Yellow Fever; its origin, improper treatment, prevention, and cure.* Dr. W. A. Shubert, Savannah, 1860.

A dissertation on the sources of malignant, billous, or yellow fever, and means of preventing it. Dr. W. G. Chalmell, Philadelphia, 1799.

[2] *Das Gelbe Fieber beurtheilt und behandelt nach einer neuen Aussicht vom Wesen der Fieber in Allgemeinen.* G. Eichborn, Berlin, 1833. *The history of yellow fever, with the most successful method of treatment.* Dr. J. Mackrill, Baltimore, 1796.

EPIDEMIC CHOLERA.

Epidemic cholera is an acute general disease, which prevails epidemically, and in certain localities is endemic. It is characterized by copious watery discharges from the alimentary canal, by cramps, and by suppression of the excretions. It has also received the names of *cholera Asiatica, cholera asphyxia, and epidemic, malignant, algid,* or *blue cholera.*

Morbid Anatomy.—The post-mortem appearances vary with the period at which death takes place; in the stage of collapse or in that of reaction, there is usually marked emaciation; the extremities are noticeably shrivelled, and the surface of the body in the dependent portions is bluish or mottled; sub-conjunctival ecchymoses are often observed. The face has a pinched and drawn expression, and the eyes are deeply sunken. The body cools slowly after death, and frequently there is a *post-mortem* rise in temperature of two or three degrees Fahr.

Rigor mortis is marked immediately after death, and muscular contractions often cause changes in the position of the limbs and body. The skin is often so shrivelled as to resemble the condition called "parboiled," which is best marked upon the extremities. Putrefaction commences much later than in other diseases, on account of the withdrawal of large quantities of fluid from the body.

The visceral lesions are as follows. The small intestine is distended and of a bright red color; its muscular coat is somewhat relaxed. Its mucous membrane is injected with a fine aborescent vascularity; it is sometimes œdematous and its folds are often prominent, especially around the lower part of the ileum. Peyer's patches and the solitary follicles are at first enlarged, the latter more than the former; if the solitary glands rupture, the membrane presents a reticulated appearance. The comma bacilli are found in the intestinal contents, in the mucous membrane and glands throughout the canal. Ulcerations resembling typhoid ulceration may occur, the glands become flattened and pigmented. There is an almost complete detachment of the epithelium; if any patch is left undenuded there is a subepithelial exudation which loosens its attachment to the villi. The intestine may be partially or completely filled with a "rice-water," whey-like fluid, alkaline in reaction, which contains an abundance of cast-off epithelium, and the cholera bacilli, and varies in consistency from the ordinary cholera stool to that of putty. The mucous surface may be of a bright red, grayish, or, rarer than all, a greenish color. In some instances the intestine contains a moderate quantity of dark grumous blood. During the fever of reaction gray diphtheritic patches, very difficult of removal, which later become dry, brown sloughs, are sometimes found in both the small and large intestine. Similar patches have also occasionally been observed upon the mucous membrane of the biliary passages, vulva, and vagina. In severe cases the basement membrane is wholly denuded.

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[1] *Yellow Fever in Charleston*, 1871, *with Remarks upon its Treatment.* Dr. F. P. Porcher, Charleston, 1872. (Trans. S. C. Med. Asso.)

The *peritoneum* of the small intestine is of a rosy color and dry, or is covered with a thin layer of plastic matter.

The intestinal glands are congested, swollen, and prominent; while the mucous surface has large ecchymoses and patches of extravasation upon its substance. Diphtheritic ulcerations may be present in the colon.

The œsophagus is sometimes congested and ecchymosed, and its glands are swollen. It may have its epithelium detached, and at times it is covered with a diphtheritic exudation.

The stomach is at first distended and filled with fluids similar to those which are found in the small intestine; later its mucous lining is hyperæmic, swollen, often relaxed and ecchymotic. Still later it is collapsed and empty.

The kidneys are intensely congested and enlarged, the capsule is adherent, the surface presents a stellate or "marbled" vascularity, and on longitudinal section both cortical and medullary portions exhibit punctate or striped blood injections, and numerous ecchymoses. The small veins, especially around the glomeruli, are engorged, and the cortical portion of the kidney is more or less discolored. The uriniferous tubules have their epithelium loosened, and the cells are cloudy, swollen and filled with a granular albuminoid material; often transparent cylinders fill the lumen of the uriniferous tubes. For the most part the lesions resemble those of acute croupous nephritis. All these changes may occur during the first day of the choleraic attack. Later, during the secondary fever, the discoloration and tubular changes are increased; the size of the kidney being one-sixth to one-third greater than normal, and the epithelial cells undergo progressive fatty degeneration, and the whole organ becomes soft and friable. Chemical examinations have shown the kidneys to contain an abnormal quantity of urea, uric acid, leucin, and some bile-pigment.

The bladder is at first contracted and empty; but later it may be partially filled with albuminous, milky urine. Its mucous membrane and that of the ureters and pelvis of the kidneys undergo changes similar to the other mucous surfaces;—viz.: hyperæmia, ecchymoses, and perhaps diphtheritic processes.

The lungs are engorged at the entrance of the pulmonary artery; but the parenchyma of the lung is collapsed and exsanguinated, and crepitates less than normal lung-tissue. If death occurs during or after the reactionary fever, extensive œdema, hypostatic congestion and hemorrhagic infarctions may be found. Capillary bronchitis, lobular and lobar pneumonia, and emphysema are present in those cases where death occurs during convalescence. Pulmonary gangrene is a rare lesion. The trachea and bronchi are engorged and covered with a muco-pus, while later a secondary diphtheritic process may be established upon their mucous surface.

The pericardium is dry, and its visceral layer is ecchymotic, while the parietal is coated with a sticky, pasty material.

The heart is hard, dry and contracted, containing in its right cavity, which may be distended, soft clots, which sometimes extend into the pulmonary artery and into the veins. The left cavity is empty, or has only a few small black, loose coagula in it.

The blood is darker and thicker than normal, there is an increase in its albumen and corpuscles, as well as in its specific gravity and in organic solids; while there is a decrease in its saline elements and in its coagulating power. Urea is occasionally present.

The spleen is small, wrinkled, flabby and shrunken, though when typhoid symptoms co-exist, or when it is the seat of blood extravasations it is enlarged and softened.

The liver is usually pale, containing patches of commencing fatty degeneration, and the large veins are distended with blood. There is exfoliation of the epithelium of the mucous surface of the gall-bladder, which causes plugging and distention of the ducts.

The meningeal vessels of the brain and the sinuses are engorged, while the cerebro-spinal fluid is frequently absent. Medullary hyperæmia is common. But when death has occurred late, the brain contains less blood and is often superficially œdematous.

The sub-cutaneous connective-tissue is hard and dry. Parotid swellings, furuncles, purpuric and scorbutic spots, ulcerations of the cornea, and bed-sores are often present.

Etiology.—Cholera is an acute, infectious, non-contagious disease. The comma bacillus, which is found in the intestinal canal of cholera patients, is almost universally recognized as the specific cause of the disease. It prevails epidemically and may be endemic. It first appeared in the East, and thence spread in all directions, following the routes of commerce without regard to climate. No country has been entirely exempt from its ravages. It has prevailed, however, chiefly in hot climates during wet seasons. In this country it prevails most in midsummer. It is more liable to occur in low lands than in mountain regions. Badly drained malarial districts favor its development, especially where a cup-shaped rock or clay substratum is covered by a thin layer of permeable earth, favoring the decomposition of vegetable matter. Bad food, overcrowding, mental depression, excesses in venery and alcohol drinking, predispose to cholera. Epidemics of cholera occur most when the atmosphere is moist and sultry, or when a sultry period follows a warm rainstorm. Districts where these conditions prevail are regarded as favoring the development of the cholera bacillus.

As soon as the cholera discharges undergo decomposition, the bacillus is rapidly developed and may be conveyed from one locality to another by the wind, by waters, and in clothing. The specific poison of cholera is contained in the discharges from the mucous surface of the alimentary canal; it is not infectious when fresh, but it acquires virulent infectious properties in from two to four days, and is rendered innocuous by cold. There is no evidence that the bodies of cholera patients are infectious. The establishment of these facts readily accounts for its sudden appearance in different places remote from one another. An individual travelling rapidly from one place to another becomes the carrier of the germ, which is to develop the infection in those localities in which the conditions favor its reproduction.

Symptoms.—The length of the stage of incubation of cholera is not determined, but it undoubtedly varies from a few hours to as many days. Its symptoms may be divided into four stages. These divisions are arbitrary; *first*, the stage of invasion, or premonitory stage; *second*, the stage of painless diarrhœa; *third*, the algid or collapse stage; *fourth*, the stage of reaction.

The prodromal symptoms are a feeling of weight in the precordium, rumbling of the bowels, general malaise, a peculiar pallid anxious countenance, and nervous phenomena, such as vertigo, tinnitus aurium, headache, and tremor. Sometimes there is apathy, again a condition of exhilaration. Not infrequently, for a couple of days, there are frequent and moderately fluid dejections, sometimes accompanied by exhaustion, rarely by griping. This is called the *cholera diarrhœa.* These premonitory symptoms continue from a few hours to a week; usually, however, about two days. They may be, and frequently are, absent, the disease commencing precipitately with a painless diarrhœa. Occasionally the prodromata assume the character of cholera morbus, but cramps are more prominent, and there is little or no fæcal odor to the discharge.

The *second stage* is characterized by a profuse diarrhœa, generally commencing in the morning or in the middle of the night, and the patient describes the dejection as passing from him in a stream. These painless discharges sometimes, after the second evacuation, lose their fæcal odor and color, and assume a light straw-colored or whey-like appearance. They vary in number from three to twenty a day, and are often accompanied by attacks of regurgitative vomiting with each evacuation. The average amount of fluid discharged in this stage by a cholera patient in twenty-four hours is about sixty ounces; the patient becomes exhausted and assumes a peculiar apathetic condition; dizziness, headache, and vertigo sometimes are present. Complete anorexia is present from the onset, and the thirst is tormenting and constant. Bile pigment disappears from the stools, and the rice-water appearance is assumed; there may be a pinkish tint on account of the admixture of blood. The rice-water discharges often have a whey-like appearance consisting of the watery elements of the blood; their specific gravity varies from 1.005 to 1.012, and they contain a small proportion of albumen and an excess of sodium chloride. On standing, the rice-water fluid deposits a sediment holding fine granular cells, amorphous granular matter, shreds of tissue, minute nucleated cells, epithelium and blood globules. Occasionally the blood globules are so numerous that the vomited matters are red. Vibriones, bacteria, urea, triple phosphates and a few leucocytes are also not infrequent ingredients. The vomited matter, after the contents of the stomach and bilious matters have been ejected, is a clear, watery fluid containing urea and carbonate of ammonia; it is ejected in a stream, without nausea or effort, and is characteristic of cholera. Everything introduced into the stomach causes vomiting.

The tongue is dry and covered with a thick white coating; the countenance becomes pinched and of a leaden hue, the expression is staring and dull; as the exhaustion verges on collapse, the pulse becomes imperceptible at the wrist. Often there is distressing hiccough, and more or less dyspnœa. In rare instances the abdomen is tense, hard and sensitive to pressure; it may be retracted. Suppression of the urine is not of infrequent occurrence at this stage.

The algid stage commences with a well-marked fall of temperature; first in the hands, feet, and face, but soon over the entire body. The axillary temperature may fall as low as 72° F., or even lower, while the rectal temperature registers 101° or 102° F. The accompanying sweat makes the surface feel colder than it really is; the patient himself rarely complains of being cold. The skin is in distinct, hard folds ("washerwoman's skin") and of a bluish or livid color. The features and extremities are pinched, the eyes are deeply sunken, and have purplish rings about them. The patient is in a state of apathy or stupor; and is roused therefrom only by the severe cramps, which cause him to shriek and throw himself about the bed. These cramps chiefly affect the muscles of the calf of the leg.

In the last portion of this stage (called the *asphyxial*) the condition of the patient is apparently hopeless; the deadly coldness is so marked in the tongue and mouth that the thermometer may show a temperature of only 79° F. The lividity and cyanosis, the imperceptible heart sounds, the absence of the radial pulse, the "cholera face," and the hoarse sepulchral "cholera whisper," the agonizing cramps that now recur oftener than at first, complete the desperate picture of the disease. The vomiting and diarrhœa now markedly diminish and the discharges are less fluid when they do occur. The stools are passed involuntarily or heedlessly.

The urine is either completely suppressed, or a few highly albuminous drops are passed. The respirations are shallow and hurried, often being 40 per minute, and alternate very often with paroxysms of intense dyspnœa. There is a loss in weight during this period, and so drained is the blood that there is an absorption of pathological fluid accumulations as in pleurisy and synovitis. The saliva and all secretions are suppressed. Late in this stage of cholera the stools, from being odorless, change, and assume a smell something like decayed fish. The state of collapse may last forty-eight hours, and yet recovery take place; or death may occur in two or three hours from the onset of this algid condition.

The mind is clear throughout, and consciousness is retained till the last; it is even recorded that insane patients have, in "cholera collapse," regained (temporarily) their sanity.

The "*reactive stage*," when reached, is often marked by as speedy a return of favorable signs as was the algid stage by unfavorable ones. The pulse appears in the carotids and at the wrists, and the heart-sounds become distinct and regular. The temperature rises, the skin becomes warm, the face loses its "deathly" look, the cramps cease, and the diarrhœa continues; the stools soon acquire a fæcal odor and a brown color; although in cases where the algid stage is prolonged, foul-smelling, greenish, fluid discharges continue for some time. The urine next appears, although its return may be delayed from ten to thirty hours; at first it is scanty, high-colored and albuminous, containing casts, and turning pinkish with nitric acid. Soon it becomes copious and normal in character. The duration of this period varies from one to ten days. This is a history of a typical case of cholera. I shall now briefly consider some of the more common variations.

Cholera typhoid is perhaps the commonest sequela of the collapse stage. After a few days, in some cases a week, of well-marked reactive symptoms, when the secretions are fully established and excretion is being normally performed, a quickening of the pulse is noticed, usually toward evening, and soon a febrile movement is established, which recurs with regular paroxysms. These are accompanied by adynamic symptoms, such as low, muttering delirium, a dry tongue, injected conjunctivæ, coma, and often bed-sores and purpuric spots. The patient sinks into a state of extreme exhaustion, and gradually the coma deepens, the bowels and bladder are involuntarily evacuated and death occurs. If patients recover from cholera typhoid, the convalescence is very protracted and uncertain.

Uræmia is a frequent condition; following the stage of collapse no urine is secreted in the reactive stage; and in about thirty-six or forty-eight hours the pulse becomes abnormally slow, the face slightly flushed, and the eyes darkly injected. The urine is entirely suppressed or very scanty, and will be found to contain albumen and casts in abundance. There is constant headache, rarely a mild delirium. The patient becomes drowsy and listless, vomiting a spinach-green material. Epileptiform convulsions are followed by coma and death. The bowels are constipated, and the febrile symptoms are negative.

A "*cholera eruption,*" so-called, sometimes makes its appearance either in the typhoid variety, or in the stage of reaction. This eruption varies in character: it may be an erythema, or resemble urticaria or roseola. It appears first on the hands and feet, then spreads to the trunk, the face being very slightly affected. Macular, papular and vesicular eruptions sometimes occur; in all cases the appearance of a cholera eruption is a favorable symptom. The eruption lasts about two days, and is often accompanied by a "burning" sensation. Although in *children* the disease runs the same general course, collapse supervenes much more rapidly, and death often occurs after a few choleraic discharges.

Cholerine is a mild form of cholera occurring during a cholera epidemic, and attended by all the characteristic symptoms of the disease, except that there *is no algid stage*. There is often a slight coolness of the extremities and cramps in the calves of the legs. Recovery is usually rapid. It may be followed by a severe and well-marked attack of cholera.

Differential Diagnosis.—During an epidemic, cholera is not likely to be mistaken for any other disease; but when it occurs in isolated cases, it may be confounded with acute *poisoning*, as from *arsenic* or *antimony*, and with the *gastro-enteric* variety of pernicious fever.

In cases of *poisoning* there will be the evidences of the action of the poison on the mouth and pharynx which are absent in cholera. The vomiting in cholera is regurgitative and painless, whereas in cases of poisoning it is distressing, and is preceded by an intense burning pain in the œsophagus and stomach. Diarrhœa, if it occurs in poisoning, is never of a "*rice-water*" character, but mucous and blood-stained. A chemical analysis of the ejected matters will detect the presence of a poison.

In the *gastro-enteric* variety of pernicious fever the first two or three discharges from the bowels are bloody; while in cholera they are never bloody at first, and soon assume the "rice-water" appearance. In gastro-enteric pernicious fever vomiting is rare, but if present, is painful and retching in character; while in cholera it is regurgitative. The temperature in pernicious fever is high, often reaching 106° or 107° F., while febrile movement in cholera is slight. There is free pigment in the blood in pernicious gastro-enteric fever, which is never found in the blood of a cholera patient.

Prognosis.—The mortality-rate varies in different epidemics from 20 to 80 per cent.; generally one-half recover. The more dense the population in any locality and the nearer the sea-coast the higher the mortality-rate. The mortality-rate is always less toward the end than at the commencement of an epidemic; it is greatest in those under *one* or over *fifty* years of age. Habits of life and hygienic surroundings influence very greatly the prognosis. The duration of an attack varies from a few hours to two weeks. Fatal cases usually terminate within two or three days, while the average duration of those that recover is nine days. Each epidemic in this country has been milder than the preceding.

The symptoms which indicate recovery are a general improvement in the appearance of the patient; he becomes less restless, his breathing slower and more natural, the radial pulse returns, the lividity of the surface disappears, the shrunken tissues expand, the temperature rises to normal, the urinary secretions are re-established, the discharges from the bowels are again stained with bile, and the patient falls into a quiet sleep. The unfavorable symptoms are involuntary pinkish discharges from the bowels, absence of the radial pulse and the second sound of the heart, extreme cyanosis, a complete suppression of urine, coma, persistency of the vomiting and diarrhœa, and the occurrence of complications.

Cholera may be complicated by capillary bronchitis, lobular pneumonia, œdema and congestion of the lungs, pericarditis, peritonitis, and pleurisy. The sequelæ are uræmia, membranous enteritis, cerebral œdema and hyperæmia, gangrenous or purpuric patches, ulcerated corneæ, furuncles, bed-sores, and gangrene of the lungs. Death may result from the direct effects of the cholera poison without the occurrence of the diarrhœa, from the exhaustion produced by the diarrhœa, from heart-failure, and from any of its complications or sequelæ.

Treatment.—Prophylactic and hygienic measures may limit the duration, extent, and the mortality-rate of a cholera epidemic. When a cholera epidemic is prevailing quarantine regulations must be vigorously enforced, and those attacked by the disease should be isolated. All cess-pools, privies, and bodies of stagnant water in the neighborhood should be drained or disinfected, and each member of the community should be placed under the best hygienic conditions, and his diet carefully regulated. All excesses in food and drink, and all sources of intestinal irritation should be avoided. A diarrhœa occurring during a cholera epidemic should be immediately checked.

Cholera stools should be immediately disinfected and buried in trenches, as in typhoid fever. The linen and all utensils used in the sick room must also be thoroughly disinfected. Instead of sulphate of iron and hydrochloric acid mingled with the fæces, carbolic acid may be used; indeed, many regard it as superior to any other disinfectant for the purpose. All persons, who are able, should be immediately removed from the infected district.

The first great object of *medicinal treatment* is to control the *prodromal diarrhœa.* For the accomplishment of this, *opium* is the most reliable drug; it may be combined with nitrate of silver, sulphuric acid, small doses of calomel, or with vegetable astringents. Brown-Séquard states that morphine hypodermically in sufficient doses at the onset *will prevent cholera.* The patient is to be at once placed in bed, kept absolutely quiet, and the abdomen swathed in flannel bandages. If there are slight signs of exhaustion early, stimulants may be given carefully. Turpentine stupes over the stomach and bowels in the early stage, when the symptoms are urgent, are often serviceable. Nausea in the premonitory stage is often allayed by carbonic-acid water, cracked ice, or effervescing draughts.

When the disease is fully established, as indicated by the projectile vomit and rice-water stools, the treatment becomes "symptomatic" or "expectant." To relieve the agonizing thirst patients may take freely of cracked ice, very *cold* seltzer water, or carbonic-acid water combined with lime water. If the pulse becomes imperceptible at the wrist, indicating heart insufficiency, stimulants are indicated, but they must be carefully administered. English physicians in India give opium, calomel, and acetate of lead (or tannin) during the stage of painless diarrhœa. If the cramps are not severe, they may be relieved by friction. But when they become severe, hypodermics of morphia combined with chloral are indicated. If the extremities become cold they should be wrapped in hot cloths, or hot water bags may be placed around them, or they may be rubbed with stimulating liniments or capsicum preparations. In the stage of collapse iced brandy or champagne given repeatedly and in small doses is the best stimulant; musk and ammonia are also recommended. The inhalation of amyl nitrite has been tried, and found very efficient in combination with alcohol, in the advanced stage of collapse. When death is impending, whiskey may be injected hypodermically, or milk may be administered intravenously. In the use of stimulants one must be guided by the pulse, and the effects of the stimulation. The India cholera pills, given in the collapse, are made of camphor, asafœtida, pepper, and the essential oils or ether.

As the reactionary fever comes on, and the temperature begins to rise, nourishment must be given with the greatest care; the rule being to postpone a solid diet as long as consistent with maintenance of strength; milk, beef-juice, and very light broths are the only articles of diet admissible for some time. When the stomach is weak and irritable, and there is a tendency to vomiting, bismuth and cherry-laurel water can be given with advantage. Cerebral symptoms must be promptly treated by ice-bags about the head, heat to the feet, and bromide of potassium internally. The surroundings of the patient, the maintenance of cheerfulness and *calm*, and even temperature—these are important points to be observed.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

Epidemic cerebro-spinal meningitis, or cerebro-spinal fever, historically belongs exclusively to the nineteenth century, although it unquestionably prevailed prior to this period. French writers were the first to accurately describe it. It is a continued fever belonging to the class of *miasmatic-contagious* diseases, which generally prevails in quite limited areas. It has received various names; as *spotted*, *petechial* and *congestive fever; malignant purpuric fever; cerebro-spinal* and *syncopal typhus*.

Morbid Anatomy.—Pathologically as well as etiologically there are two forms of cerebro-spinal meningitis; the *first* is simply an acute inflammation involving the meninges of the brain, spinal cord and medulla.

Its local symptoms predominate over the constitutional, and it occurs exclusively as a *sporadic* disease; the *second,—epidemic cerebro-spinal meningitis,*—is accompanied by all the signs of an infectious disease, and, at the autopsy, are found, in addition to lesions that are the counterpart of those occurring in simple cerebro-spinal meningitis, those grave visceral and sanguineous changes, which are present in other acute infectious diseases.

On examining *the brain* of one who has died of epidemic cerebro-spinal meningitis, the convexity and base will be found most extensively involved. Its dura mater is tense, shining, and studded with numerous punctate spots of extravasation. The cerebral convolutions are flattened and the sulci deepened. The pia mater of the brain and spinal cord is thickened. The *vessels* of the pia mater are always more or less intensely injected and the surface of the membrane roughened. In some cases extreme hyperæmia may be the only discoverable lesion. The *exudation* is the characteristic lesion of this disease. A more or less abundant sero-fibrinous or sero-purulent exudation takes place into the meshes of the pia mater. Clear serum is first effused, then it becomes milky and clouded, then yellowish, and finally a thick, viscid, greenish-yellow mass, consisting of granular fibrin, pus-cells and red blood globules, which gives to the surface a "leek-green" color. In the severest cases the fibrin and pus form a continuous sheet in the sub-arachnoidean space, always thickened above the sulci. The vessels are inclosed in the exudation, looking like red threads in a gelatinous filmy mass. When the layer is removed, the subjacent gray substance is dotted with red points. In rare cases the exudation is deeply stained with blood; at other times it is a thin, colorless fluid. The sinuses are full of dark, soft coagula, or thin fluid blood. Hard thrombi are occasionally found in them.[1]

The *brain substance* is frequently softened, especially near the largest patches of exudations. This is the "mechanical softening" of French authors.

On section there is more or less congestion and punctate extravasation in the brain substance, and the ventricles are usually full of serum; more rarely of pus; and this pus enters the ventricles by means of the velum interpositum, or along the cerebellar or choroid plexuses.[2] All the local changes have their primary starting point in the pia mater. Finally, if absorption occur, the pia mater often remains thickened. In prolonged cases cheesy metamorphosis occurs in various spots in the thickened pia mater.

The changes in the *spinal canal* are similar to those within the cranium. The *dura mater* is injected, and extravasations of blood are often found upon its parietal surface; it is tense and shining. The meshes of the spinal *pia mater* are occupied by the exudation, which occurs either as stringy, interlacing bands forming a network, or in the form of a thick sheet completely enveloping the cord's substance. The color and character of the exudation are the same as that in the cranial cavity. The largest collections of pus are about the second and last dorsal and the lumbar vertebræ. The posterior portion of the cord is the part most involved; the anterior portion suffering only in those cases where the whole cerebro-spinal tract is involved. The *pia mater* itself is hyperæmic; it may be thicker than normal, shaggy and adherent to the cord. In some instances the exudation occurs in the form of lozenge-shaped, irregular masses whose ends are connected to one another by bands of fibrino-pus. In the severest cases suppuration is so rapid that a complete sheath of pus is formed about the whole cord in a few hours after the onset of the malady. The gray substance of the cord is of a pinkish color, and may be infiltrated with serum. It is sometimes reduced to a mere pultaceous mass.

In addition to these local changes there are blood and visceral changes. The fibrin-factors of the *blood* are diminished, and hence there is a loss in its coagulating power. The number of white corpuscles is increased, and the red ones are shrivelled, serrated and partly disorganized. The blood is darker than normal, fluid, and rapidly decomposes when taken from the body.[3]

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[1] Merkel states that he has found "a nuclear proliferation in the vessels, extending from the cerebral meninges to the spinal cord."

[2] Virchow's Arch. Bd. 34, Heft 31, 866.

[3] The ventricular fluid contains chloride of sodium, phosphate of soda and ammonia and oxalate of urea. —*Meschede.*

The *heart* and the *voluntary muscles* undergo the same degeneration and present the same appearances as in typhoid fever.

The *lungs* are frequently the seat of œdema, and hypostatic congestion is present when the disease is prolonged. Passive hyperæmia, lobular, and, less frequently, lobar pneumonia, are often found at the *post-mortem*.

The *liver* is congested. The liver-cells are often cloudy and granular—*i.e.*, there is albuminoid or fatty degeneration.

The *spleen* is enlarged and softened; the lymphatics are usually hyperæmic, having a fleshy look.

The *intestinal mucous membrane* is hyperæmic and the follicles are congested. The projecting agminated glands are sometimes ulcerated. There is more or less congestion of the *kidneys;* the microscopical changes are those of the first stage of acute Bright's. Abscesses (as in typhoid) often form in the *subcutaneous connective tissue.* Bed-sores are not rare in those parts subjected to pressure, and gangrene is sometimes present.

The integument is often the seat of petechial spots, and large, irregular, discolored patches are sometimes seen over the body. Herpetic spots are frequently seen on the surface of the body, on the face, and about the lips especially. *Rigor mortis* is marked and very much prolonged. Finally the *serous membranes* are frequently *all* covered with petechial spots and small extravasations.

Etiology.—I have included cerebro-spinal meningitis in the list of miasmatic contagious diseases, as it has more in common with this class than with any other. It has prevailed as an epidemic and as an endemic disease, and occasionally sporadic cases have occurred in localities where it has been epidemic. Epidemics have occurred at all seasons; by far the greater number, however, occur in cold weather. All classes and ages are subject to it, but it is most likely to attack those between ten and eighteen years of age. Young troops on the march are especially liable to it.[1] Its strongest predisposing causes are over-crowding, bad ventilation, insufficient or improper food, dampness, and all other bad hygienic surroundings. Mental excitement, excessive brain-work or bodily fatigue, exposure to excessive cold or heat, are also predisposing causes. Cerebro-spinal meningitis is in no sense a contagious disease. It is more closely allied etiologically to lobar pneumonia than to any other disease, although some have regarded it as a variety of typhus fever; others of malarial fever. Its specific microbe has not been discovered.

Symptoms.—Cerebro-spinal fever follows no regular order, either in its prodromata or in its subjective symptoms. Arbitrary classifications and many subdivisions have been made, such as the typhoid, the paralytic, the adynamic, the intermittent, the petechial, etc. Such classifications are useless and confusing, for cases differ in the same epidemic. If the general phenomena of the disease be known, the accidental circumstances that are the basis of this complex nomenclature will be of very little importance.

The *premonitory* symptoms of cerebro-spinal meningitis vary in different epidemics. In some the invasion is abrupt; the patient, apparently in perfect health, is suddenly seized with a chill, loss of consciousness, becomes comatose and dies in a few hours. In others a feeling of lassitude, dull headache, pains in the joints and muscles, and sometimes nausea and vomiting precede its development. Again, patients complain of pains in the back of the head and neck—they have no chills, but after twenty-four hours a febrile movement is developed and they pass rapidly into the *active* symptoms of the disease. The prodromata may last from a few hours to three or four days. In sporadic or endemic cases there is generally a period preceding its invasion during which patients suffer from a feeling of general indisposition.

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[1] Out of forty-seven French epidemics, Hirsch attributes forty-six to the military population.

When its onset is sudden its advent is marked by a distinct chill, intense headache, pain in the back and upper part of the spine, nausea, vomiting, a rise in temperature and an acceleration of pulse. The *chill may* last an hour or more, but is usually of short duration. The skin is abnormally cool and dry in its early stage.

Headache in most cases is a prominent, agonizing and persistent symptom, and the pain, even in a condition of coma, causes the patient to groan. In rare instances the headache intermits, and frequently it remits. Vertigo almost always is an attendant, and the patient may suddenly stagger and fall during the period of the headache.

Pain in the *back* and upper part of the spine is a characteristic symptom of the disease; attempts to flex the head on the chest increase the pain during the first twenty-four hours of the disease, and pressure up under the *ligamentum nuchæ,* against the cord, often induces excruciating agony. Soon the muscles at the back of the neck become stiff, then rigid, the neck becoming fixed, and the head extremely extended—opisthotonos. So intense may be the opisthotonos that attempts to swallow are so painful that the sufferer soon ceases to make the effort. The signs of prostration are present early.

The *temperature,* as a rule, is *low;* although 107°, 109° and 110° F., are recorded by trustworthy observers. It may rise rapidly to 104° or 105° F., and then suddenly fall to 102° or 103° F., there to remain, with unimportant and irregular variations until a gradual return to normal marks the beginning of convalescence. Often, before death—and an almost sure indication of it—a low temperature will suddenly give place to a high one, and death will occur during the time of the highest temperature. In children the febrile movement is less marked than in adults.

The *pulse* at first is slightly accelerated, beating from 90 to 100 per minute; but in twenty-four hours after the commencement of the attack it may range between 120 and 150. It bears no relation to the range of temperature, often varying 40 or 50 beats in a few hours. It is feeble, rapid and compressible in those cases where there are early symptoms of exhaustion. In many cases it is small and wiry in character; sometimes it is dicrotic. In children the pulse is more accelerated and much more excitable than in adults. In a few cases the pulse is slow at the onset of the attack, but soon becomes accelerated, irregular and intermittent. Photophobia, contracted pupils, great and increasing restlessness, nausea and urgent vomiting, and abdominal neuralgia, are among the early symptoms.

The *pupils* are often unequal in size, and usually respond slowly to light. The *face* is pale and anxious, and the features have a fixed, rigid expression; in some the countenance has a dusky hue like that of one who is under the influence of narcotic poison; indeed, in some instances, the patient believes, from the severity and suddenness of the attack, that he is the victim of wilful poisoning. About the second or third day of the disease, if the headache has been very severe, *delirium* comes on; it may be mild and muttering, wild and uncontrollable, or "maudlin," like that of a drunken man. In women the delirium may be attended by, or merge into, a form of hysteria. The most fanciful hallucinations often visit the minds of such patients, and if left to themselves they are constantly getting out of bed. These patients are frequently roused from their wanderings by excruciating pains in the head and extremities. Muscular contraction is rarely absent even in the mildest cases.

By the third or fourth day a tetanic and contracted state of the muscles of the extremities begins, and then the arms become flexed on the chest, the forearm on the arm, the thumb on the palm, the knee on the abdomen, and the leg on the thigh. When these excito-motor spasms of a tonic character are marked in the groups of muscles in the back of the neck and in the back, trismus may occur, and then the case is hopeless. Twitching of groups of muscles often causes the patient to start from a state of semi-stupor. General convulsions are absent in adults, but are frequent in children. Pains in the extremities and in the abdominal region are always more or less severe. They are shooting and lancinating in character. Pains when located over the abdominal region cause *vomiting*, and *dyspnœa* when in the thoracic region. The skin may be hyper- or anæsthetic, and is early the seat of an eruption. In the majority of cases the surface is so sensitive that palpation and percussion are exceedingly painful; the patient cries out and starts at every attempt, and will usually say that it is that particular spot which is the "sorest." Voluntary movements cause pain. Cutaneous anæsthesia rarely exists throughout the course of the disease, but follows the hyper-sensitiveness.

The *eruption* is usually limited to the face, neck, and lips; it is herpetic in character. It may appear on the trunk and limbs. Vesicles appear earliest on and about the lips, and may be confined to them. Sometimes the eruption is mottled like that of typhus, and covers the body; it may have a distinctly petechial character. Ecchymotic spots are often scattered irregularly over the body, especially on those parts that are subjected to pressure. Purpuric maculæ, erythema and urticaria are sometimes present (indeed, there are many varieties of the eruption), but herpetic and petechial spots are the most common. As there is no definite time for the appearance of these eruptions, so their duration varies; sometimes they last only for a day—at other times they are visible throughout the whole course of the disease. Epidemics in which eruptions are marked have given rise to the name of *spotted fever*.

With the *photophobia* the *eye* is subject to many disturbances. Paralysis of the orbicularis palpebrarum may result in keratitis; there may be more or less intense conjunctivitis; or a neuro-retinitis or choroiditis, the result of an implication of the optic nerve, may occur. Ptosis is present in nearly every case. Temporary or permanent blindness, squint, double vision, and nystagmus are not infrequent optical lesions. Atrophy of the eyeball and cataract are occasional sequelæ.

Taste is perverted or entirely lost; yet the patient will often take with avidity any article of food which may be placed in his mouth. Thirst is often a constant and tormenting symptom. *Deafness* is even more frequent than loss of, or disturbances in sight. There is always intolerance to noise, and tinnitus aurium exists from the very commencement. Otorrhœa may be extensive enough to result in tympanic perforation; and the internal ear may become the seat of an inflammatory process which sometimes ends in suppuration. The semicircular canals would seem to be involved here, for in many recorded cases "an uncertain gait" is mentioned as accompanying the deafness. The *respiratory tract,* as a rule, is involved, the respirations generally being accelerated out of proportion to the frequency of the pulse, but when the exudation presses on the medulla and respiratory centre, dyspnœa and slowed respiration occur, and in some few cases the Cheyne-Stokes' breathing is noticed. Usually the violent headache and the "wandering" are attended by great restlessness, tossing and jactitation that frequently demand restraint. Insomnia is a common symptom. There is often great tremulousness and subsultus tendinum; in the advanced stage of the disease the pupils are dilated, the respiration markedly sighing, deglutition difficult, the sphincters relaxed, or there is retention of urine and fæces, the removal of which, by means of the catheter or copious enemata, causes a slight return of consciousness.

The *tongue,* at first, is moist and covered with a whitish coating; soon it becomes dry and brown; the parotids may enlarge, and even suppurate; the abdomen is flattened. Rigidity, contraction and opisthotonus give way to palsies. The skin becomes cyanotic as in the asphyxia stage of cholera. In other cases tetanic spasms are the most prominent signs, the rigidity and contraction of the muscles of the back and neck are excessive, and the sufferer dies with the grin of lock-jaw upon his face. In protracted cases the patient becomes emaciated and loses strength in a degree out of proportion to the duration of the disease.

The *joints* are usually tender, and often inflamed; suppurative arthritis occurs in a few instances.

The *urine* is but slightly altered. There is an increase in the urates and phosphates, and albuminuria not infrequently occurs, especially late in the disease. Polyuria is often present in children.

The *bowels* are constipated; exceptions to this rule are seen only in children. If the disease is prolonged, the symptoms assume a typhoid character; and so "typhoid cerebro-spinal fever" is one of the many varieties. The term intermittent cerebro-spinal meningitis has been applied to those cases where all the symptoms remit on the second or third day from the onset of the attack, and soon reappear or exacerbate and the patient rapidly passes into stupor and coma. As an epidemic advances the cases grow milder, so that toward its end the patients may hardly be ill enough to be confined to their beds.

In the form called "*meningitis foudroyante*" the patient is struck down in full health, and death may occur within twenty-four hours from the first symptom. The initial chill and headache are severe, there is stasis in the capillary circulation of the surface, purpuric maculæ soon appear over the body, and active delirium is followed by profound coma and death. The course is often so rapid that there are no tetanic exhibitions. Exhaustion, paralysis, and anæsthesia are complete before the fatal issue; albuminuria is rarely absent in such cases. In fact all the prominent severe symptoms of the disease are crowded into a few hours, and the patient rapidly passes into a state of collapse.

When recovery is to occur, the restlessness, jactitation, insomnia and headache remit and finally disappear, or the patient emerges from a condition of coma into consciousness. The muscular paralysis continues, however, as well as the pains in the head and back of the neck, and in all cases the convalescence is tedious. Stiffness of the muscles of the nape of the neck is a persistent symptom during convalescence. Mental-psychical disturbances are also common attendants of the convalescence. Sometimes when the disease has pursued a mild course for a week or ten days and convalescence seems about to be established, the patient gradually gets worse, and after weeks of suffering, death will occur from inanition and general marasmus, the respirations becoming more and more irregular, and deglutition often becoming impossible.

Differential Diagnosis.—Cerebro-spinal fever may, in children, be confounded with *pneumonia,* since convulsions and opisthotonus may occur in either. It may be mistaken (at any age) for *typhus, small-pox, tubercular meningitis,* the *cerebral* form of *pernicious malarial* fever, and *acute myelitis.*

When, from the ushering-in symptoms, doubt arises as to whether a child has *thoracic* or cerebral disease, a careful physical examination of the chest will at once remove the doubt.

The differential diagnosis between cerebro-spinal fever and *typhus* and *pernicious* malarial fever will be considered in the history of those fevers.

In *small-pox* the pain in the head is confined to the frontal region, while in cerebro-spinal meningitis it has its seat in the occipital region. In meningitis there is early stiffness and rigidity of the muscles at the back of the neck. In small-pox this is a later symptom if it occurs at all. In small-pox, on the fourth day of the fever, the characteristic eruption appears about the roots of the hair, while in cerebro-spinal meningitis there is no peculiar eruption, and no regularity in the date of its appearance. The temperature in small-pox is much higher than in cerebro-spinal meningitis. On the second day of small-pox there are redness, swelling, and soreness of the throat; in spinal fever these are absent. Coma may occur early in cerebro-spinal meningitis, but is a late symptom in small-pox. After the initial pains in the back and limbs, pain is not a prominent symptom of small-pox; while the severe and excruciating pains in the head, limbs, and trunk increase in severity with the advance of cerebro-spinal meningitis.

The diagnosis between *tubercular* and cerebro-spinal meningitis is always difficult and often impossible. A careful study of the previous history, the insidious and characteristic slow advent of tubercular meningitis, the *slowed* pulse at the beginning, the "hydrocephalic cry," the absence of eruptions, the very mild delirium, and the late appearance of muscular rigidity, are the points on which we may differentiate the otherwise analogous diseases.

The ushering-in symptoms of *acute myelitis* are very similar to those of cerebro-spinal meningitis; but when the myelitis is fully established there are the peculiar "girdling" pains—the feeling as if an iron band were around the waist—with paralysis of the lower limbs, which rapidly extends upward. The temperature of the paralyzed limbs is *first* elevated, but subsequently falls *below* the normal; there is almost complete anæsthesia of the surface, and impaired muscular contractibility; later there is atrophy of the muscles of the paralyzed parts;—all of these symptoms are in strong contrast with the symptoms of cerebro-spinal fever. Again, pressure on the spine in myelitis causes severe pain which is not increased by motion; while in meningitis motion rather than pressure causes pain. The *rectal* and *vesical* sphincters are involved in myelitis, so that ammonæmia, pyelitis, cystitis, and various urinary complications are early attendants on the disease; these are rarely present in cerebro-spinal fever. Trophic nerve derangement is shown, in myelitis, by the extensive formation of acute bed-sores, while this is a comparatively rare, and always a late, occurrence in cerebro-spinal meningitis. Reflex power is diminished or wholly absent in myelitis, while it is exaggerated in spinal meningitis.

Prognosis.—Cerebro-spinal meningitis is always a grave form of disease, and a guarded prognosis should be given. The death rate in severe epidemics is 80 per cent., and about 30 in mild ones. Toward the close of all epidemics the death rate markedly diminishes. Hence, the period as well as the severity of an epidemic will influence the prognosis. Its average duration is about fourteen days, but cases are recorded where death has occurred in five, twelve, fifteen, twenty-four, and thirty hours after the first symptoms. In the majority of cases which prove fatal, patients die during the second week; if recovery takes place the disease is apt to last two or three weeks. In quite mild cases the disease lasts about two weeks; and in the intermittent form, when the so-called relapses occur, the disease may be protracted seven or eight weeks.

Age influences the prognosis. Statistics show that under fifteen, the mortality-rate is much greater than between fifteen and thirty-five; and that after thirty-five, each year diminishes the chances of recovery. Every day that is passed after the seventh renders recovery more and more probable; the symptoms that tend to render the prognosis unfavorable are a rapid, and especially an irregular or intermitting-pulse, an abundant eruption, excessive hyperæsthesia and nervous excitement, absolute insensibility of the pupils, as well as symptoms of great mental depression and prostration early in the disease. Convulsions, a low temperature with attendant collapse, paralysis of the muscles of deglutition, continued vomiting, shallow and irregular respirations and the occurrence of any of the complications, all render the prognosis unfavorable.

The *complications* of cerebro-spinal fever are bronchitis, broncho-pneumonia, croupous pneumonia, pulmonary œdema, pulmonary atelectasis arising from obstruction in one or more bronchi, and pleurisy. Endocarditis or pericarditis and nephritis are frequent complications ; the lesions of the eye, ear, joints, and subcutaneous areolar tissue can be regarded as belonging to, and complicating the ordinary course of the disease.

The sequelæ of cerebro-spinal fever are numerous : even in the most favorable cases basilar headaches and attacks of dizziness are liable to occur for years after recovery. Deafness or blindness may result, and in children deaf-mutism is a not uncommon sequence, especially if the disease occur before the child has learned to talk. The eye lesions, already mentioned, may become permanent. The psychical disturbances may vary from complete idiocy to stupidity, impaired memory, and marked diminution in intelligence. General motor weakness is rather unusual; but paralysis of various muscles or groups of muscles is a frequent sequel. Single nerves are sometimes paralyzed. Death may result from the pulmonary complications, from paralysis of the muscles of deglutition and of the thoracic groups, from heart failure, and consequent œdema of the lungs, from intensity of poisoning at the onset, from asthenia, and from coma.

Treatment.—The *prophylactic* measures to be observed during an epidemic of cerebro-spinal meningitis may be summed up in careful attention to the surroundings :—remove all anti-hygienic influences, and when possible isolate the sick. Indeed the general principles of prophylaxis are the same as in all miasmatic-contagious diseases.

A patient with cerebro-spinal meningitis should be immediately put to bed, in a dark, cool, well-ventilated room, removed from noise and confusion. During the entire course of the disease the diet should be of the most nutritious kind, easy of digestion ; milk is to be preferred. The exhaustion and emaciation that render convalescence so tedious must be combated from the onset by a nutritious and generous diet. The thirst which is so tormenting may be relieved by allowing the patient to drink as much ice or seltzer water as he desires. If constipation exist it must be overcome by promptly acting cathartics,—a calomel purge is to be preferred. It is well to administer a turpentine enema to aid the action of the calomel. A free catharsis must be early obtained. The condition of the bladder must be carefully attended to throughout the disease. If the patient does not evacuate it at the proper intervals, recourse must be had to the catheter. Sometimes cystitis has resulted from neglect of this.

As with all severe forms of disease, various *plans* of treatment have been adopted. The plan of general blood-letting and depletion has no doubt raised the death rate. In no case is blood-letting indicated or allowable in this disease any more than in typhoid fever or diphtheria. The internal and external use of calomel in its treatment, although it has been extensively employed, is not sustained by the result of experience. Nor has *iodide of potassium* the reputation which it once had for promoting the absorption of inflammatory products. *Quinine*, if useful at all, is only so at the very onset, and if used then it should be administered in large doses. It has no antipyretic power in this disease.

The medicinal agents which are generally accepted as most useful in the treatment of this disease are the narcotics: among these opium stands first in the list; administered hypodermically, it not only promptly relieves the pain in the head, the restlessness, jactitation, insomnia, delirium and convulsions, but it likewise increases the arterial tension. This drug should be given until the desired effect is produced, namely, complete relief; there must be no hesitation in the administration of large doses if required, for there is a remarkable tolerance of the drug in this disease. It may be combined with atropia. *Bromide of potassium* is regarded by some as especially indicated in this disease, and it has been given quite extensively with apparent benefit, especially in children.

When cerebral symptoms are violent, cannabis indica[1] may be cautiously administered. Chloral hydrate is contra-indicated, and ether and chloroform inhalations should not be resorted to unless neurotics have failed to relieve convulsions or pain. Ergot is recommended by many on the ground that by its action on the vasomotor system it produces cerebral and spinal anæmia. It is proposed to give the ergot until dizziness is produced. Experience, however, does not sustain the theory.

When symptoms of great exhaustion are present, stimulants are demanded; decreasing restlessness and a continued fall of temperature are some of the signs that indicate that stimulants are acting remedially. The rules and methods of stimulation are the same as in typhoid fever. When cerebro-spinal fever is long continued and there is reason to believe that there is an abundant serous effusion, iodide of potassium in large doses may be of service.

Cold applications to the head and spine, by means of evaporating lotions, sprays, or ice-bags, are regarded by some as a most important adjuvant to its treatment. In all cases their use demands great caution, and in this country the profession favors the application of heat rather than cold to the spine, in the form of hot-water douches or hot-water bags. Many are in favor of first blistering the region over the spine from occiput to loins, and then covering the parts with a poultice. Blisters at the nape of the neck are of service in most cases after the acute stage is passed. In sthenic cases leeches may be applied over the temples and mastoid processes; they diminish the headache at the beginning of the disease. The extremities must never be allowed to get cold, and warm flannel is to be continually wrapped about the legs and body. Mustard foot-baths, stimulating enemata, and the external use of turpentine, are indicated when the simple means fail to accomplish the desired result. Cold baths do harm. As soon as convalescence shall have been established, a tonic plan is to be adopted. The vegetable bitters, arsenic, and iron are to be used. In some cases electricity may be employed with benefit.

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[1] Mannkopf.

SEPTICÆMIA.

Septicæmia is a constitutional disease due to the absorption into the blood of a septic material which is developed in decomposing animal matter by the action of putrefactive bacteria. The disease is closely allied to "*surgical*" or "*traumatic fever.*"

Morbid Anatomy.—The changes in the blood in septicæmia are similar to those which occur in fevers. It is darker than normal, coagulates less readily, and tends to rapid decomposition. This loss of coagulating power has been supposed to be due to the destruction, by the septic poison, of the white blood corpuscles, which contain the main factors for producing a clot. Bacteria and micrococci are present in the blood in septicæmia.

The *spleen* is enlarged and often softened. The heart, kidney, and liver exhibit more or less cloudy swelling. The mucous membrane of the *stomach and intestines* is congested and œdematous, and the agminated and solitary glands are prominent. Enteritis is not infrequent. In severe cases ecchymotic spots are found in the intestinal tract.

The *serous* membranes *may* be inflamed, but generally they are only ecchymotic. There is always more or less lymphangitis present. It seems evident that the septic material enters the circulation chiefly through the lymphatic vessels.

Etiology.—The nature of the septic poison that is the product of the decomposing animal tissues is still a matter of dispute. Some claim that it is a chemical substance, formed in a wounded part, which acts as a ferment in the blood, and produces the septic symptoms. Others regard the bacteria which are present as themselves the sole cause of the septic infection. Several pathologists have attempted to find the true poisonous principle, and have isolated—from decomposing fluids—"*sepsin*" and other ptomaïnes. It has been observed that the *blood* of an animal with septicæmia produces greater disturbances and graver results, when injected into a healthy animal, than decomposing fluids.

Dr. Sanderson, who says "that the agency of bacteria is essential to the production of the septic poison," also says that "they are incapable of producing the poison in a healthy organism." After considering the various theories which have been advanced, it seems most probable that there is no one body which causes the septic infection, but a combination of a number of poisonous substances which produce changes analogous to those caused by fermentation, and that the poison is absorbed by the blood-vessels only in exceptional cases, when their walls have undergone degenerative changes.

Decomposing tissues which cause septicæmia may be *in* the body, on the *surface* of the body, or *outside* of the body.

I. Thus, a decomposing placenta *in utero,* sloughing ulcers in typhoid fever, necrotic processes in chronic phthisis, diphtheritic sloughs, ulcerative endocarditis, abscess and gangrene of the lung,—these are some of the internal conditions which may induce septicæmia.

II. Wounds, gangrene, decomposing membranes, or the suppuration and necrosis in small-pox, any ill-conditioned wound, especially if lacerated and contused, may cause septicæmia.

III. Dissecting wounds and *post-mortem* manipulation of those who have died of infection, even without a surface abrasion, may induce septicæmia. The respiratory and the gastro-intestinal tracts are sometimes the mode of entrance of the infection.

Symptoms.—The symptoms of septicæmia will vary with the amount of the septic material introduced into the system and the length of the infection; a slight infection will produce fewer and less grave symptoms than one more extensive; hence the symptoms will vary:—sometimes urgent, sometimes so mild as to be overlooked. In a well-marked case, after a rigor, or feeling of chilliness, but rarely a distinct chill, there is a rapid rise in temperature; 105° or 107° F. may be reached within the first twenty-four hours. There is no typical range to the temperature. The pulse is rapid (120 to 140), feeble and thread-like. The mouth, tongue, and surface of the body become hot and dry. If sweats occur they are very slight, and only present during the initial stage, and can hardly be confounded with the profuse sweats of pyæmia. Vomiting is not infrequent. The nervous symptoms are always well marked. The expression of countenance is dull and apathetic, the patient lying in a listless condition, generally free from pain. There is restlessness and low, muttering delirium. The respirations are feeble, labored, and hurried. The skin may be *slightly* jaundiced. Diarrhœa is present in about 50 per cent. of all cases, and in nearly all severe cases. The urine is scanty, high colored, of high specific gravity, and contains urates and often albumen.

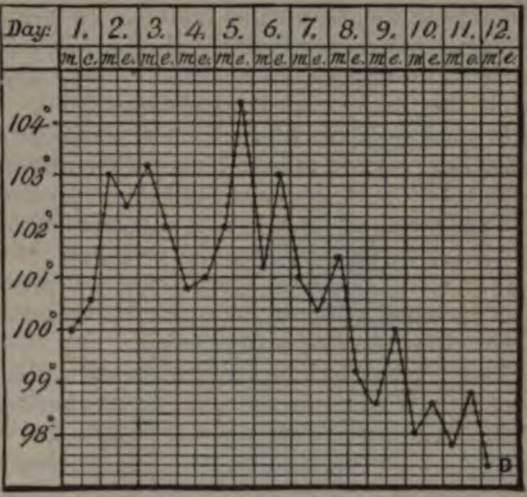

FIG. 158.
Temperature Record in a case of Septicæmia, following an Amputation.

In mild cases the symptoms may remit, and complete recovery be established within a couple of days. This happens when the septic cause is discovered and removed. In severe cases death may occur within twenty-four or seventy-two hours, the patient dying in complete collapse. Typhoid symptoms, a dry tongue, rise in temperature, diarrhœa, and muttering delirium, following an abortion or child-birth, should always excite suspicion. Septicæmia often gives rise to pyæmia, or is combined with it, which is shown by the initial chill being severe or often repeated, and by the occurrence of profuse sweatings.

Differential Diagnosis.—Septicæmia may be confounded with *pyæmia*, *typhoid* and *typhus* fever.

Pyæmia is ushered in by a distinct chill; septicæmia by slight shivering or mild rigors only. In pyæmia the chills recur; in septicæmia there is but one—*the initiatory*—chill. In pyæmia there are profuse sweats which *recur;* in septicæmia there are slight, if any, sweatings, and they are never recurrent. In pyæmia the temperature gradually rises to 102° to 104° F.; in septicæmia it is high at the onset, *i. e.*, 105° to 107° F. The skin is of a dark, leaden yellow, jaundiced hue in pyæmia, while the discoloration of the skin is never so marked in septicæmia. There is a sweet "sickish" odor to the breath in pyæmia, absent in septicæmia. Pyæmia develops slowly, septicæmia rapidly. In pyæmia the heart impulse is less forceful than in septicæmia. Finally, *infarctions, thrombi* and *multiple abscesses* develop in pyæmia and are its distinguishing objective evidence, while they *never* occur in simple septicæmia.

Prognosis.—This, in most instances, depends upon the extent of the poisoning, "when the symptoms of the disease are well marked the prognosis is bad." The possibility of the removal of the source of the infection, and the length of time that the decomposing mass has been in contact with the living tissues, influence the prognosis. Its duration is from two days to two months. Death occurs from asthenia, exhaustion, or rapidly from overwhelming of the system with the *materies morbi.* Collapse is nearly always the precursor of dissolution.

Treatment.—The first thing to be accomplished in the treatment of this condition is the discovery and, when possible, the removal of the cause. Antiseptics should always be used at the seat of the infection. The bowels must be freely acted upon by salines throughout the whole course of the disease. The tonic, stimulant and antipyretic plan laid down for the treatment of pyæmia should be employed here. Quinine, salicylic acid, and brandy are the three drugs on which we place our reliance. Tanner recommends quinine and nitric acid. The diet must be as nourishing as possible.

Billroth's treatment is cooling drinks, a fever diet, morphine at night to secure sleep, from six to ten grains of quinine during the afternoon; the induction of profuse perspiration when the skin is dry, by warm baths, afterward wrapping the patient in blankets.

PYÆMIA.

Pyæmia is an infectious disease, and under certain conditions is contagious. It is attended by the formation of infarctions, metastatic abscesses, and diffuse local inflammation. Many authors make no distinction between septicæmia and pyæmia. The bacteria found in the blood are the same as are present in ordinary suppuration.

Morbid Anatomy.—The blood in pyæmia is characterized by a tendency to coagulate spontaneously wherever there is slowing of the blood-current. Colonies of micrococci are very frequently found in the blood and on the walls of the vessels;[1] venous thrombosis and embolism are essential features of this disease. The thrombi are usually near the seat of the pus absorption; these emboli have a specific action on certain organs, stamped as they are with the peculiar pyæmic infection. When these emboli become lodged in the small arteries of different organs they lead to the development of infarctions which terminate in the formation of abscesses.

"*Metastatic abscesses*," the result of suppuration of a pyæmic infarction, caused by venous thrombosis and embolism, may form in the lungs, liver, kidneys, spleen, muscles, heart and brain.[2] In the *lungs* there is usually more or less pneumonic inflammation about the abscesses. Even patches of gangrene may be found near them. In the *kidney* the tubules and vessels are found crowded with micrococci.

The *spleen* is swollen and shows more or less parenchymatous degeneration, according to the amount of fever. Generally will be found, scattered through the organ, a few firm wedge-shaped nodules with their apices inward, or their interior partly broken down into pus. Metastatic abscesses vary in size from a pea to a large walnut. When multiple abscesses are found scattered through the various viscera, softened puriform and decomposing thrombi are rarely found in the veins; but when the abscesses are *few* the reverse is the case. The

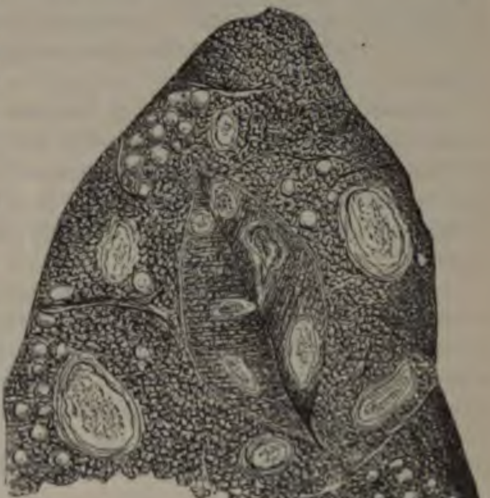

FIG. 159.
Pyæmia.
Metastatic Pyæmic Abscesses of the Lung.

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[1] Weigert states that small thrombi are often formed solely of bacteria.

[2] Recklinghausen says that these abscesses depend on "*extra-vascular accretions of fungi.*"

joints, the *serous* membranes of the body, and the connective-tissue of various parts are often involved.

Pleurisy, pericarditis, and peritonitis of pyæmic origin are frequent and always fibrino-purulent in character. I have known the pleural cavity to fill with pus twenty-four hours after the first evidences of pyæmic suppurative pleurisy. Suppurative arthritis is a rare complication. Lymphangitis is usually established in the neighborhood of the injury or source of infection.

Ulcerative endocarditis with the presence of large quantities of bacteria is not infrequent. Pyæmic pan-ophthalmia with sloughing of the cornea is of rare occurrence. In some cases nearly all the tissues and serous and mucous membranes exhibit deep post-mortem staining; the gastric and intestinal mucous membrane being swollen and congested, the solitary glands and Peyer's patches prominent. Ulcers may form at points along the intestine. The skin always shows more or less jaundice. Suppurative cellulitis often occurs. Occasionally there are cases of pyæmia, or conditions closely resembling pyæmia, where there are no recognizable pathological lesions.

Etiology.—Recent observations and experiments seem to show—*first*, that pus with micrococci causes suppurative pyæmic inflammation; *second*, that the micrococci *alone* can establish a similar inflammation; and *third*, that without micrococci pus is inert. Many regard the pyæmic and septicæmic poison as identical, and pyæmia as nothing but a metastatic septicæmia, claiming that pyæmia is *invariably* associated with more or less septicæmia, and therefore have advised the use of the term septicopyæmia.[1]

Inflammation of bone is a very frequent cause of a *phlebitis* which leads to pyæmic infection. Thus a blow on the head of one saturated with alcohol is followed by a phlebitis in some of the diplöic veins; as a result, thrombi are formed, which break up into emboli and thus lead to pyæmic infarction and abscess. Suppuration of the eye or middle ear has led to the same results.

Cellulitis, carbuncle, erysipelas, "malignant pustule," and dissecting wounds are often complicated by pyæmia. Endometritis or lacerations about the genital tract are fruitful sources of pyæmia in the puerperal state. The question of pyæmic contagion is still unsettled. From a surgical standpoint it seems evident that certain atmospheric conditions and surroundings and want of cleanliness predispose to the development of the pyæmic bacteria, but it has never been shown that pyæmia can be contracted as small-pox or scarlatina can.

Symptoms.—Pyæmia is ushered in by well-marked symptoms. First, there is a chill or decided rigor, followed by a gradual rise of temperature to 101° or 104° F., the rise of temperature being proportional to the phenomena of the chill. The chills of pyæmia occur irregularly, rarely at night, and are followed, after the first two or three, by profuse and exhausting sweats, which only afford marked relief for a time, the skin soon becoming hot and dry. An irritability of the nervous system has been noticed as preceding the occurrence of these chills. During the chill the temperature will be higher than in the sweating stage, the thermometer often showing a temperature of 103° or 105° F., or 108° F., which often suddenly falls below the normal, soon to rise again.[1] The heart power is notably and early diminished. The pulse is frequent, 120 to 140, small and often intermittent; it does not vary with the range of temperature. The conjunctivæ and skin assume a sallow tinge; later they may become markedly jaundiced. The breath has a peculiarly sweet, sickish odor. The tongue is at first covered with a white fur; later it becomes glazed, dry, brown, and fissured. Sordes collect on the teeth. Anorexia is marked from the onset. The patient complains of great thirst. The bowels are usually relaxed. The copious diarrhœal discharges, with the attendant nausea and vomiting, soon bring about a condition of asthenia. The mind remains clear up to the time of great exhaustion and the appearance of multiple abscesses in some central organ or organs; then the patient becomes dull, apathetic, and often slightly delirious. The respirations are hurried and shallow, and are always more accelerated just before a chill or sweat. As death approaches, delirium occurs, the pulse becoming more feeble and intermittent, reaching at times 150 or 170, or 200; the face has a yellowish, leaden hue, and finally the patient passes into a comatose state and dies.

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[1] *Holmes' Sys. Surg.*, Vol. v. Sanderson claims that "pyæmic poison multiplies in the organism." Whether the poison becomes more infective or virulent as time elapses or as it is developed from new foci, is a mooted question. Panum claimed that "there is, in putrefying fluids, a specific chemical substance soluble in water and which, when introduced into the blood, causes the symptoms of putrid or septic infection." Others claim that pyæmia is due to a peculiar miasm which has a specific action similar to the exanthemata, and which may be introduced through the lungs, mucous membranes, and through abraded surfaces.

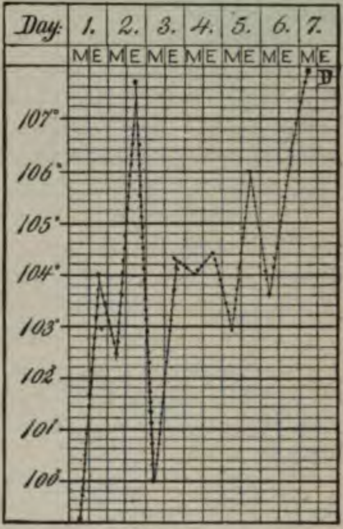

FIG. 160.
Temperature Record in a case of Pyæmia.

When the internal organs are involved the local signs of multiple abscesses will be present. The physical signs of pyæmic pulmonic infarctions are at first obscure, for the foci are so small and so scattered through the lung that percussion fails to detect them. Usually the evidences of a severe bronchial catarrh accompanied by a cough, with frothy, blood-stained, watery expectoration, are followed by the physical signs of lobular pneumonia.

The *kidney* changes are marked by albuminuria and hæmaturia, together with the presence of epithelial and gelatinous casts. The amount of urea is always increased. The changes in the liver and spleen cause abdominal tenderness, accompanied by a marked increase in these organs as shown by percussion. In pyæmia there is generally more or less jaundice. The signs of arthritis, pleuritis, peritonitis and cellulitis can be early recognized, and should be looked for in severe cases after the second or third day.

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[1] This intermittent type of fever is peculiar to this disease. Billroth says statistics favor the idea that recurrent chills depend on new inflammations, having their chief source in repeated purulent infections about the wound.

Chronic pyæmia is met with among the robust, in whom the infection is moderate, and not often repeated. The abscesses are confined to the cellular tissue and followed by suppuration in the joints, marked debility and muscular weakness. Weeks, or even months, may elapse before death or recovery takes place in this class of cases.

Differential Diagnosis.—The diagnostic points of pyæmia are, irregularly recurring chills and sweats, great variations in temperature, with the signs of multiple abscess in the internal organs. It may be mistaken for *septicæmia, intermittent* (malarial) *fever, acute yellow atrophy* of the *liver, acute articular rheumatism, typhus* and *typhoid fever*. The diagnosis between pyæmia and septicæmia has already been considered under septicæmia.

The paroxysms in *intermittent* fever are regular in their development and time of occurrence; they are not so in pyæmia. The temperature in intermittent fever ranges higher than in pyæmia. There is slight, if any, jaundice in malarial fever, while deep hematogenous jaundice is common in pyæmia. The history of the case, together with the presence or absence of small points of local infection, helps to differentiate between the two diseases. The sweet, nauseating breath, marked muscular prostration, and dull expression of the face, are noted in pyæmia and not in intermittent fever.

The points of differential diagnosis between pyæmia, yellow atrophy of the liver, rheumatism, and typhoid fever are found in the history of these affections.

Prognosis.—In pyæmia the prognosis is always unfavorable. Some deny the possibility of recovery in a well-marked case; still recovery is possible in cases that are mild at the onset and slow in their development, in which the chills are not often repeated, the intermissions between the exacerbations of fever are long, loss of strength is not rapid, and the tongue remains moist. Thus we see that the prognosis depends entirely on the course of the disease. The duration of pyæmia varies: it is usually acute, lasting from two to ten days, often, subacute, lasting from two to four weeks, and rarely chronic, when it may run on for months. The duration of puerperal pyæmia is usually about one week. If death occurs in four or eight days, it is due to the intensity of the pyæmic poison. If later, it depends upon the exhaustion incident to the formation of abscesses and the occurrence of complications. The earlier the symptoms of multiple abscesses appear the more hopeless the case.

It must be remembered that pyæmic patients differ in their power of eliminating poison; hence in some cases the system will be at once overwhelmed, while in others the shock will be recovered from. Every day after the eighth that the patient survives increases the chance of recovery.

Treatment.—The treatment of pyæmia may be divided into the prophylaxis, and the treatment of the developed disease. Its prophylactic treatment is by far the most important: it consists in avoiding everything that may favor the development of the disease, the details of which are included under the general management of surgical operations and the treatment of wounds. The history of these antiseptic methods comes within the domain of surgery rather than medicine. Obstetricians cannot be too careful in these matters.[1] *Cleanliness*, good ventilation, sunlight and quiet are important prophylactic measures. There are undoubtedly certain atmospheric conditions which influence the development of pyæmic marasmus, and are always to be considered.

Pyæmic poison, if eliminated at all, is eliminated by the intestinal tract and not by the skin or kidneys, and treatment should be directed to aid this elimination.[2] With the idea of neutralizing the pyæmic poison after it has gained access into the body, or counteracting its effects, nearly all the antiseptics have been employed. The sulphites and hyposulphites of sodium, calcium, and magnesium; carbolic and salicylic acids; the oil of turpentine, and many like agents,—are still under trial. Quinine is the drug which is most extensively employed for its antiseptic and also for its stimulant and antipyretic powers.

The most important thing in the treatment of pyæmia is to support the patient; and, with this end in view, the largest possible amount of nourishment and stimulants should be administered. There is no disease in which so large an amount of stimulants can be administered with benefit as in pyæmia. The indications for their administration are the same as in the essential fevers. If life can be prolonged, in mild cases, until the violence of the infection is passed, recovery is possible.

DIPHTHERIA.

Diphtheria is a specific constitutional disease, characterized by a granular, fibrinous exudation upon the surface and into the substance of mucous membranes, and upon abraded surfaces. Various countries of Europe were visited by epidemics of diphtheria in the sixteenth and seventeenth centuries. In the middle of the last century it reached England, and in the early part of the present century it prevailed at different points in the New England States.

Dr. Samuel Bard, of New York, gave the first accurate description of the disease in this country, and brought clearly before the profession its specific contagious character; his clear accounts tally perfectly with the experience of the present day.[3] The labors and investigations of Louis,

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[1] An interesting proof of this is shown in the case related by Dr. Teale: Three gentlemen who aided him in dissecting a subject who died of hernia attended the same day five midwifery cases in all. Four of these cases died of puerperal fever, while no other cases of it occurred in their extensive practice.

[2] Billroth says diarrhœa is a severe complication which quickly induces collapse.

[3] He published his pamphlet in 1812.

Trousseau, Rilliet, Graefe, and Virchow have done more than those of any other investigators to perfect our knowledge of diphtheria.

Morbid Anatomy.—The characteristic pathological lesion of diphtheria consists in a membranous infiltration of some mucous surface. Of the mucous surfaces, those of the pharynx, tonsils, uvula, and nasal passages are its most usual seats ; beginning on the tonsil and anterior wall of the pharynx, the diphtheritic process may extend upward into the posterior nares, forward into the anterior nares, or pass down the larynx, larger bronchi, bronchioles, and even enter the air-cells. From the pharnyx it may pass down into the œsophagus, the larynx often escaping ; or it may first appear in the larynx and extend upward into the pharynx : this latter is rare. Occasionally the mucous membranes of the mouth, stomach, vagina, rectum, and biliary passages are the seat of the diphtheritic process. If the skin is abraded it may become covered with diphtheritic exudation.

The first change in the part that is to be the seat of this exudation is *a passive hyperæmia ;* the capillary vessels are gorged, and the mucous membrane is of a dark, purplish-red color ; somewhat swollen at the point where the membrane is to develop. The hyperæmia is *not* active, the color is *not* the bright red of active inflammation, but dark, livid, and "angry." The amount of serous infiltration of the sub-adjacent tissues determines, in each case, the amount of tumefaction. On the surface of the affected part there is an abnormal secretion of mucus, and the epithelial cells covering it become enlarged and cloudy, from exudation into them. Little by little they lose their nuclei and become transformed into a homogeneous mass, presenting numerous ramifications—in other words, the metamorphosed epithelium-cells form a reticulated membrane.

The first and most superficial diphtheritic exudation is into the epithelium. The cells of the deeper structures may be simultaneously or secondarily involved. The surface exudation becomes thicker and of a grayish color as the sub-epithelial mucous and sub-mucous coats become successively involved. In mild cases the membrane first resembles a gauzy film, then it assumes a light yellow color, or occurs as white patches of varying size. In severe cases a leathery, gray exudation from one-eighth to one-fourth of an inch in thickness will form in five or six hours, which can be removed, and when removed leaves a raw bleeding surface which will immediately be covered by a new exudation. The membranous exudation may become infiltrated with blood, and assume a black color ; this is not a condition of gangrene, but gives evidence of great blood changes. Absorption of the exudation is only possible when the epithelial layer is alone involved ; when the mucous and sub-mucous tissue is involved, the membranous exudation can only be removed by a suppurative or gangrenous process.

As the exudation is taking place into the epithelium, micrococci or spherical bacteria will be found ; as the diphtheritic process involves the deeper tissues, the bacteria greatly increase in number. Some regard the bacteria originating *within* the epithelial cells as the result of the pathological processes; others as the cause of the pathological changes.[1] It seems reasonable to regard the diphtheritic exudation as a granular fibrin of low vitality, which possesses no power of organization.

The arrest of the diphtheritic process may be first by *suppuration*. Underneath the diphtheritic exudation, there may be a suppurative process established, which separates the layer of exudation from the tissues which it involves. In such a case the membranous exudation becomes more sharply defined at its boundary, the tumefaction of the surrounding mucous membrane subsides, the inflammatory zone draws closer and closer to the margin of the patch, whose edges curl up, and finally the mass is removed from its base, and is thrown off spontaneously. The duration of this exfoliative process varies from two to five days. In some cases the diphtheritic process is so mild that while the exudation occupying the epithelium is thrown off, that in the subjacent structures is absorbed, no suppurative process occurring. In this, the mildest form, there is absorption accompanied by a simple epithelial desquamation.

Another termination of the local diphtheritic process is in *gangrene*. The nutrition of the tissues is so extensively and rapidly interfered with by the abundant exudation that the blood supply is cut off and death of the parts is the result. Some observers claim that gangrene is caused by the obstruction of the lymphatics with bacteria. With the gangrenous process large numbers of putrefactive bacteria develop in the membrane and in the tissues underneath, which break down into a semi-fluid, dark mass, which has the peculiar odor of gangrene. Sometimes the sloughs are quite firmly attached to the adjacent tissues. The so-called septic variety is characterized by the formation of an extensive necrotic membrane, the color of which is a dark gray, or brown with streaks of capillary hemorrhages throughout its entire extent. Larger colonies of micrococci are found in its deeper layers, and pressing against the fibrinous bands they form alveoli, in which myriads of the bacteria lie. The exudation in this variety occurs most frequently in the nasal cavities, where in and beneath the Schneiderian mucous membrane is an extremely rich plexus of lymphatic and blood-vessels. This variety may involve tissues other than the mucous and submucous layers; cases are recorded where the vomer was eroded, and little depressions in it were filled with nests of micrococci.

A piece of membrane on examination will be found soft and friable, breaking down into an ichorous, semi-fluid, dirty brown pulp. If a portion of "septic" diphtheritic membrane is removed, ulcers are found in the tissue beneath, which may be shallow or deep. When shallow, they bleed very readily; when deep, they are covered by dirty gray sloughs. The variety of the epithelium, and the number of mucous glands in the mucous membrane involved in the diphtheritic process, modify the pathological course of the exudation. When diphtheria invades the bronchioles and alveoli, the characteristic microscopical appearances of the exudation are discovered, and some air cells are filled with micrococci.

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[1] Zahn thinks he can distinguish three varieties of diphtheritic membrane: (1) one that is the result of morbid processes situate in the pavement epithelium; (2) one that arises from solidification of a muco-fibrinous exudation; (3) and one that is the result of solidification of a fibrino-puruloid exudation. This careful histolo-pathologist states further that any or all forms of the bacteria may be found in each of his varieties, that they may also be absent, and that they are not an essential factor in diphtheria.

The *heart* is pale, flabby, and friable, presenting changes similar to those in typhoid fever. The right heart is often filled with clots ; and the pericardium may be the seat of numerous ecchymotic spots, rarely of large size. Endocarditis is not an infrequent complication, and when present it is in many cases *ulcerative*, particularly if the disease has been severe and there are extensive blood changes.

The *blood* sometimes is but slightly altered; in the severer forms it is thick, of a dirty brown color, slightly coagulable, and *after death* contains micrococci. The arteries and veins are equally filled. A transient increase in the number of the white blood corpuscles is very common.

The *spleen* is usually enlarged, the capsule is tense, shining, and covered with numerous points of capillary hemorrhage. The splenic parenchyma is congested, softened, and friable, darker than normal, and often the seat of multiple infarctions.

The *lymphatic* glands become swollen and inflamed on account of their free communication with the infected parts. The hyperplasia occurring within the gland causes a swelling which may be tense and hard or doughy. This doughy feel results from œdema of the peri-glandular and subcutaneous connective-tissue, in which vegetable parasites are frequently found. The lymph vessels are often clogged with micrococci. Suppuration in the lymphatics is of very rare occurrence.

The *kidneys* are congested or the seat, in severe cases, of parenchymatous nephritis, differing in no respect from that occurring in scarlatina, which has received the name of scarlatinal nephritis. Those who favor the parasitic origin of diphtheria regard the micrococci as the starting-point of the morbid nephritic processes; yet they state that if a child die rapidly from suffocation, only a cloudy swelling of the epithelium exists; but if the disease shall have progressed for several days, attended by severe symptoms of intense blood poisoning, then the micrococci are discovered. Thus the morbid processes, by their own statements, are shown to be primary and not secondary to bacterian developments or migration.

The Brain and Cord.—In severe cases there are numerous small spots of capillary hemorrhage scattered throughout the meninges of both brain and cord. These extravasations may sometimes be large enough to form clots, and then softening of the brain in localized spots will occur. Cells and nuclei swell the spinal nerves at the point where their roots join, so that their thickness may be twice the normal; blood may be extravasated at this point, and then the swelling will be distinctly red. Punctate hemorrhages into nerve centres where white matter is predominant are said to be the cause of diphtheritic paralysis, and after degenerative processes have occurred in these hemorrhagic spots the paralysis gradually disappears.[1]

Etiology.—Diphtheria is a contagious disease, often prevailing epidemically. It is undoubtedly of microbic origin, although its specific microbe has not as yet been universally accepted. Many of its etiological conditions are identical with those of typhoid fever : filth, bad sewerage, overcrowding, etc. I have met with diphtheria in houses where the water and sewerage pipes were defective, and where no other causative factor could be found ; nevertheless I have a belief that the miasm of diphtheria *must* be present with the other etiological conditions before diphtheria will be developed. Trousseau claims that the infectious element is confined to the exudation, but many clinical facts indicate that it is present in the exhalations and in the excretions, *as well as in* the exudation itself. Diphtheritic contagion clings to the objects that have been in contact with the diseased individual, and may thus be carried a long distance.

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[1] *Einiges über Diphtherie.* L. Buhl, Zeit. für Biol., III. 4. 1867.

Another vexed question in its etiology : Is it first local, and then constitutional, or *vice versâ?* From a clinical standpoint it seems that the disease starts locally ; that some particle, for instance, of the exudation, too small to be detected with the unaided eye, is received upon the mucous membrane of the pharynx, nose, mouth, larynx, trachea, vagina, or upon a cut or abraded surface, and thence contaminates the whole system. The point of infection cannot be determined until after constitutional infection has taken place. It seems to be well established that when the local signs of diphtheria are present, there is already a constitutional infection. The experiments of Oertel[1] and others show that the point of inoculation is the point from whence radiates the disease.

The stage of incubation usually varies from one to eight days ; it may last one month ; when the disease is directly communicated, as in some recorded instances when a piece of diphtheritic membrane is dislodged and coughed into the mouth, nose, or eye of the physician or attendant, the disease has developed within twenty-four hours. But the question arises : May not the one thus attacked have been under the influence of the diphtheritic poison for some time, and thus prepared for the rapid reception of thé local poison ? During epidemics the period of incubation is shorter, and there is reason to believe that the more virulent the poison the less time elapses between the exposure and the initial symptoms. As a rule, the latent period of diphtheria rarely exceeds five days.

Diphtheria may prevail as an epidemic, or be endemic. It also occurs sporadically. Sporadic cases occur most frequently in those localities where the disease has prevailed as an epidemic. Climatic influences have little to do with its development, but autumn and spring are the seasons when the disease is most fatal. *Age* is a powerful predisposing cause ; from the second to the fifth year is the period of greatest susceptibility ; but no age is exempt. Previous attacks afford no immunity against subsequent ones ; certain individuals seem to be perfectly proof against the diphtheritic infection. Filth, bad sewerage and drainage, overcrowding and a general bad hygienic condition favor the development and spread of diphtheria. Exposure to cold and wet, and sudden chilling of the body, may bring on, or hasten an attack of diphtheria during an epidemic. In a region where the soil is porous, the spread of the disease is much less extensive than in clay soil.

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[1] *Experimentalle Untersuchungen über Diphtherie.* M. J. Oertel, Deutsch. Arch. für klin. Med., viii. 1871.

Symptoms.—The symptoms of diphtheria are local and constitutional. The constitutional may precede the local; or both may appear at the same time. In some cases the local are the primary, and for a time the only signs of the disease. The ushering-in symptoms vary not only in different epidemics, but in different cases during the same epidemic. It is a disease which has no typical course.

The *local* symptoms begin with a sensation of dryness and prickling in the throat, with, perhaps, slight pain independent of attempts to swallow. There is more or less stiffness along the angle of the jaw. Deglutition becomes more and more painful; solids do not cause as much dysphagia as fluids. There may be marked and painful swelling of the glands at the angle of the jaw—in the bifurcation of the common carotid artery—but many severe and fatal cases are accompanied by only slight glandular enlargement, and occasionally the disease runs its entire course without any glandular swellings. In some epidemics most of the cases will be attended by extensive glandular swellings, while others, of equal severity, only exhibit this symptom in a slight degree. However the disease may commence, when fully established there will be noticed upon the anterior pillars of the soft palate, the velum, and tonsils, sometimes also upon the posterior pharyngeal wall, whitish patches, surrounded by a livid, tumefied and congested mucous membrane. At this early stage the exudation can be removed without causing even punctate hemorrhage; and under the microscope the epithelial cells will exhibit the appearances that have already been described. The sub-epithelial tissue becomes œdematous, and later, both tonsils, the uvula, and the anterior part of the soft palate will become œdematous. At this period the membrane may be easily removed, but soon after its removal it will reappear in the same situation, and have the same extent. As a rule the more extensive the exudation, the thicker will be the membrane and the firmer its attachment.

When the posterior *nares* are involved it is usually the result of the extension of the diphtheritic process upward: the tonsils, uvula, and posterior pharyngeal wall having been the primary seat of the exudation. A coryza is soon developed, and the sanious, ichorous discharge irritates, reddens, and excoriates the surfaces over which it flows. At the same time rapid swelling of the cervical, lymphatic, and sub-maxillary glands occurs; and there is undoubtedly a special connection between enlargement of the glands at the angle of the jaw, and diphtheria of the nares and posterior wall of the pharynx. The nostrils are soon clogged, often completely so, and repeated attacks of epistaxis may mark the later stages of nasal diphtheria. When the disease extends upward the parotid is not infrequently swollen and tender. Nasal diphtheria is in a few instances primary. The nose, in cases of invasion of the posterior nares, is often swollen and red, or shining and œdematous; the parts excoriated by the flow from the nostrils are soon covered with ulcers, and the latter are often covered with the gray diphtheritic exudation.

When the *Eustachian tubes* are involved, there will be tinnitus aurium, darting pains on attempts to swallow, marked loss of hearing, and perhaps otitis; perforation of the tympanum, and caries of the adjacent bones may result. The external ear has in rare cases been the seat of secondary and also of primary diphtheria. The middle ear has also sometimes been implicated. The eye may be invaded in diphtheria when the nasal duct is the seat of the exudation; or a piece of membranous exudation being coughed into the eye of the examiner may excite a diphtheritic conjunctivitis. If the diphtheritic process passes downward it may enter either the digestive or the respiratory tract.

If the *œsophagus* is involved there will be a marked dysphagia, and fluids will be regurgitated; accompanying paralysis of the muscles of deglutition in many instances increases the dysphagia. If no special signs of pharyngeal exudation are present in a suspected case of œsophageal diphtheria, portions of the exudation may appear in the vomited matters, for vomiting is an important sign of œsophageal diphtheria. Portions of membrane in the fæcal discharges point to the existence of the œsophageal diphtheria when there are evidences of tonsillar and pharyngeal exudation.

When the *vagina*, *rectum*, or *labia* are involved there will be more or less swelling of the inguinal glands in the immediate neighborhood.

The diphtheritic process may extend from the pharynx into the larynx and trachea. Sometimes laryngeal diphtheria is developed when the primary seat of the diphtheria is in the nasal passages, or in the mouth. Laryngeal diphtheria occurs most frequently in children; the younger the child the greater the liability to laryngeal complication; when adults are attacked, it is the weak and feeble and the *aged*. The epiglottis becomes hyperæmic, livid, and swollen, its edges are harder than the remainder of its substance, and the diphtheritic patches are developed irregularly upon its surface.

The first *symptom* indicative of laryngeal diphtheria is a change in the *voice*, which loses its volume, becomes hoarse, rough and indistinct, then falls to an inarticulate whisper. The *respirations* become noisy and whistling, and dyspnœa becomes more and more urgent as the exudation advances. The *cough* is first dry and stridulous—"brassy,"—but soon changes, losing the brassy tone, and, indeed, it has no distinct tone whatever, and is so peculiar as to be almost diagnostic of laryngeal diphtheria. In children the invasion of the larynx is often sudden. In a short time there is complete aphonia; a cough is developed that is "barking" or "croupy" in character, but (as in adults) it soon becomes abortive. The dyspnœa is extreme; all the auxiliary muscles of respiration are called into play. The attacks of difficult breathing assume a paroxysmal form, and in one of the paroxysms death may occur. If the upper part of the larynx only is involved there is difficulty of inspiration, but when the whole larynx is involved expiration is also affected. There is falling in of the supra- and infra-clavicular spaces during inspiration, showing that imperfect inflation of the lungs results from the mechanical obstruction to the entrance of the air into them. Cyanosis becomes marked, and there is either stupor, or restlessness and jactitation. When the child dies it is with all the symptoms of croup (*q. v.*). Death is not from the action of the poison but from local obstruction which has mechanically forestalled its constitutional effects.

A *laryngoscopic examination* shows epiglottis, vocal cords, and the interior of larynx to be the seat of a diphtheritic exudation, the ventricle being usually wholly obliterated.

Auscultation reveals abnormal laryngeal sounds, together with râles of various kinds, and a loss of vesicular respiration. In some instances the diphtheritic process may have its primary seat in the larynx, and then extend upward, the same processes occurring upon the tonsils, uvula, and soft palate, as when they are the primary seat of the disease. In such cases laryngeal symptoms are present from the onset of the disease.

Constitutional Symptoms.—There are no regular stages in the development of diphtheria, therefore no typical clinical sketch can be given which shall include all cases. The division of diphtheria into the catarrhal, croupous, gangrenous and septic forms can only be made at the expense of facts, for they may rapidly merge into each other, and are only stages of one and the same diseased process. Diphtheria may begin with well-marked, active symptoms, as a chill, fever, pain in the head and back, nausea, vomiting, and even convulsions. Or it may come on insidiously, the patient complaining only of the throat symptoms. It may run so mild a course that the patient at no time feels sick ; the throat symptoms are not marked ; and there is a small patch of exudation upon the tonsils, but it does not extend.

There is little or no febrile movement, and at the end of a week the patient is fully convalescent. In those cases where well-marked symptoms usher in the disease, the temperature ranges higher than in any other form of the disease ; in rare cases it may reach 105° F. by the end of the second day. Insidious cases are marked by a gradual rise in temperature, 102° or 103° F. being the highest point reached during the whole course of the disease. Mild and severe cases often occur in the same household during the same epidemic.

In whatever manner diphtheria is established, the constitutional and local symptoms do not always progress with the same severity. In some cases while the exudation is rapidly extending the temperature falls to normal, the pulse diminishes in frequency, the patient seemingly having decidedly improved in every respect—in fact, the constitutional symptoms remit if they do not intermit. The pulse, however, will show the influence of the poison on the nervous system, either in irregularity or abnormal frequency, or both. But during all this time the membranous exudation is spreading

After a time the temperature rises to 103° or 104° F., the pulse to 120 or 130, and all the severe constitutional symptoms reappear. Death often occurs early in such cases. In another class of cases the constitutional symptoms are severe from the onset, while the local manifestations of the disease are but slight and not progressive. In some cases the nervous system may be overwhelmed by the intensity of the diphtheritic poison at the onset of the disease, and death result before any local manifestations of the disease have had time to make their appearance.

In the more common forms of diphtheria the first symptom will usually be the soreness of the throat, which will be found congested; and either upon a tonsil or at some point in the pharynx, a small white patch of exudation will be seen. Slight febrile movement may accompany the throat symptom, or it may not come on for forty-eight hours. The exudation gradually extends until a large surface is covered with a thick layer of membrane, which assumes a gray or brown color. The sub-maxillary glands become more or less swollen. There is always some obstruction in the throat and difficulty in swallowing. As the disease becomes fully developed, patients are unable to sit up. There is nausea and often vomiting. The urine is usually albuminous. The pulse becomes frequent and feeble; the temperature ranges from 101° to 103° F. The membranous exudation continues to extend, involving more and more of the throat. The patient's general condition becomes worse each day until about the end of a week; when the membrane is thrown off, the pulse becomes less frequent and the patient slowly recovers. Or, as happens in some instances, as the exudation disappears from the tonsils it extends into the larynx: then are developed all the symptoms of laryngeal obstruction, the breathing becomes difficult, cerebral symptoms are prominent, and patients die in a few days after the laryngeal symptoms appear. Occasionally after the local manifestations of diphtheria have disappeared, patients experience a degree of prostration and feebleness that is met with in no other disease. The pulse becomes feeble, frequent and intermitting: and the heart-sounds are muffled and indistinct. Death occurs in such cases as if a poison—such as prussic acid for instance—had been taken.

Symptoms which indicate danger.—Diarrhœa, although not often present in diphtheria, may be so profuse as to cause exhaustion which will hasten the fatal termination. Nausea and vomiting coming on late are most unfavorable symptoms. Albuminuria occurs in mild as well as in severe cases, rarely lasting longer than a week, except in those severe cases where œdema is present, and where epithelial, granular, small hyaline and exudative casts are found in the urine. It is stated by some that the amount of albumen in the urine is in direct proportion to the intensity of the diphtheritic infection. In a few rare instances diphtheritic nephritis has been so intense that death has resulted from it, before either the constitutional or local symptoms of the disease were present. Albuminuria generally comes on toward the end of the first week of the disease. Coma may occur as the result of the nephritis. An erythematous eruption sometimes makes its appearance in diphtheria between the first and third days. Its usual seat is the upper part of the chest and back.

The pulse is peculiar and varies greatly in different cases. There are three distinct varieties:

I. In a large number of cases the pulse from the very commencement is feeble, small, and rapid, ranging from 120 to 160, or, in young children, even to 170 in the minute.

II. There is a class of cases where the pulse rises to 120 or 130 early in the disease, but falls to 60 or even 40 within twenty-four or forty-eight hours from its onset.

III. There is a class of cases in which the pulse is irregular and intermittent throughout the entire course of the disease. The prognosis in the latter variety is always bad.

If the temperature falls to normal, or below, and the exudation shows no signs of exfoliating, no matter how trifling its amount, or how slight the glandular swelling, the case is grave and death is not usually long delayed. Convulsions occurring late in diphtheria are always unfavorable symptoms, while as ushering-in symptoms they have no special significance. Swelling of the lymphatic glands, although not present in all cases, is so frequently present that it must be regarded as one of the symptoms of the disease. If it is extensive so as to interfere with deglutition and respiration, the prognosis is unfavorable.

After the exudation disappears and convalescence is apparently established, sequelæ may develop, which may continue for months and even years. The commonest is *paralysis* of some of the voluntary muscles; the muscles most frequently affected are those of the soft palate and pharynx. Usually the first thing indicating the occurrence of this paralysis will be difficulty in swallowing—first fluid and then solid food—with an inability to articulate clearly. When paralysis is unilateral the velum is drawn to the healthy side; when both sides are involved the velum hangs pendulous and motionless; there is also a loss of *sensation* as well as of motion, for pricking it causes no pain. The voice is altered, and children cry as with a cleft-palate, while adults have a sort of nasal twang. These changes come from paralysis of the velum, anterior pillars of soft palate, and pharyngeal wall. Fluids are only partially swallowed, the greater portion being regurgitated through the nose, especially if the individual is standing or leaning forward while drinking. This variety of paralysis sometimes comes on while the exudation is yet visible in the fauces, just as it is disappearing, or in a week or ten days after it has entirely disappeared. With the dysphagia there is sometimes difficulty in expectorating; the patient will choke, cough and strangle in vain endeavors to get rid of mucus that has collected in the pharynx.

As the pharyngeal paralysis is disappearing,—or from two to ten days after,—the muscles of some other part of the body will be involved—the lower extremities being much more frequently affected than the upper. Though usually beginning in the feet, diphtheritic paralysis follows no regular order; a hand may first be affected, then a leg, and subsequently the other hand, arm and leg. Before the occurrence of the paralysis there will be a sensation of coldness, pricking, crawling (formication) and numbness in the part about to be affected. The patient cannot determine precisely where he has placed his foot—on the floor or some object higher than the floor; movements are ungainly and hesitatingly made, the gait becomes tottering, and finally he cannot stand, the paralysis becoming complete. Sensation is likewise more or less impaired. The hand loses its usual dexterity; the patient being unable to button his coat, or even write his name. When the muscles of the neck are involved the head "wobbles," or is held upright with the greatest difficulty. The neck is usually the last part to be attacked. The power of ocular accommodation is often seriously interfered with on account of the paralysis of some of the ocular muscles. The ciliary and recti suffer oftenest. First, sight is diminished or lost for objects close at hand; and later, distant objects become invisible. There is always diminished refraction, and there may be strabismus and double vision.

When diphtheritic paralysis is general the laryngeal muscles will usually be wholly or partially involved. The voice is hoarse, non-resonant, and often there is complete aphonia. There is no loss of sensation until the superior laryngeal branch of the pneumogastric is involved; this is attended with danger, for particles of food may pass into the bronchi, and suffocative dyspnœa may result in death. In nearly all forms of laryngeal paralysis there is more or less dyspnœa, greatly increased by exercise. If the paralysis involves the sphincters there will be involuntary discharges from the bladder and rectum. The genital organs may be paralyzed, and all sexual desire and power may be lost for months. Paralysis of the muscles of the thorax, trunk and diaphragm gives rise to grave symptoms, pulmonary œdema and death usually resulting. Finally, paralysis of the heart may occur.

Diphtheritic paralysis is always entirely recovered from. In mild cases its duration is two or three weeks, while in others it has continued one or two years. Another sequel of diphtheria is parenchymatous nephritis. When developed during the exudative stage it usually ends in complete recovery. Rarely does it *directly* cause death. When so developed it may be regarded as part of the active history of the disease. But when it occurs during convalescence it may lead to chronic Bright's disease. Inflammation of a serous membrane may complicate or be a sequel of diphtheria; the most frequent serous inflammation is endocarditis.

Pleurisy, peritonitis and pericarditis are of rare occurrence. Chronic pharyngitis is a sequela only in those cases where there has been paralysis of the pharyngeal muscles.

Differential Diagnosis.—The diagnosis of diphtheria rests on the presence of a membranous exudation. When it prevails as an epidemic, a form of "sore throat," a pharyngeal catarrh usually prevails at the same time, sometimes called "diphtheritic sore throat."

This *sore throat* is ushered in by a chill, followed by a more or less intense febrile movement. There is a sense of fulness in the throat with swelling of the sub-maxillary glands, and more or less dysphagia. The mucous membrane over the tonsils is intensely congested, the uvula is œdematous, and a few points of whitish exudation stud the mucous membrane. If these little dots are examined closely they are found to be *mucus*, exuded from the enlarged follicles. The process is purely catarrhal, and not membranous. This is called by some "catarrhal diphtheria," but is nothing more than a catarrhal pharyngitis. It is never contagious, but is due to atmospheric influence, and has none of the characteristic local or constitutional features of diphtheria.

The points of differential diagnosis between diphtheria and *croupous laryngitis* are the following: croupous laryngitis, or membranous croup, is a local affection, while diphtheria is a constitutional disease. Croup is not contagious or inoculable; while diphtheria is markedly so. In croup the exudation is *on* the surface of the mucous membrane; in diphtheria it is *in its substance* as well as on its surface. Laryngeal symptoms are primary in croup, while in diphtheria they usually follow severe constitutional symptoms, and in the majority of cases also follow the appearance of the exudation upon the nasal or pharyngeal membranes. Croup rarely attacks those who have passed the age of puberty; diphtheria attacks all ages. Croup is sporadic; diphtheria is often epidemic. The sub-maxillary glands may be, and often are, enlarged in diphtheria, but *never* in croup.[1] From a clinical standpoint they must be regarded as distinct diseases.

Diphtheria may be distinguished from *scarlatinal sore throat* by the following points:—in scarlatina there is a diffuse redness of the mouth and pharynx; in diphtheria the redness is local and of a darker color. In scarlatina the exudation is mucus, and on the surface of the tonsils soft palate and pharynx; in diphtheria it commences at one point, and spreads, is adherent and tough, and has a grayish or brown color. When an eruption occurs in diphtheria it will have the characteristics already referred to, will last but a few days, and will appear on the *trunk* only. In scarlet fever the characteristic eruption rapidly spreads over the whole surface, lasts three to four days and is followed by desquamation. In diphtheria there is no characteristic eruption, only occasionally transient roseola. The temperature is far higher in scarlet fever than in diphtheria, the ushering-in symptoms more severe, and there is the peculiar strawberry tongue in scarlatina which is not present in diphtheria.

Typhoid or *typhus fever* may be suspected when intestinal diphtheria exists. The only means of diagnosis is to watch the passages for the membrane, and take the temperature carefully; in typhoid there will be the typical range, and in typhus the rise will be sudden and higher. Finally idiopathic *erysipelas of the throat* is very difficult to distinguish from diphtheria. In erysipelas the tongue is blackish brown, dry, and fissured; and there is more puffy swelling of the parts than in diphtheria. The glands are not enlarged and the process is limited in its extent in erysipelas.

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[1] The sides of the neck are to be examined for enlarged glands; those at the anterior border of the sterno-mastoid are always palpable, but it is important to note that the glands at the angle of the jaw are not enlarged.

Prognosis.—The prognosis in diphtheria varies with different epidemics, and with the type of the disease. The prognosis is more unfavorable the younger the subject, since extension into the larynx is more frequent in the young child. The death rate varies in different epidemics from twenty to fifty per cent. A peculiar fact, and one to be remembered, is that a mild case—one where all things are progressing favorably—is liable to assume, in a few hours, a most malignant type. The system may be overwhelmed with the poison even when the exudation shall have disappeared; and, again, in convalescence heart-paralysis may suddenly occur; all of these points make a very guarded prognosis not only safest but necessary. Its duration varies from three to twelve or fourteen days, but death may occur within thirty-six hours; and again the disease may continue three or four weeks.

The symptoms which may be regarded as unfavorable are extreme glandular swellings, huskiness of the voice, a dark-colored extensive exudation, and, above all, laryngeal implication; when diphtheria extends into the larynx, about 95 per cent. of the cases end fatally. Repeated convulsions are unfavorable; and a pulse that is irregular and intermittent, or one that drops to 60 after having been rapid at the onset, indicates danger. When at the same time that the exudation is extensive, it has a dark gray, green, or black color, and when it emits a gangrenous or sickly, sweet odor, the prognosis is unfavorable. Nausea, vomiting, diarrhœa and epistaxis, when they occur late in the disease are very serious symptoms. Coma, accompanied by casts and albumen in the urine, or by entire suppression of urine, is a most dangerous occurrence. If pharyngeal paralysis occurs before the exudation has disappeared, the case is a very serious one; the future course will be troublesome, owing to intense involvement of the nervous system, and these cases are often fatal. The temperature is not a reliable element in prognosis: in the most malignant types of the disease it may range low, between 101° and 102° F. A sudden rise or a sudden fall, especially to sub-normal limits, is exceedingly unfavorable. Primary diphtheria of a wound, in which the throat shows no manifestations of the disease, generally runs a favorable course. Primary infection of a wound with diphtheria, in which the throat becomes secondarily involved, is always unfavorable. Secondary wound infection, during the course of a pharyngeal diphtheria, shows an intense degree of poisoning, and is a bad prognostic omen.

All *complications* render the prognosis unfavorable. Among the complications are meningitis, endocarditis (usually ulcerative), pleurisy, peritonitis, pericarditis, pneumonia, bronchitis, tracheitis, laryngitis, pulmonary œdema and congestion, œdema glottidis, acute Bright's disease, and a septic fever that ordinarily complicates the malignant form. Septicæmia and pyæmia may occur, and intestinal hemorrhage, purpura, and jaundice

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Treatment.—The treatment of diphtheria will be considered under four heads: I. Hygienic; II. External local; III. Internal local; IV. Internal constitutional treatment.

Hygienic.—A patient sick with diphtheria should be kept in bed from the advent of its first symptom until convalescence is fully established, and the pulse is normal in frequency and regular in its rhythm and force. The membranous disappearance is not the guide; it is the exhausted and anæmic condition which demands absolute rest in bed. Only attendants that are agreeable to, and can manage the child well should be admitted into the sick room, which must be large, well ventilated, and have a temperature of 70° to 75° F. Perhaps one of the most important indications is cleanliness; the patient should be kept scrupulously clean,—eyes, nose, ears and mouth, as well as the face and limbs. All utensils of whatever kind, all clothing and linen, must be frequently cleansed and *disinfected*. The disinfection may be accomplished as in typhoid (*q. v.*). The patient must be strictly quarantined, the attendants must mingle as little as possible with the rest of the household, and must avoid taking the breath of, and unnecessary manipulation of, the patient. The rule is, not to disturb a diphtheritic patient except so far as it is necessary for cleanliness. The physician should be careful not to make unnecessary examinations of the throat. The instruments used in the examination should be thoroughly cleansed after each examination. Freshly slacked lime mixed with powdered charcoal may be placed about the room as disinfectants. Fresh air and sunlight are important, and should be so admitted into the sick room so as to avoid draughts. A grate fire in cold weather is the best method to attain ventilation.

External Local Treatment.—This treatment may be considered under four heads:—1. *blood-letting* by means of leeches at the angle of the jaw; 2. *cold applications*—ice-bags—to the throat; 3. *counter-irritation*—blisters, etc.—over the neck and enlarged glands; and 4. *hot poultices* or other hot applications to the throat.

Blood-letting, local or general, while it does not arrest the exudative process, diminishes the resisting power of the patient; clinical experience teaches us that *all* antiphlogistic remedies are contraindicated in the treatment of this disease.

Ice to the throat is with some a favorite plan of local treatment. It is to be remembered that the exudation is a local manifestation of a constitutional disease, and that its extension is arrested and its removal accomplished by the establishment of a suppurative process ; and for this reason the local application of cold is contraindicated. It may relieve pain, but it does not arrest the diphtheritic process.

Counter-irritation is also powerless to check the membranous exudation ; besides, whenever a surface becomes abraded, the diphtheritic process is liable to be established upon it.

If the diphtheritic exudation is arrested and removed by a suppurative process, the external application of *heat* is indicated, and may be of service. Hot fomentations must be regarded as the safest and best means of hastening the removal of the membrane, and they afford the greatest relief to the patient.

Internal Local Treatment may be considered under three heads :—1. Mechanical means employed for removing the membrane. 2. Escharotics employed for its destruction. 3. Astringents to prevent an extension of the exudation by their action on the unaffected mucous membrane.

It is not difficult to pull off a patch of diphtheritic exudation by *mechanical* means ; but the membrane will reappear as soon as the removal is effected ; and the second membrane has a deeper intimacy with the tissues than the primary. For this reason no attempt should be made to remove a diphtheritic exudation unless it hangs loosely detached, and *then* the dependent portion may be carefully snipped off. Any irritation produced by instruments favors the extension of the diphtheritic process. It is to be remembered that, however pleasant it may be for parents and friends to see patches of the membrane removed, after each removal the diphtheritic process is increased both in *depth* and *extent.*

The powerful *escharotics* which have been used for the destruction of the diphtheritic exudation are hydrochloric and nitric acids, nitrate of silver, bromine, chromic acid, etc. It is claimed by partisans of this plan that, when seen very early, and when the diphtheritic patches are small, extension of the exudation may be arrested by the destruction of its local manifestations. There seems no more reason for the use of escharotics than for the mechanical removal of the exudation ; for each one of the escharotic sloughs leaves an ulcer, which is a favorite spot for the development of a new membrane in the deeper tissues.

Astringents, by constringing the mucous membrane about a diphtheritic patch, thus prevent the spread of the exudation. But as the primary action of all astringents is to cause *irritation* of the mucous surface, and as the irritation favors the development of the diphtheritic membrane, their use is contraindicated.

The thing to be accomplished by local internal treatment is to hasten the suppurative process ; the local means which will aid the process of suppuration is the inhalation of the vapor of hot water. The external and internal local treatment of diphtheria resolves itself into the application of poultices externally and vapor inhalations internally. The vapor inhalations should be commenced as soon as the exudation is detected, and continued until all signs of it have disappeared. As the steam inhalation marked and more or less extensive œdema. In more severe cases there is often suppuration, which is generally a diffuse infiltration, but may be circumscribed.

The inflammation may terminate in resolution, vesication, abscess, or gangrene. The former is the usual ending. The hyperæmia subsides, the infiltration is absorbed, and the process terminates in desquamation. When vesication occurs previous to resolution, the cuticle is raised by serous effusion, and when thrown off leaves healthy skin or superficial ulceration. Abscess and gangrene present the same pathological changes as under other conditions.

Erysipelas is not always limited to the skin, but often involves deeper parts. It is somewhat doubtful, however, whether the inflammations of the pleura, pericardium, and peritoneum, which often complicate severe attacks of the disease, are the result of direct extension of inflammation or are due to the systemic poison.

When meningitis complicates erysipelas of the scalp, or laryngitis and œdema glottidis accompany the inflammation of the neck, the relation is probably one of continuity, but the peri- and endo-carditis which are occasionally present in a similar erysipelatous condition, together with the implication of the veins and evidences of nephritic complications, point to a more general etiological basis for such conditions.

The lymphatics are implicated, and their course can be traced by red lines running from the inflamed area to the adjacent glands, which are enlarged and indurated. The specific cocci of the disease are found most abundantly in the lymph spaces. If the veins are involved, a phlebitis may result in infarction, or, more rarely, in pyæmia.

Following an attack of erysipelas there remains some thickening and induration of the skin, which may become permanent after repeated attacks.[1]

Etiology.—All the causes of general debility—as, indulgence in drink, and anti-hygienic conditions—predispose to erysipelas. In a large proportion of cases it is preceded by some abrasion of the surface or a distinct wound, and is then considered traumatic.

Some individuals show a constitutional predisposition to the disease, and certain unknown atmospheric conditions favor its dissemination.

It is fully determined not only that erysipelas, once developed, is highly contagious, and spreads rapidly among surgical patients and puerperal women, but that the disease is the result of a specific contagion which may render buildings, clothing, and the persons of attendants infectious centres.

Inoculation of the bacteria which fill the inflamed tissues produces erysipelatous inflammation. Its contagiousness, period of incubation, and evidences of constitutional disturbance preceding the local inflammation, and the fact that it can be propagated by inoculation, establish its contagious character.

Symptoms.—The symptoms of erysipelas are constitutional and local.

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[1] Virchow.

Both traumatic and idiopathic erysipelas have a period of latency of from two days to a week or more, during which there will be some fever, possibly of a remitting type, with slight chills, headache, and malaise. As the local symptoms appear, the fever increases, and is marked by decided evening exacerbations. It seldom passes 106° F., and the morning remission may be two or three degrees, and be attended by sweating. The fever is accompanied by a rapid pulse, coated tongue, nausea, anorexia, and disordered bowels. In some sthenic cases the attack is sudden, and attended by severe chills and a rapid rise of temperature. The duration of the fever is very varied.

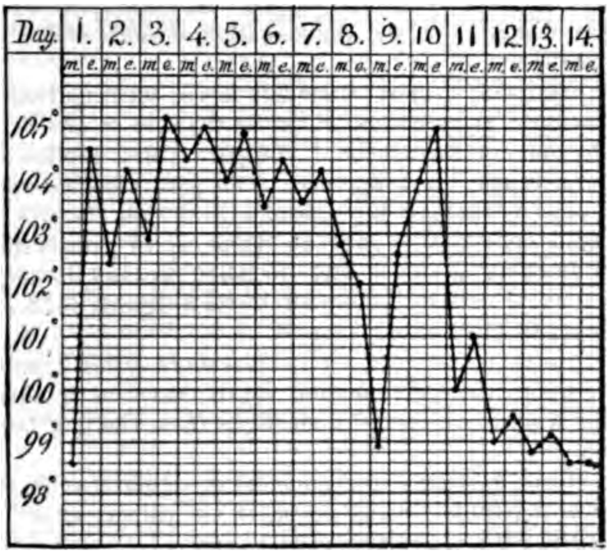

FIG. 161.
Temperature Record in a case of Facial Erysipelas.

When the inflammation is localized and recovery occurs, generally from the fifth to the tenth day there is a rapid decline in the fever and disappearance of all unfavorable symptoms. The temperature may even become subnormal and continue so during early convalescence. When the local condition is progressive, however, the fever continues at 104° or 105° F. for two, three, or more weeks.[1] In these cases the fall in temperature is commonly more gradual and convalescence more prolonged.

In children and nervous patients a mild delirium may be present without any meningeal complications, or in sthenic cases it may become continuous and violent.

When the course of the disease is unfavorable it assumes a typhoid character, the temperature rises rapidly, the pulse is frequent and feeble or irregular, the delirium becomes low and muttering, and passes into coma which ends in death. Sudden extension of the inflammation, or relapses when convalescence is apparently established, are frequent and are marked by a rapid rise of temperature, corresponding with the extent of new tissue involved. Final recovery may thus be delayed for weeks, during which there will be periods of normal or subnormal temperature.

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[1] In a case where the inflammation involved by degrees the entire surface from a wound on the head to the toes, the temperature was between 103°-104° for over four weeks.

The urine is always scanty and high-colored. It contains an excess of urea and often a small amount of albumen.

This course of the fever is often greatly modified by complications.

The local symptoms are equally variable. Traumatic erysipelas begins as a diffuse rose or bright red blush about the point of injury, in which a white line follows the finger as it is lightly drawn across the surface. This is generally preceded for a day or two by some enlargement and tenderness of the adjacent lymphatic glands. In uncomplicated cases there are no further changes, but the inflammation subsides and is followed by flaky desquamation.

Idiopathic erysipelas is most commonly facial, starting from either the nose, eyelid or ear. It may begin either in the skin or areolar tissue, and is attended by more or less œdema. The part is first swollen and secondarily the characteristic blush appears. The part becomes enlarged, hot and painful, and the swelling may extend so as to close the eyes and involve the whole head and neck to such an extent as to greatly disfigure the patient. In from six to twelve days the color becomes darker, the swelling recedes and the cuticle peeling off leaves a slightly reddened surface which slowly regains its normal color.

When the disease is "*erratic*" it extends more or less rapidly from its primary seat, where the inflammation slowly ceases as it advances elsewhere, so that it may be present in all stages from the first faint blush to desquamation.

When the disease assumes a phlegmonous or suppurative form, the pus, if diffuse, gives a peculiar boggy sensation on palpation, but if circumscribed it has the usual appearance of an abscess.

Approaching gangrene is indicated by an intense burning pain, and the parts become livid and finally black and crackling.

Erysipelas may be complicated by meningitis, which will be indicated by its usual symptoms somewhat modified by the preëxisting fever. Pericarditis and pleurisy are at times recognized by dyspnœa, or often only by their physical signs.

Differential Diagnosis.—When erysipelas attacks a joint it may be mistaken for a short time for *acute articular rheumatism*, but the peculiar deep rose color and the rapidity with which the inflammation extends will speedily distinguish it. Similar symptoms with the œdema will also differentiate the disease from *simple erythema*. High febrile movement lasting twenty-four to forty-eight hours, and attended by pain, swelling and tenderness of the lymphatics, has been considered diagnostic of developing erysipelas.

Other questions of differential diagnosis belong entirely to surgery.

Prognosis.—Traumatic erysipelas is a very unfortunate complication in surgical injuries. It arrests all reparative action and adds largely to the gravity of the previous condition.

Idiopathic erysipelas, when it attacks the face and head, is a dangerous and uncertain disease under any circumstances, and is especially so in aged people. Many patients suffer from a simple erysipelas of the face at almost regular intervals without serious discomfort, but there is always danger that the meninges will become involved and the disease at once assume a most serious aspect.

Death may result from the overpowering effects of the poison, from the complications, or from exhaustion due to suppuration, gangrene, or a prolonged course of the disease. Œdema of the glottis from extension of inflammation may cause sudden death. The disease is especially fatal in chronic alcoholism, Bright's disease and gout, and in patients over sixty. Recurrent attacks indicate a debilitated condition and are apt to be of increasing severity.

Treatment.—In common with other contagious diseases, great care should be taken to avoid extension of the disease, and as we are unable to control the poisonous emanations, complete isolation of such patients affords the only sure means of prophylaxis.

In mild cases local treatment is unnecessary, and it is doubtful if it ever restricts the inflammation. Cold dressings with mildly astringent and anodyne lotions are the most grateful to the patient and as efficacious as any. More powerful astringents and distinct caustics—as iodine and a saturated solution of nitrate of silver, or even the actual cautery—have been employed with a view to cut short the inflammation or to prevent its spreading.

Erysipelatous inflammation often improves in five or six days under such treatment, or halts at a line where iodine or silver has been employed; but it quite as frequently, when extending, is not perceptibly restricted by such boundaries. A saturated solution of nitrate of silver may be applied, however, two or three times in twenty-four hours. Subcutaneous injections of carbolic acid in surgical erysipelas have seemed to give more appreciable and better results than any other local treatment. If œdematous swellings are excessive, minute punctures will afford marked relief. Hot applications and poultices are to be used only when suppuration or gangrene is present. We have no means of neutralizing the poison of erysipelas, and internal treatment is confined to general tonic measures. Concentrated nutriment should be administered frequently in small quantities, and stimulants employed as in other acute febrile conditions. The bowels and kidneys should be kept active by cathartics and simple diuretics. Various remedies have been employed, but the tinctura ferri chloridi seems to be generally accepted as the most useful drug, and is even considered to have specific effects in erysipelas.

Quinine and other tonics may be employed with advantage.

The bromides and chloral are preferable to opium or hyoscyamus for producing sleep.

ACUTE MILIARY TUBERCULOSIS.

Although acute miliary tuberculosis is etiologically an acute contagious disease, clinically the dangers of contagion are comparatively slight. In the majority of instances it is secondary to, and a part of, a more chronic tuberculous process, the symptoms of which, in some cases, are so obscure as to escape notice, while the manifestations of the more acute process alone attract attention. More frequently the preceding chronic condition and the acute disease appear as a part of a general tuberculosis. It is only when it occurs without any recognizable previous tubercular infection that it can be considered a distinct disease.

Morbid Anatomy.—While acute miliary tuberculosis is not a local affection and is to be carefully distinguished from acute phthisis, its pathological changes are more abundant and far more frequently found in the lungs than in any other organ. They are also generally present and may be principally located in the pia mater (acute hydrocephalus), intestines, lymph glands, serous membranes, and, rarely, liver, spleen, and brain. The characteristic lesion of acute miliary tuberculosis consists of an irruption of delicate, gray, translucent, miliary granules, varying in size from a pin's head to a poppy-seed. They are quite evenly distributed throughout the affected organs and show little tendency to coalesce.

In the early stages, affected lungs show little change from the normal, aside from the presence of the tubercle granules. Later they become slightly hyperæmic and œdematous, with some infiltration, about the granules, of an amorphous matter. Although the air cells may become partially filled with epithelium, pus-cells, and fibrin, hepatization is of rare occurrence. The pleura is studded with similar tubercular granulations, and they are also present more or less abundantly in the peritoneum and the various glands and organs throughout the body. They can be recognized in some cases in the choroid. In the pia mater they occupy the perivascular lymph spaces.

All tubercle manifests a strong tendency to undergo caseous degeneration, but in acute miliary tuberculosis the patient usually succumbs to the disease before any such change occurs.

Etiology.—The predisposing causes are very prominent in the etiology of acute miliary tuberculosis, and it is very doubtful if it ever occurs when they are not present.

The specific cause of acute miliary tuberculosis, and the manner in which the general infection occurs, has been definitely stated under the heads of tuberculosis and acute phthisis.

Symptoms.—When acute miliary tuberculosis complicates the last stage of phthisis, its symptoms are so modified that it is not easily recognized. In such a case a sudden and decided increase in the fever, and marked aggravation of the dyspnœa, will be the most characteristic symptoms, and, occurring in connection with unchanged physical signs, may lead to a corresponding diagnosis. When the disease attacks an individual in apparent health, the symptoms are well marked.

It is generally ushered in by repeated chills, a rather rapid rise in temperature and pulse rate, and the other symptoms of an acute general disease, accompanied by rapid respiration and a short, dry cough. The temperature ranges from 103° to 106° or 107° F., with irregular but marked remissions, and is more frequently high in the morning and low in the evening than in any other acute affection. The pulse is soft, small, and compressible, varying from 120 to 150 per minute, but in no constant or definite ratio to the temperature. The respirations are from 50 to 60 per minute, and later the dyspnœa becomes intense. The persistent sharp, hard cough is rarely accompanied by expectoration; when present, the expectoration consists of viscid mucus, occasionally blood-streaked. The skin is pale and cyanotic; there is anorexia, rapid emaciation, and diarrhœa, as a rule; the lips and tongue become dry and covered with brown crusts; the patient is dull and semi-comatose, and at night delirious, presenting all the symptoms of the typhoid state. The spleen is generally slightly enlarged.

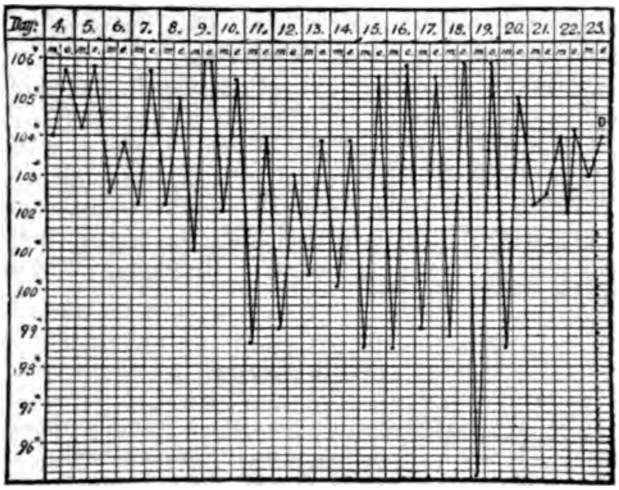

FIG. 162.

Temperature Record in a Case of Acute Miliary Tuberculosis.

The patient may survive for five or six weeks, but more frequently succumbs within two or three. As death approaches, the pulse rapidly grows weaker and more frequent, the cough ceases, the temperature falls, or, if already low, suddenly rises, the cyanosis deepens, and death occurs from pulmonary œdema and asphyxia.

Physical Signs.—In most cases the physical signs are entirely negative. Percussion may show points of slight dulness, surrounded by extra resonant areas, and auscultation occasionally reveals moderate bronchial catarrh, with fine moist râles, but they are not characteristic. A soft friction sound may be produced by a roughened nodular pleura.

Differential Diagnosis.—The symptoms of acute miliary tuberculosis are often so similar to those of typhoid fever that a diagnosis is exceedingly difficult. The points have been given under typhoid fever.

Pneumonia and *acute diffuse bronchitis* in their early stages may simulate acute miliary tuberculosis, but the rapidly developing physical signs and the absence of the constitutional symptoms of an acute wasting disease render an early diagnosis possible.

Prognosis.—The prognosis is unfavorable. The duration of the disease is from a few days to six or seven weeks, with an average of three weeks. The more general the infection, the more violent the fever and the nervous symptoms, the sooner is a fatal termination to be expected. When complicating phthisis, its course is very rapid. Asphyxia from pulmonary œdema, asthenia, cerebral anæmia and collapse, are the principal causes of death, but in about one-third of the cases it occurs from implication of the meninges.

Treatment.—The only indications for treatment which afford any hope of attaining favorable results are in the way of *prophylaxis*, and are largely included in the treatment of the predisposing diathesis. In treating scrofulous patients, acute miliary tuberculosis should be remembered as among the impending dangers. Caseous matter, wherever situated, should be removed when the attending danger is not great.

When the general tuberculosis has once occurred, treatment is confined to the reduction of temperature and supporting the patient. For the first, quinine is of little avail. It is sometimes used as long as recurring chills are present, but is of doubtful value. Cold will be found more useful in reducing temperature, and may be used in baths, packs, or by sponging. Stimulants and highly nutritious food fulfil the second indication, and must be used as in other wasting diseases. Morphia must be used for the relief of the cough and dyspnœa.

TYPHUS FEVER.

Typhus fever is a contagious disease, which usually prevails epidemically. Although it has many phenomena in common with miasmatic-contagious fevers, and was at one time classed with typhoid fever, to-day it is regarded as a distinct type of fever, dependent upon a specific poison with certain pathological and etiological phenomena, which distinguish it from all other forms of disease. It has received a great variety of names, such as *ship fever, hospital fever, jail fever, camp fever, petechial fever, putrid fever, Irish ague, brain fever, spotted fever, continued fever, typhus fever, petechial* and *exanthematous typhus.*[1]

Morbid Anatomy.—Those pathological lesions which are common to typhus and typhoid fever will be first considered, and as the line of distinction between them is drawn it will be noticed that, in many respects, the difference is one of degree, rather than of kind.

The changes in the *blood* are as follows: it is darker in color than normal, and when drawn from the body during life coagulates imperfectly or not at all; if a clot is formed it is of the consistency of putty. The fibrin-factors are diminished, or the blood loses its coagulating power to a greater or less extent. At first the red globules are increased in number, but as the disease progresses they diminish; the salts of the blood are also changed, and urea and ammonia are present in excess; by some the latter is supposed to be produced by the decomposition of the former. The blood of a typhus fever patient, when drawn from the body, rapidly undergoes ammoniacal decomposition. When the blood is examined microscopically, many of the red globules will be seen to have lost their normal outline, and their edges to have become serrated and irregular. In some instances they will be found to have undergone degeneration; their coloring matter will then pass through the walls of the blood-vessels and stain more or less deeply the tissues and effusions which may have taken place in the serous cavities. These blood-changes are very similar to those which take place in the miasmatic-contagious fevers—they differ in degree only.

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[1] The Germans describe an abdominal and cerebral typhus. Their abdominal typhus corresponds to our typhoid fever and their cerebral typhus is our typhus fever.

Parenchymatous Degenerations.—There is the same tendency to parenchymatous degenerations of the different organs and tissues of the body in typhus as in typhoid. Usually the body is not very much emaciated; it undergoes decomposition rapidly after death. In severe cases decomposition apparently commences before death. The muscles are usually of a brownish color, dry, presenting an infiltration of fine granules in the primitive fibres; sometimes hemorrhages take place into them. The liver and spleen undergo degenerative changes similar to those described as occurring in typhoid, but they are not so extensive nor are they so constant. One may make very many autopsies on persons dying of typhus fever, without finding any softening, or only a very moderate softening, and enlargement of the spleen, while blood extravasations are not uncommon. In severe cases the cortical portion of the kidneys is swollen, opaque and more or less fatty, according to the duration and severity of the disease. The primary enlargement of the kidneys is mainly due to a cloudy swelling of the epithelium of the renal tubes.

This tendency to cloudy swelling and granular fatty degeneration, the so-called "vitreous degeneration of Zenker," which occurs in the voluntary muscles and the kidneys, also occurs in the muscular tissue of the heart. If the fever is protracted, the cardiac walls become flaccid, of a brownish color, and parenchymatous changes are found similar to those which occur in typhoid fever, though less marked. There is often a considerable amount of serum in the pericardium. Pultaceous clots are found in the heart cavities, and thrombi are found firmly adherent to the walls of the larger veins. There is the same tendency to ulceration of the mucous membrane of the mouth and larynx as in typhoid fever. In typhus fever the ulcers are deeper, involving more extensively the submucous tissue. Splenization of the lungs also occurs in typhus as in typhoid fever. Hypostatic congestion of the lungs and pulmonary œdema are as common as in typhoid; some claim that they are found much oftener. Thus far I have noticed only these lesions which occur both in typhus and in typhoid fever. I now come to those which are found only in typhus.

Brain.—Although there is nothing in the appearance of the brain which is characteristic of this fever, yet it is very unlike that met with in typhoid fever. In the latter disease it usually presents an anæmic appearance. In all cases of typhus the cerebral vessels will be found more or less congested. In some epidemics all the sinuses and blood-vessels of the brain will be found engorged with dark blood, so that when the calvarium is removed the vessels will stand out upon its surface. In other epidemics, instead of finding intense congestion, there will be more or less extensive serous effusion into the meshes of the pia mater: the quantity of the effusion varies from one to eight or ten ounces, and it is most abundant upon the convexity, although it takes place to a limited extent into the ventricles. Whenever there is a large amount of fluid effusion there will be little cerebral congestion. The fluid effusion is usually clear; if it is turbid one may be certain that the fever is complicated by meningitis. The arachnoid loses its natural glistening appearance, and in many instances one will find the membrane dotted over with yellow or yellowish-white spots. The brain undergoes little or no change unless the fluid effusion is abundant, when by its pressure the sulci are deepened and the convolutions are sharpened.

Abdominal Lesions.—In typhus and typhoid fever, the lesions found in the abdominal cavity widely differ. The real pathological distinction is in the presence or absence of intestinal changes. These are present in typhoid and absent in typhus. In typhus fever there are no changes which show a tendency to ulceration of the intestinal glands, except those which are produced by congestion, such as are frequently seen in scarlet fever and measles, where the Peyerian patches present the shaven-beard appearance; while in typhoid fever, either ulceration of the intestinal glands will be present, or the glands will present the appearance which just precedes ulceration. At the post-mortem examination, if ulceration of the agminated and solitary glands is found, one may be certain the patient died of typhoid fever. The presence or absence of intestinal changes settles the question as to whether the fever is typhus or typhoid.[1]

Complications.—Although the complications which occur in the course of typhus fever are in no way peculiar to it, yet they are of such frequent occurrence, and are developed during its active progress, and modify its phenomena to such a degree, that it is necessary that they should be taken into account in the study of its pathological lesions. These complications will vary according to the peculiar type of the epidemic which is prevailing. In one epidemic the complications will be pulmonary, in another they will be almost exclusively cerebral and spinal, in another nearly all will be glandular in character. The pulmonary complications are bronchitis, pneumonia, pleurisy, pulmonary congestion, and œdema. In most cases these pulmonary complications are developed during the primary fever, before convalescence commences. Their advent is always insidious. An extensive capillary bronchitis may develop with very few of the rational symptoms of bronchitis until within a short time previous to the death of the patient; in fact, the bronchitis might pass unrecognized but for the presence of its physical signs. All the rational symptoms of pneumonia may also be absent and still a physical examination of the chest may reveal a whole lung in a state of pneumonic consolidation. The pneumonia which complicates typhus is usually hypostatic. It sometimes leads to pulmonary gangrene. At most of the autopsies there will be found pulmonary congestion and œdema. In many cases when these are associated with capillary bronchitis or pneumonia they are the immediate cause of death. Laryngitis is often associated with more extensive bronchitis which occurs during the active part of the fever.

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[1] Lebert states that rarely, in epidemics, small ulcers of Peyer's patches and of solitary glands and swelling of the mesenteric glands have been seen. (Ziemssen's Encyc.)

The only cerebro-spinal complication which is met with in typhus fever is meningeal inflammation. As has been stated, in a large majority of autopsies of typhus fever serum is found in the meshes of the pia mater, but that is not a certain sign that meningeal inflammation has existed prior to death. In addition to the subarachnoid effusion, there must be an exudation of plastic material into the meshes of the pia mater, causing it to become thicker than normal. When such appearances are found it shows that the case has been complicated by meningitis.

Glandular Enlargements.—The glandular enlargements and inflammations which occur in the course of typhus fever are peculiar in their character, and are rarely met with in typhoid, and then are not extensive; but in typhus fever the superficial glands—especially those about the neck, the parotid and sublingual—often become so much enlarged and inflamed as to interfere with deglutition, and not infrequently these glandular enlargements are apparently the immediate cause of death.[1] The inguinal glands sometimes become so enlarged as to interfere with the return circulation, and, as the consequence of this interference, swelling of the lower extremities may be developed. The bronchial glands are nearly always enlarged and softened. There is a swelling of the lower extremities which depends upon a different cause. It may occur at the beginning of convalescence; then the limbs will present very nearly the same appearance as that noticeable in the condition called *phlegmasia dolens*. Under such circumstances phlebitis might be suspected.

It has been stated that the voluntary muscles undergo a peculiar waxy or vitreous degeneration, and that the same kind of degeneration occurs in the muscular tissue of the heart. When this does occur the walls of the heart become very flabby, and when this change has reached a certain point there is developed a tendency to the formation of clots in the heart cavities, and a slowing of the general circulation. The result of such retarding or obstruction of the return circulation is the formation of thrombi in the superficial veins, which interfere with the venous circulation, and a swelling of the lower extremities follows; this closely resembles that which is seen in *phlegmasia dolens*. With this swelling of the lower extremities, suppuration and cellular inflammation may occur, which often result in the formation of quite extensive abscesses.[2]

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[1] Lebert regards enlargement and suppuration of the parotid as a very dangerous complication.

[2] It is an established fact that whenever the return circulation is slowed from any cause in any disease where there is great feebleness of heart power, thrombi are liable to form in the veins of the lower extremities. This is often well illustrated in the later stages of phthisis, when swelling of one or both lower extremities occurs as the result of the formation of venous thrombi in the superficial veins.

Diseases of the organs of special sense, which so frequently complicate typhoid, rarely occur in typhus fever, and there are no serious or constant complications of the digestive organs; the *gastric mucous membrane* is sometimes softened, reddened, and mammilated.

Etiology.—At the present day this fever is regarded as depending upon a specific poison, of whose exact nature we are ignorant. From the manner of its development and its contagious properties it must be regarded as due to a specific microbe. All observers agree that in the majority if not in all instances it is the product of *contagion,* and that the contagion only emanates from the bodies of those who are affected with the fever. Careful clinical observation has established the fact beyond a doubt that there exists a specific typhus poison which can be communicated from the sick to the healthy. It is possible to develop a fever from overcrowding, imperfect ventilation, filth, and a combination of causes belonging to this category, but such a fever is not typhus.

The results of my investigation of the origin of the epidemic of typhus fever which prevailed in New York, from July, 1861 to 1864, have led me to the belief that typhus poison is of endemic origin—in other words, that there are certain endemic centres; that Ireland, Italy, and Russia are the great centres, and that, whenever it occurs in other localities, it has been conveyed from these endemic centres to those localities.[1] The histories of those cases which were developed within the limits of the hospital, showed that a residence in an atmosphere necessarily more or less tainted with typhus poison, is not sufficient to develop the disease, but that it is necessary for the subject of the contagion to have been brought in contact with an infected person, or within the atmosphere immediately impregnated with his exhalations. The fact that no employée in the hospital who was only brought in contact with the clothing of fever patients contracted the disease, as well as the absence of any evidence that the disease was propagated by such clothing, goes far to show that typhus is not readily propagated by fomites alone,[2] although most authorities claim that it *can* be thus propagated; that it is thus that ships, barracks and jails become hot beds of it; that the poison may be latent and held in garments, especially those that are dark and woolen. The certainty with which every unprotected person who was brought in personal contact with fever patients contracted the disease, proves the contagious power of the poison.

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[1] In the month of July, 1861, fourteen cases of typhus fever were admitted in one day to the fever wards of Bellevue Hospital, of which wards I had the charge. Previous to this time, for several years (I think for more than ten years), there had been no case of typhus fever in the wards of the hospital. I immediately commenced investigations in order to ascertain the origin of the fever in these cases. I found that it had its origin in the upper story of a rear tenement-house in Mulberry Street, in the most filthy portion of the city. The first case was that of a little girl, who had been brought into the house, ten days before she sickened, from a ship which had come from Ireland, and which had cases of typhus fever on board. Two weeks after her illness commenced, her aunt, the only other occupant of the apartments (consisting of a room and dark bed-room), sickened of fever and died. In gradual succession, nearly every family residing in the building took the fever. Becoming frightened, some of these families moved into other streets, formed the nucleus for the development of the disease in the different localities to which they removed, and it soon became a wide-spread epidemic. There were two hundred typhus fever patients at one time in the hospital. These families were as well nourished and lived in as well ventilated apartments as thousands of their class in other parts of the city. The only difference was that typhus poison was brought to them in the person of the little girl, and, on account of their badly ventilated apartments and their utter disregard of all hygienic laws, they furnished a fit soil for the reproduction and spread of that typhus poison, the constant and unrestrained intercourse between the healthy and the sick being the means by which the fever was spread. I found unmistakable evidence that persons living in healthy localities, simply by visiting friends sick with the fever, contracted the disease.

[2] In Quain's Dictionary it is stated that it is "not carried by clothing or excreta, and free dilution with fresh air destroys its virulence."

The distance that typhus poison can be transmitted through the atmosphere (from the manner in which the disease was contracted by some of the house physicians), would seem to be limited. It has been proved by actual experiment that the contagious distance of small-pox, in the open air, does not exceed two and one-half feet, and it would seem that the contagious distance of typhus fever is even less.[1] Typhus poison is undoubtedly present in the body exhalations and the expired air of typhus fever patients; but it requires a concentration of the poison to render it infectious. Slight exposure is not sufficient; it requires a concentrated poison and a prolonged exposure. The more numerous the typhus fever patients are, the more powerful does the contagion become; yet a single exposure even to such an atmosphere is rarely sufficient to develop the disease in an individual who is in good health at the time of the exposure.

The length of the period of incubation varies. It usually requires about two weeks of exposure such as comes to one who is around those sick with the fever. Repeatedly have I noticed this fact in my own case. I have never had typhus fever, and have never taken special care to avoid infection. My immunity is probably due to some special constitutional idiosyncrasy. I have noticed that whenever I enter upon a typhus fever service I do not experience any effects from the exposure to typhus poison until after about *two weeks*[2] have elapsed, then I begin to suffer from a peculiar form of headache which continues for about two weeks; the period before the commencement of the headache corresponds to the period of incubation, and the period of headache to the average duration of the disease.

The established belief is that typhus fever attacks an individual but once, and that those who have had typhoid fever are to a certain degree protected from typhus. Of all the typhus fever patients treated in Bellevue Hospital, only three gave histories of having previously had the disease. From these facts one may reach the following conclusions:—

First.—That typhus fever is due to a specific poison.

Second.—That this poison is communicated from the sick to the healthy mainly by personal contagion—that is, the recipient of the poison must be brought in contact with the exhalations of the infected person.

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[1] The question now arises:—can this poison be conveyed in the clothing? During the epidemic to which I have referred, when typhus fever patients were brought into the hospital, their clothing was removed in the reception-room and afterward washed and packed away in a lower room of the building. Upon a most thorough investigation made at that time, I found that not a single person contracted the disease whose duty it was to wash or pack away the clothing; but every one whose duty it was to carry the fever patients from the reception-room to the hospital ward took the fever. Every physician and nurse who had the care of typhus fever patients contracted the disease; those who were on the surgical service escaped. Every clergyman who came to administer spiritual consolation to the patients in the fever ward fell a victim to the disease. I have brought forward these facts to show that during this epidemic there was no evidence that the disease was either of spontaneous origin, or that it was transmitted from the sick to the healthy, except by direct personal contagion.

[2] Lebert puts five to seven days, and Murchison says, "no longer than twelve days" for the period of incubation; one week is the average: some patients have the fever one-half to two hours after first and second exposure (*St. Thom. Hos. Rep.*, vol. ii.).

Third.—That where there is free ventilation, contagion is confined to narrow limits.

Fourth.—That the evidences of the spontaneous origin of typhus are not conclusive, although there can be no question but that overcrowding and bad ventilation favor its spread and increase its severity.

Fifth.—Typhus poison passes into the body mainly through the respired air. Whether it can be taken into the system in the food and drink is still an unsettled question.

Sixth.—Immunity from a second attack is enjoyed after the first in a large majority of cases.[1]

Symptoms.—An outline of the phenomena which attend the development of typhus will first be given and afterward some of its more prominent symptoms will be considered in detail.

Its advent is usually sudden; there are no constant premonitory symptoms. In some cases, for a few days, there may be a feeling of indisposition, perhaps of headache, restlessness at night, nausea, loss of appetite, and vertigo; but in a large majority of cases it is ushered in by a distinct chill. This differs from the chill of pneumonia or that of malarial fever, in that it is short, sharp, and sudden. It may amount to nothing more than a chilly sensation. There may be several chilly sensations on the day of attack with distinct intervals between them. Following the chill there is a severe and steadily increasing headache; it is frontal and increases in intensity from hour to hour. This is accompanied by a more or less severe pain in the back and limbs, especially in the thighs. The headache of typhus is more constant and persistent than that which attends the development of any other fever; usually, after a few days it diminishes in intensity. Headache is associated with dulness and confusion of mind, and in the case of children with vomiting. A sense of extreme prostration very soon follows the ushering-in chill.

In some cases the patient is compelled, within twenty-four hours from the commencement of his sickness, to take to his bed from muscular weakness. This loss of muscular power will sometimes show itself by the unsteady, tottering gait of the patient, and is more marked in the early stage of typhus fever than it is in any other disease. At one time, while I was making my visit in the fever ward, my house physician, who was sickening from typhus fever, staggered and fell by my side from loss of muscular power. He died on the eighth day of the disease.

Within the first twenty-four hours after the chill the temperature may rise as high as 105° or 106° F., although at the same time the patient may complain of a chilly feeling, and will draw up to the fire or cover himself with blankets. It is a peculiarity of this fever that, during the first two or three days the patient experiences a sensation of coldness, while the thermometer shows the temperature to range at 105° F. or higher. During the first week of the disease the temperature remains at 104° F. or 105° F. There will be morning and evening variations, most marked at noon and midnight; but these variations follow no regular course, as in typhoid fever. From the eighth to the fourteenth day the temperature is liable to sudden depression. As a rule, the temperature falls between the eighth and fourteenth days. There is, without doubt, a day of crisis in this disease. Just before the critical fall in temperature there may be an abrupt temporary rise of 3° or even 4° F. In typical cases, before the fourteenth day there is a marked decline, and often a sudden fall in temperature. By the beginning of the second week the temperature ranges at its highest. If there is a sudden rise in temperature during the second week, it is almost certain evidence that some complication exists.

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[1] Lebert says: "all agree that the disease is spread by a typhus germ. Some say it is microspheres, others that it is bacteria, spiral forms, fungus, etc. It must be either organic poison or organized germ."

At first the tongue is swollen and covered with a white coating. It presents very much such an appearance as is seen in many nervous affections. As the disease progresses, after a day or two it assumes a yellowish-brown color, and the coating becomes thicker; later it becomes dry, dark and fissured. Nausea is sometimes present, rarely vomiting. The abdomen is free from pain, except over the liver; the bowels are constipated. Some enlargement of the spleen can usually be detected quite early.

The pulse is accelerated from the very beginning of the fever, ranging from 100 in the morning to 110 or 130 in the evening; the acceleration is greater in children than in adults. At the onset of the fever the pulse is full, but it soon becomes soft and compressible, and finally feeble. It is rarely dicrotic. It is only in the severest cases, just preceding death, that the pulse becomes irregular and intermitting. The face is flushed, the conjunctivæ injected, the expression of countenance is dull, heavy, and weary, and as the fever progresses, the cheeks assume a mahogany color. The sleep is disturbed, and when the patient is awake his mind is confused; in very severe cases delirium is very early present, and the patient needs careful watching at night.

Between the fifth and eighth, usually on the fifth, day of the disease, an eruption makes its appearance upon the surface. The skin is extremely hot, and there is no tendency to perspiration. It appears first upon the sides of the abdomen, and gradually extends over the whole anterior portion of the body, except the face and palms of the hands. In a few cases it first appears on the back of the hands and wrists. It is more marked upon the trunk than on the extremities. At first the eruption consists of dirty pink-colored spots, varying in size from a mere point to three or four lines in diameter. These spots are slightly elevated above the surface, and temporarily disappear on firm pressure. After a day or two the eruption becomes darker in color, and assumes a purplish hue. It is no longer elevated above the surface, does not entirely disappear on firm pressure, and the spots have no well-defined margin. This eruption is made up of irregular spots, varying from a point to two or three lines in diameter, either isolated or grouped together in patches, presenting a very irregular outline; in children it often resembles the eruption of measles. When the eruption is abundant it imparts to the skin a mottled aspect, which has given rise to the term "mulberry rash" of typhus. Another distinctive peculiarity is, that each spot or patch remains visible from its first appearance until convalescence is established or death occurs, and it is often seen upon the bodies of those who have died of typhus fever.

In some cases of typhus there are only a few spots of the eruption, while in other cases they are very abundant, and the surface of the body presents a well-marked mottled appearance. In a certain proportion of cases, after the eruption which I have just described has been visible for a few days, there will appear, scattered over the surface, small dark spots, due to minute subcutaneous hemorrhagic extravasation; these are called petechiæ. On this account the disease has been called *petechial typhus;* but these petechiæ are by no means distinctive of typhus, for they are also met with in other diseases. The majority of cases of typhus which one meets will have no eruption except the "mulberry rash." When the petechial spots are present they indicate a severe form of the disease, and more extensive blood-changes than usual. This mottling or marbling of the skin begins as the mulberry rash fades; it appears once for all—*not* in crops—and resembles slightly the rose-rash of typhoid fever. It is the subcutaneous eruption, so-called.

In all severe cases, at the close of the first week the headache, which has been the most troublesome symptom, disappears, and delirium comes on. The delirium will vary in character and severity in different epidemics, being much more violent and active in some than in others. Sometimes at the very outset of the disease the delirium is very active, the patient shouts and talks more or less incoherently, and is more or less violent. If not restrained, he may throw himself out of the window. This period of intense nervous excitement may last two or three days, during which the countenance becomes livid, the conjunctivæ injected, the hands tremulous, and suddenly the patient may pass into a state of apparent coma. It is not that of complete coma, for the patient can be easily aroused; but he lies upon his back, with a tendency to slip down in bed, picking at the bedclothes. The mental faculties, the special senses, are all blunted, and the patient is in a condition of stupor for three or four days preceding the delirious period, and sometimes, when the delirium is not active, this stupor lasts till the end of the disease. It is not a state of unconsciousness, although one of apparent coma, for the mental processes are going on with great activity, and the imagination will conjure up a great variety of horrid fancies, and the visions which pass before the patient will be distinctly remembered after recovery has taken place.

This condition has been called "*coma vigil.*" During this period the experience of years may be crowded into a day or an hour, and the patient may feel that he has lived a lifetime while in this state. Those who have the greatest mental power and possess the highest culture have the most distressing fancies during this somnolent period. If, in this condition, there is a tendency toward a fatal issue, the patient will pass into a more complete stupor and the coma will become more and more profound; the respiration becomes less and less frequent; the pulse, which has ranged about 120 per minute, rises to 140 or 150, and finally becomes imperceptible at the wrist; the tongue, rolled into a round mass, becomes brown and dry, so that the patient is unable to protrude it from the mouth; or, if he protrude it, he does not withdraw it until asked to close his mouth; sordes collect upon the teeth; the conjunctivæ are red, and the eyes, when open, present a leaden appearance. The face has a dusky pallor. The patient has no longer power to move his body; he lies on his back with his head thrown back, perhaps is only able to make slight tremulous motions with his hands. There may now be some intestinal catarrh, with diarrhœal discharges. The urine collects in the bladder, and, if not removed with a catheter, dribbles away. The extremities become cold, but the body temperature remains at 105° F., or it may rise as high as 107° or 108° F. In one case under my observation it rose to 110° F. just preceding death, while the extremities were cold.

If the case is tending to a favorable termination, about the tenth to the fourteenth day of the fever there is an amelioration of all the symptoms. The patient falls into a quiet sleep, from which he awakes conscious and convalescing. The pulse and temperature fall, the tongue becomes clean and moist, the delirium subsides, and there is a desire for food. After two or three days the pulse reaches its normal standard and strength gradually returns. Critical sweats, diarrhœa, and large flows of urine are not infrequent occurrences.

This is an outline of the progress of the disease in a severe case of typhus fever, terminating either in death or in recovery. In a mild case there will be no delirium. The temperature may not rise above 102° F.; the tongue is neither brown nor dry. There is no great acceleration of the pulse, the rate never being over 120 per minute, and that only for a very short period. During the entire course of a severe or mild case, there is no gastric or intestinal disturbance, no diarrhœa, no distention of the abdomen, no pain in the right iliac fossa, no gurgling—in a word, no abdominal symptoms. In mild cases the eruption is never very abundant, but it appears on the fifth day, and remains visible until convalescence is established.

Those more important symptoms which determine the character of the fever will be considered in detail. As has been already stated, symptoms indicating disturbance of the *nervous system* are among the earliest and most prominent. Of these, *headache* is the most constant. For the first week or ten days it is severe and persistent, after which time it gradually abates and disappears towards the close of the second week; it is confined to the forehead and temples, rarely to the occiput.

Delirium comes on usually about the eighth day; sometimes it is present at the onset of the disease. At whatever period it may be developed, it will continue until the termination of the disease. Delirium is preceded by a dull, apathetic state, which follows the abatement of the headache. At first it shows itself at intervals during the night, or lasts all night to disappear during the day. Its character varies from a low, muttering form to a very active and noisy delirium. Every possible variation is met with during an epidemic of typhus fever.[1] Acute delirium is more liable to be present with the intelligent and highly cultured, while the delirium is usually low and muttering in character in the case of the aged or uncultured: other things being equal the intensity of the fever can be measured by the kind and amount of delirium.

Stupor or somnolence in some degree is seldom absent. It may develop with or without previous delirium. Usually, as the case progresses toward a fatal termination stupor comes on; this becomes more and more profound as the disease advances. The patient often lies for hours apparently unconscious, with his eyes open as though awake, but he is absolutely indifferent to all that is going on around him. This is another condition to which the term "coma vigil" has been applied. It is almost invariably followed by a fatal termination. Sometimes coma comes on suddenly, without any antecedent somnolence; under such circumstances the urine will be found loaded with albumen.

Loss of muscular strength is an early and striking symptom. In the majority of cases it is present from the very first day of the fever. In many cases, as the fever progresses, the loss of muscular power is so great that the patient is unable to turn in bed; the prostration always increases as the disease advances. In some cases there is little loss of strength during the first week, but the prostration comes on suddenly during the second week.

In addition to the general loss of muscular power, in certain cases there is paralysis of some muscles, such as the sphincter ani and the muscles of the bladder, so that the urine and fæces are discharged involuntarily. Dysphagia, partial or complete aphonia, and inability to protrude the tongue, are common symptoms. Muscular tremor is an indication of very great muscular prostration, and is usually met with in the aged and infirm and in those who have been addicted to the use of intoxicating drinks. Muscular spasms and subsultus tendinum are present to a greater or less degree in all severe cases; the tendons of the wrist are most frequently affected. One form of these spasmodic movements is manifested by the patient's picking or fumbling the bed-clothes; another by obstinate hiccough. Trismus, strabismus, and in rare cases opisthotonos, have occurred. All these symptoms must be regarded as grave. Emaciation is never a marked symptom. It is rarely present to any great degree before the third week of the fever.[2]

Temperature.—During the first week of typhus fever there are no such marked typical variations in temperature as are met with in typhoid—none that will enable one to make a diagnosis. Usually the temperature rises rapidly from the very onset of the fever, and in cases of average severity attains its maximum from the third to the sixth day. At this period the evening temperature will range between 103° F. and 106° F. In severe cases, the maximum temperature is not reached until the eighth or tenth day. Before the temperature reaches its maximum, the morning and evening variations are slight, about 1° or 1½° F. After the temperature has reached its maximum for several days there will be little change, but at some time, usually between the seventh and tenth days, there will be a slight remission until the twelfth or fourteenth day, when it rapidly falls, in typical cases that terminate in recovery, to its normal standard. Any sudden rise or fall (except in crisis) indicates a complication. Occasionally an elevation of two or more degrees precedes the fall. This sudden fall about the fourteenth day is peculiar to typhus. The fall may amount to 4° or 5° F.

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[1] The incoherent, nonsensical muttering is beyond the control of the patient, who is himself aware of its disjointed and erratic character.

[2] The eyes at first are suffused; later the conjunctiva becomes dry. The pupils are contracted and are insensible to light in many cases. Vertigo, dizziness, noises in the head, partial and even complete deafness are observed. Coryza, epistaxis, slight hyperæsthesia, and, finally, general anæsthesia, are infrequent symptoms.

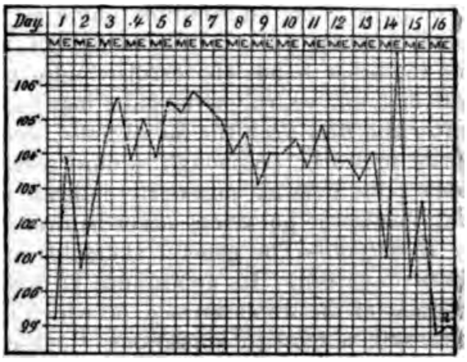

FIG. 168.

Temperature Record in a Case of Severe Typhus Fever, ending in Recovery.

A very high range of temperature during the first week is an indication that severe cerebral symptoms will be developed during the second week of the fever. A case of typhus fever may terminate fatally, in which the temperature at no time has exceeded 103° F. In all fatal cases, just *preceding death* there is usually a rise of from 2° to 5° in temperature. During the first week of convalescence the temperature often remains below the normal standard, especially in the morning.

Pulse.—The pulse in this fever is usually frequent, soft, easily compressed, and often irregular. The heart may partake of the general muscular weakness, so that the first sound may become inaudible. There is a soft systolic ("fever") murmur heard over the heart. In the severe cases, during the first week the pulse may reach 120 beats per minute, after which time it increases in frequency and feebleness with the severity of the general symptoms. By the third day it may reach 120 beats per minute; usually in the milder case it does not exceed on that day 100 beats per minute. If during the first week it continues for three consecutive days so frequent as 120 beats per minute, it indicates danger. The rate may be 106 in the morning and 120 in the evening. The higher the temperature, and the more frequent the pulse during the first week, the more severe will be the symptoms during the second week. If during the second week it becomes small, feeble and frequent, perhaps beating 140 or 150 per minute, the case may be regarded as unfavorable. *Absence of pulsation* in the radial artery for several days has been observed, and is explained on the ground of embolic obstruction of the medium-sized vessels.

During the first week, if the pulse increases in frequency the temperature rises, and if the pulse diminishes in frequency the temperature falls; but during the second week the pulse may increase in frequency, and the temperature fall, or it may diminish in frequency and the temperature rise. The pulse is not an infallible guide as to the condition of the heart, for sometimes the pulse is full and distinct, while the heart power is very feeble; on the other hand, the cardiac pulse may appear strong and the sounds distinct, and yet the radial pulse may be imperceptible. In most fatal cases, after the first week the radial pulse becomes imperceptible for several days prior to death. Although in most severe cases there is a rapid pulse, yet a slow pulse does not necessarily indicate a mild attack. In some severe and fatal cases the pulse may never be over 100.

Eruption.—The general character of the typhus eruption has already been described. Its appearance is preceded and accompanied by a bright redness of the whole surface, on which dark red spots are scattered, giving the skin a mottled appearance "sub-cuticular rash." These spots have an irregular outline, and vary in size from a point to three or four lines in diameter. Sometimes they are few in number, but more commonly they are numerous; the large spots are formed by the coalescence of the smaller ones. It is a macular, not a papular eruption. At first they have a dusky pink hue, partially or wholly disappearing on pressure, and as the finger passes over them they seem to be slightly elevated. After a day or two they assume somewhat of a brick-dust color, and are but slightly changed by pressure; then the color of the spots becomes darker in hue, and finally they are not affected by firm pressure. Another peculiarity is that each patch or cluster remains visible from its first appearance until the termination of the disease. The eruption may appear upon any portion of the body. Usually, it first makes its appearance upon the trunk, soon spreading to the extremities; very rarely is it seen on the face. When the eruption is scanty, it is limited to the chest and abdomen. In some patients the eruption, though well developed, is not prominently marked; the spots are pale and undefined; and though grouped in patches are so irregular that they give to the entire surface a faint, dingy appearance. Blood-extravasations into the centre of the typhus spots may occur at the end of the second week.

Respiration.—Usually, during the first week the respirations do not exceed twenty or thirty per minute; but during the second week they often run up to forty or fifty per minute. In cases where there is great prostration accompanied by stupor, the respirations sometimes fall to eight or ten per minute. Under such circumstances they are often irregular and puffing in character. Hypostatic congestion of the lungs, if extensive, is attended by great frequency of respiration and evidences of cyanosis. The occurrence of these changes in respiration ought always to lead to careful examination of the chest.

The digestive system is very little disturbed in typhus fever. Nausea and vomiting are rare, and an examination of the abdomen presents nothing abnormal. There is no tympanitis or tenderness on pressure. Spontaneous diarrhœa is of exceedingly rare occurrence; the bowels are generally constipated. Intestinal hemorrhage is of rare occurrence, and when it is present depends either upon congestion of the mucous membrane of the colon or on hemorrhoids, which accompany an engorged portal circulation.

Urine.—The urine in typhus undergoes important changes. The quantity varies somewhat with the amount of fluid taken into the stomach; usually, it is diminished during the first week to one-fourth or one-half the normal quantity. In the advanced stage of severe cases there is sometimes complete suppression of urine, but more frequently the quantity of urine increases during the later stages of the fever. The reaction is first acid, later neutral or alkaline. The quantity of urea excreted in twenty-four hours during the first few days of the fever is increased, and the increase is in proportion to the intensity of the fever. In the majority of cases it remains abnormally increased until the period of crisis is reached, when it gradually, or in some instances rapidly falls below the normal standard. Uric acid is similarly increased. The chlorides grow less and less till the second week, when they disappear. In all severe cases, during the first week of the disease, a small amount of albumen is found in the urine; when the quantity is large the case may be regarded as severe. In the severe cases the urine will also be found to contain vesical and renal epithelium, and when the quantity of albumen is large, epithelial and fatty casts of the uriniferous tubes will be present with blood.

In this connection it is important to bear in mind the necessity of daily inquiry into the expulsive power of the bladder. When there is little cerebral disturbance, the urine is passed without difficulty; but when stupor and a tendency to coma exist, there is often retention or an involuntary dribbling of urine, which might lead one to think that there was no accumulation of urine in the bladder. It is safe to inquire at least once a day as to the state of this organ, and if involuntary discharges of urine occur, the contents of the bladder should be evacuated by means of a catheter. Copious sediments form in the urine on the day of crisis.

Differential Diagnosis.—Before the appearance of the eruption, the diagnosis of typhus fever is always difficult, and sometimes impossible. The diseases with which it is most liable to be confounded are *typhoid fever, relapsing fever, measles, pneumonia, acute Bright's disease, meningitis, delirium tremens,* and some of the other acute blood diseases, such as *erysipelas, pyæmia, septicæmia,* etc.

The early characteristic symptoms of typhus fever are chilliness, pain in the back and limbs, and headache. During the first week the headache increases in severity from hour to hour, and is accompanied by a rapid rise in temperature. These symptoms occurring in one who has been exposed to typhus poison are always sufficient for a diagnosis. The appearance of the eruption settles the question. On account of the similarity in appearance of the eruption of typhus fever and that of measles in children, the one disease is sometimes mistaken for the other. In both diseases the eruption may appear on the fifth day, but the eruption of *measles* is of a brighter tint than that of typhus fever, and its appearance is preceded by a cough and coryza, which are not present in typhus fever.

Meningitis.—The differential diagnosis between typhus fever and cerebro-spinal meningitis is often difficult. Not infrequently, days may elapse before one is able to decide whether a case is one of typhus fever or of cerebro-spinal meningitis.[1] The distinguishing points between the two diseases are as follows:—the headache of meningitis, at the outset of the disease, is much more distressing than that of typhus, and it alternates with delirium. When delirium comes on in typhus fever, the pain in the head *ceases*. Vomiting is prominent in meningitis, absent, as a rule, in typhus. Photophobia and contracted pupils are among the early symptoms of meningitis, and the patient is greatly disturbed by noise, while in typhus fever he seems indifferent to both. Dulling and blunting of all the senses are common in typhus. All the senses are abnormally acute at the onset of meningitis. Inequality of the pupils, strabismus, ptosis, and paralysis are common in meningitis and rare in typhus. In meningitis the countenance is pale and expressive of pain, wildness, and anxiety; in typhus fever it is dusky, blank, and stupid. Convulsions are an early symptom in meningitis and rare in typhus. Again, in meningitis the pulse is hard and wiry, rapid and irregular, and at the last intermitting; while in typhus fever it is rapid at the onset of the disease, easily compressed, full and bounding. Lastly, the eruption of typhus fever is characteristic. If an eruption is present in meningitis, it has no regularity in its development; it may appear within twenty-four hours after the development of the first symptom of the disease, or it may be postponed for several days. It does not appear on the fifth or sixth day of the disease with the uniform regularity of the typhus eruption. Petechiæ may be present in meningitis as well as in typhus fever, but they are not characteristic of either disease. The temperature rises rapidly in typhus, and reaches a higher range, *e. g.*, 104° to 106° in twenty-four to forty-eight hours; while in meningitis the average temperature on the second and third days—indeed, during its entire course—is 102°, often *lower*. Rigidity of the muscles of the neck is not always positive evidence of meningitis, for it sometimes occurs in typhus fever. The ataxia, muscular prostration, and character of the tongue in typhus are also points that greatly aid in distinguishing it from cerebro-spinal fever.

Pneumonia.—Sometimes a latent pneumonia with typhoid symptoms is mistaken for typhus fever; especially is this the case when the latter is prevailing. I frequently saw cases where such a mistake had been made, while in charge of the typhus fever patients on Blackwell's Island, during the epidemic to which reference has been made. In these cases there will be active typhoid symptoms, *e. g.*, dry tongue, delirium, high temperature, etc. The countenance in this pneumonia, although the cheeks may have a purplish hue, does not exhibit that dull, heavy expression so commonly seen in typhus. Although there may be delirium in both instances, the delirium in the former disease is of a milder type than the latter. The characteristic pneumonic expectoration is not usually present in these cases; so that in making the differential diagnosis this symptom cannot be relied upon. The physical signs of pulmonic consolidation will lead one to diagnosticate pneumonia, and unless the typhus eruption is present, this will be sufficient for a diagnosis. If pulmonary consolidation is a complication of typhus fever, it will not be developed until after the sixth day of the fever, the time when the eruption should have appeared. If no eruption is present, the pneumonic consolidation may be regarded as the primary affection, and the symptoms which simulated those of typhus fever may be regarded as secondary.

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[1] To show how difficult is the diagnosis between these two affections, a circumstance may be mentioned which occurred in Bellevue Hospital. A patient was brought into the hospital directly from a ship, and the diagnosis of cerebro-spinal meningitis was made by several of the attending staff; but at the autopsy there were found none of the lesions of meningitis; all the changes corresponded to those found at the autopsies of patients dying of typhus fever.

Delirium Tremens.—The delirium of alcoholism may sometimes so closely resemble that of typhus fever that the one may be mistaken for the other. Typhus fever patients have been placed in the cells under the supposition that they had delirium tremens. If the delirium tremens is uncomplicated by pneumonia, the temperature will suffice for the differential diagnosis, for in delirium tremens the temperature is rarely above 100° F. There may be a rapid pulse in delirium tremens, and often the patient has a brown, dry tongue, and other typhoid symptoms; but there is only a slight rise in temperature; besides, *there is no eruption.* The attack is not ushered in by headache, but by an inability to sleep; and the circumstances which precede and give rise to such an attack will establish beyond a doubt the true nature of the attack.

Acute Bright's Disease.—It is not surprising that acute uræmia should be mistaken for typhus fever. The brown, dry tongue, the tendency to stupor, the contracted pupil, the low, muttering delirium, and all the phenomena of the typhoid state, as well as the albuminous urine, belong to both diseases; but the temperature is not raised in uræmia as it is in typhus fever, and the œdema which is always present in acute uræmia is absent in typhus fever.

Erysipelas, pyæmia, septicæmia, and all similar *acute blood diseases* are often attended by many of the symptoms which attend the development of typhus fever. In pyæmia and septicæmia there are irregular chills, followed by fever and profuse sweats, with evidences of septic and pyæmic poisoning; in erysipelas, there are evidences of a localized phlegmon. It should not be forgotten that erysipelas is sometimes ushered in by all the phenomena that attend the ushering-in of typhus fever; this is before the local inflammation shows itself. In such cases it is impossible to make a differential diagnosis until the local phenomena which characterize erysipelas show themselves, or until the typhus eruption appears. In many of the acute infectious diseases one is compelled to wait until the time for the appearance of the eruption before typhus fever can be excluded. When typhus fever is prevailing and the physician is watchful for its appearance, there will usually be little difficulty in diagnosis. Sometimes typhoid, typhus and relapsing fever prevail at the same time in the same locality.

The importance of early forming a correct differential diagnosis between typhus and *typhoid fever* cannot be over-estimated; and to accomplish this, the prominent symptoms of each will be reviewed and compared. The *first* point to be considered in the differential diagnosis of these two diseases is, that typhus fever is sudden in its advent, while typhoid fever comes on insidiously, and is slowly developed. In the majority of cases of the former disease there is a chill at the commencement, and severe pain in the head, whereas in the latter there is only a chilliness, some aching in the limbs, and a slight headache. Muscular prostration and progressive muscular weakness appear earlier, and are much more marked in typhus than in typhoid.

The ranges of temperature in the two forms of fever differ greatly. Typhoid fever commences, on the first day, with a slight rise of temperature, which continues, with morning remissions and evening exacerbations, until the end of the first week, when it has reached its highest point; during the second week it remains at about the same height, with only slight variations; during the third week there are more marked morning remissions; and by the end of the fourth week the temperature has reached its normal standard, by intermittent periods. In typhus fever, the temperature rises rapidly, and before *the end of the second day reaches* 104° F. or 105° F. Whatever degree is reached on the third day may be regarded as the maximum temperature; after this time there are slight, irregular variations until the tenth or twelfth day, when the temperature begins to fall, and rapidly reaches the normal standard. In typhoid there is great emaciation; in typhus it is slight, but the exhaustion and muscular prostration are far greater than in typhoid. The eruptions of these two forms of fever differ very markedly. In typhus it makes its appearance upon the fifth or sixth day; while the eruption of typhoid fever makes its appearance between the seventh and ninth days. The eruption of typhus fever appears upon the arms and chest, and more or less over the entire body; whereas the eruption of typhoid appears upon the chest and abdomen, very rarely upon the extremities; sometimes it appears upon the loins when it cannot be found on any other part of the body. As a rule, the spots in typhus are numerous, while in typhoid they are not very abundant. In typhus fever, the spots at first are small, slightly elevated, of a dark pinkish hue, and disappear only on firm pressure. As the disease advances they become darker, and finally *are not* affected by firm pressure, and remain visible from the time of their appearance until death occurs or convalescence is established. In typhoid fever each spot is rose-colored, slightly elevated, and disappears on slight pressure. Each spot remains visible for three days and then disappears, to be followed by another crop. Usually the eruption is visible about two weeks, and when it disappears it leaves the skin unstained, whereas in typhus the eruption leaves a stain upon the surface. There is a mottling of the surface in typhus fever, the *mulberry rash*, which is not seen in typhoid.

The brain symptoms in these two diseases also differ. In typhus fever they appear early, and the headache and delirium are more intense than in typhoid. Delirium in typhoid more commonly appears at the end of the second or during the third week of the disease; whereas in typhus it appears early, and before the end of the second week has disappeared if recovery is to take place. As a rule, in typhus fever constipation is present, and a mild cathartic must be given to move the bowels; whereas in typhoid fever the pea-soup diarrhœa is a prominent symptom. Tympanitic distention of the abdomen, gurgling and tenderness in the right iliac fossa, and intestinal hemorrhage, are all phenomena of typhoid fever, but are never present in typhus fever. Epistaxis is frequent in typhoid and not in typhus fever. In typhus fever convalescence will usually be established before the end of the second week; it may occur at any time between the eighth and fourteenth days. The average duration of typhus fever is fourteen days, whereas the average duration of typhoid fever is from twenty-one to thirty days. Typhus fever is contagious, typhoid fever is non-contagious.[1] Typhus fever is generally epidemic; typhoid fever is always endemic. In regard to the protection which one attack of typhus fever furnishes against a second attack, it very markedly differs from typhoid fever. One may have typhoid fever whenever the system has been exposed to the typhoid poison; but one attack of typhus is almost a certain protection against a second attack.

Prognosis.—The prognosis in this disease is always grave, and no opinion, as to its termination, can be given until every point in each case has been considered, such as the age of the patient, the character of the epidemic, and the tendency to certain complications. In all epidemics the majority of cases will recover. The ratio of mortality, as given by different writers, varies from one death in five to one death in sixteen cases.[2] The surroundings of each patient should be carefully noted, also the hygienic influences which he is under, and his habits of life should be taken into account. With the intemperate the disease is likely to prove fatal. Some of the circumstances which increase the danger in any particular case are a debilitated condition of the patient from advanced age, intemperate habits, privation, and previous disease; mental depression, presentiment of death, overcrowding and bad ventilation; a gouty diathesis is always dangerous.

Death may occur in typhus fever from three general causes: *first*, from coma, the result of overwhelming the system with typhus poison. The patient does not die from the effect of a prolonged high temperature, nor from any complication, but dies as patients die in acute uræmia, because the system is overwhelmed by the typhus poison, and the functions of organic life are arrested by its action on the nerve-centres.

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[1] When the pathological lesions are studied and the manner in which death occurs in these two forms of fever is considered, it can readily be seen how widely they differ. The characteristic pathological lesions of typhoid fever are the changes which take place in the intestinal glands, such as ulceration or tendency to ulceration. In all cases these characteristic lesions are present. Suppose a case of what has been called typhoid fever is followed to the dead-house, and ulceration or evidences of a tendency to ulceration of Peyer's patches are not discovered, then it is certain a mistake in diagnosis has been made. If, on the other hand, in a case of supposed typhus fever is found ulceration of Peyer's patches, it is equally certain that a mistake has been made, and that a case of typhoid, and not typhus fever has been treated. The parenchymatous changes which are common to both diseases have already been sufficiently considered.

[2] Griesinger and Murchison state "that in certain *epidemics* the mortality runs as high as 40 to 50 per cent." An average epidemic shows about 15 per cent. of deaths; a mild one, about 6 to 8 per cent.

Death may occur, *secondly*, from *syncope*, due to heart failure, whether the heart failure is the result of the prolonged high temperature, or the direct action of the typhus poison. A continued temperature of 105° or 106° F. is very liable to be followed by fatal syncope from failure of heart power, although the evidences of parenchymatous degeneration of the heart may not be present. Death may occur, *thirdly*, from complications. Although they do not properly belong to the primary disease, yet they so modify it that they enter very largely into its history. In a large number of cases which terminate fatally, death is due to some one of these complications. In some epidemics they are all pulmonary; in others they are all cerebral. The advent of pulmonary complications is always insidious; the cough and expectoration which usually attend pulmonary diseases are either absent, or so slight as not to attract the attention of the physician. Rapid breathing and lividity of the face are often the first obvious indications of extensive disease of the lungs. When these symptoms are present, a careful physical examination of the chest should be made.

Bronchitis may come on at any period during the fever, and it may continue after the fever has subsided. So long as it is confined to the larger tubes there is little danger, but sometimes suddenly and insidiously it extends into the smaller tubes, and is complicated with pulmonary congestion and œdema. Under such circumstances it may be the direct cause of death. The pneumonia which complicates typhus fever is *lobular* in character, and is frequently preceded or accompanied by bronchitis. It has a tendency to terminate in abscess or gangrene. During life it is not always possible to distinguish it from hypostatic congestion. If, however, the dulness on percussion is confined to one lung, if the respiration is bronchial, the diagnosis of pneumonia is readily established. The seat of pneumonia is generally at the upper portion of the lung. Pleurisy (serous or purulent) may occur.

Laryngitis is sometimes a very serious complication of typhus. The common form is that of acute œdema glottidis. Its occurrence is readily recognized by the signs of laryngeal obstruction which attend its development. Whenever there is extensive swelling of the glands about the neck, with great tumefaction of the mucous membrane of the pharynx, one must be on his guard for the occurrence of this complication.

On account of the extensive blood-changes which sometimes occur in severe cases of typhus fever, the blood readily escapes through the walls of the vessels, giving rise to extensive hemorrhages from the mucous surfaces, nose, gums, bowels, the genito-urinary tract, vagina, etc., and into the cellular tissue. The occurrence of the hemorrhages is peculiar to certain epidemics, and when they occur it is during the first week of the fever.

Meningitis is probably the only cerebral complication which will be met with in this fever. This occurs more frequently in children than in adults, and is not present in every epidemic. The cerebral symptoms which are such constant attendants upon typhus fever do not depend upon meningeal inflammation; they belong to the natural history of the disease. If, during the course of the fever, there is a deep-seated pain in the head, with restlessness, which shows itself by constant attempts to get out of bed, with photophobia, contracted pupils, and flushing of the face and eyes, followed by somnolence gradually lapsing into coma, it is almost certain that meningitis is occurring as a complication. This is most liable to occur during the second week of the fever. The characteristic symptom which marks its development is the constant attempt on the part of the patient to get out of bed. He is so persistent in this that unless watched with the greatest care he will be found upon the floor, vainly attempting to rise. The patient has more muscular power than before the occurrence of the meningeal complication, for he will perform acts which previously he was wholly unable to execute. Usually the delirium lasts two days, then the patient gradually passes into a state of coma from which he cannot be aroused; his respirations may not be more than eight or ten per minute. Dilatation of the pupils, and an intermitting and almost imperceptible pulse, immediately precede death.

I regard most of the *kidney* changes as a part of the history of the fever rather than as complications, although in some few instances croupous nephritis occurs, and must be included in the list of complications. Its occurrence in the course of typhus fever is indicated by the almost entire suppression of urine, and by the presence of albumen and exudative and blood casts in the urine.

Glandular swellings are also occasional complications of typhus fever, and sometimes may be of a very serious nature, for they may so interfere with deglutition and respiration as to destroy life. The parotid, the submaxillary, axillary and mammary glands may enlarge and suppurate. These swellings usually appear immediately after the crisis of the primary fever. They often enlarge with great rapidity, and in some instances terminate in extensive suppuration.

Bed-sores are rather infrequent. Gangrene, necrosis, cancrum oris, suppurative cellulitis, purulent arteritis—all have occurred in various epidemics, and render the prognosis unfavorable. If menstruation occur in a female with typhus, the bleeding is commonly very profuse and may cause death from acute anæmia.

Duration.—The average duration of the fever is thirteen or fourteen days. Usually the day of crisis is between the tenth and sixteenth days. It is of shorter duration in the young than in the old, in children than in adults.[1] Relapses are extremely rare in this fever. I have met with a second and third attack of the fever in the same individual, but I have never met with a true relapse. Typhus fever varies very slightly in its general character and different cases. A number of different varieties, depending upon the mildness or severity of the disease, the prominence of certain symptoms, the presence of complications, and the circumstances under which fever appears, have been described, but the general description already given includes that of (so-called) "varieties" of typhus.[1]

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[1] In 500 cases ending in recovery, thirteen and a half days was the average; and in 100 fatal cases, the duration was fourteen and a half days.—Murchison.

The individual symptoms and signs which render the prognosis unfavorable are as follows:

A *pulse* continuing a number of days at more than 120 per minute, becoming at times intermittent and irregular, bespeaks an unfavorable prognosis. A hurried and difficult respiration, with turgidity of the face, due either to cerebral or pulmonary œdema, renders the prognosis unfavorable.

Delirium which is very active and accompanied by great muscular prostration, as indicated by subsultus, slipping down in bed, and accompanied by that condition known as "coma vigil," lasting for a number of days, is almost a certain indication of a fatal termination.

The "*pin-hole pupil*," mentioned by old writers, is an unfavorable omen. It does not necessarily indicate the presence of meningitis, as was once supposed. Great muscular prostration at the very onset of the disease renders the prognosis unfavorable.

Sudden fading of the eruption, and a widely expanded pupil may be regarded as unfavorable signs. Marked impairment of the special senses, accompanied by very great rapidity of the pulse, is an element of unfavorable prognosis.

The darker and more abundant the eruption, especially if accompanied by petechial spots, the more unfavorable the prognosis. In children the eruption is lighter in color than it is in adults, presenting an appearance similar to the typhoid eruption. In adults where there is dark mottling of the surface confined to the extremities, with evidences of blood extravasation, indicated by the presence of petechiæ, the prognosis is unfavorable, but the case is by no means hopeless.

A dry, brown, retracted, tremulous tongue is seen only in severe cases. A long-continued high temperature is always an unfavorable symptom. Great diminution in the quantity of urine is an unfavorable symptom, as also is the presence of casts and albumen in the urine. Retention of urine is a more unfavorable symptom than incontinence; convulsions and coma are liable to follow such retention. It is to be remembered that in typhus fever, more than in any other disease, patients pass into an apparently hopeless condition, and afterward rally and recover. A patient who seems to be overwhelmed with the poison, who has "coma vigil," "pin-hole pupils," rolling of the tongue, and a feeble, irregular, and intermitting pulse, may recover, although these symptoms warrant an unfavorable prognosis; but "coma vigil" more than any single symptom indicates an unfavorable prognosis.

The *first indication of recovery* is a diminution in the frequency of the pulse. The pulse may have been 120, but on the tenth, twelfth, or fourteenth day it begins to diminish in frequency. The tongue has been brown and dry, subsultus and delirium may have been present, even "coma vigil" may have manifested itself; there has been great muscular prostration; the patient, attempting to rise from the bed, may have fallen upon the floor; now, the pulse begins to get slower, the patient falls into a refreshing sleep and awakes perfectly conscious; his countenance is changed from the dusky hue to an almost natural appearance, and he desires food. In other words, within twenty-four hours an entire change comes over the patient, and that change is first indicated by a diminution in the frequency of the pulse, accompanied by a fall in temperature. The fall in temperature is not extrême; perhaps a fall of two degrees is first noticed. In my experience, there is an attempt at convalescence upon the eighth day of the fever. Especially in those cases that recover, a slight fall in temperature will be noticed on this (the eighth day), although the temperature may again rise; upon the twelfth or fourteenth day there is a distinct fall in temperature and diminution in the frequency of the pulse that is indicative of convalescence. The mode of recovery in typhus and typhoid is, perhaps, the most distinguishing clinical feature. In typhus recovery is rapid, in typhoid it is markedly slow.

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[1] *Typhus siderans* is that form where death occurs in three or four days, after the most intense febrile movement and constitutional disturbance. Headaches and a feeling of malaise, etc., during an epidemic give rise to what many call "*abortive*" typhus. *Walking typhus* is that form where the patient is not confined to his bed until the second week.

Of all the conditions which influence the prognosis in typhus fever, *age* and *the habits of the patient* have as great, if not greater, influence than any other. I am convinced of this from an experience in the care of typhus fever patients which dates back almost to the very commencement of my study of medicine, for very early did I have the care of a typhus fever ward. In children, typhus fever is a very simple form of disease. The rate of mortality is very low. I remember having the care of sixty children with typhus fever, and among these only one death occurred. This is as low a rate of mortality as one can expect in measles. Under the fifteenth year of life the rate is very low, viz., two or three per cent. From twenty to thirty the rate is fifteen per cent., with advancing years the disease is more fatal. When the patient has passed the middle period of life, there is great danger from typhus fever. So with the intemperate, and those who have livid amid unfavorable hygienic surroundings. The bright, educated and cultured, those whose brains are active, are less likely to recover than the stupid and uneducated.

Treatment.—The more prominent measures which have been and are now employed in the management of typhus fever are in many respects similar to those proposed for the management of typhoid fever patients, yet the treatment of these two diseases differs in certain essential particulars. When the symptoms are mild, very simple measures are all that is required. Of these, confinement to bed, cooling drinks, mild aperients, a milk diet, and free ventilation are the chief, and, indeed, the only means required. It is also important to observe the same rules in regard to the arrangement of the sick-room which are recommended in the case of typhoid fever patients. The more perfect the ventilation, the greater the amount of fresh air around the patient, the better his chances for recovery. The majority of cases of typhus fever are ushered in by active and severe symptoms, such as would tempt one to adopt a vigorous plan of treatment, symptoms which at one time were thought to indicate the employment of heroic antiphlogistic measures.

Writers usually consider its treatment under two heads—the preventive and curative. I prefer to use the terms prophylactic and remedial, for I question our ability to *cure* this disease.

Much can be done to prevent its development, and this will constitute an important part in its management. How, then, can the development of typhus fever be prevented? Medical skill cannot prevent the importation of the disease into localities where it is not indigenous, for this is controlled by state and national authority. Consequently typhus fever will probably continue to be imported into districts where it does not originate. For example, we shall occasionally see the disease in all our large cities; it may appear in any commercial seaport, and from there it may be carried into the interior. Yet much can be done to prevent its spread after it is imported, and to prevent its development as an epidemic when it is carried into any locality in the interior.

It is important that the first cases of typhus fever which are developed in any locality should be closely watched. They should be immediately quarantined. The dwelling in which the fever has broken out should be depopulated—that is, in a tenement-house in which the fever has made its appearance, all the families should be removed, and the house should be thoroughly disinfected. The disinfection must be thorough, not for a few hours, but for one or two days, and afterward the house should remain open for the free circulation of air for a considerable length of time before persons should be allowed again to inhabit the rooms. If typhus fever occur in the dwellings of the wealthy, their houses must be quarantined. All persons must be prevented from visiting them, and all persons within the dwelling must be prevented from going abroad. After the sick have recovered, there must be the same thorough disinfection as in the tenement-house. Usually, in epidemics of typhus fever there are certain foci from which the disease spreads. Perhaps the points from which the contagion more especially emanates are within an area of half a square mile, and yet the disease may have been prevailing for two, three, or even four months. Under such circumstances it is possible to prevent the spread of the fever by the means just indicated.

As far as its management in hospitals is concerned, I would say: never undertake it within brick or stone enclosures. If possible, patients should be placed in broad pavilions or tents, so that the largest possible amount of fresh air shall be in circulation about them. It is not sufficient to have free ventilation in the ordinary acceptation of that term. The opening of a window will not accomplish the desired result. Remove all the windows in a room, regardless of the cold, and cover the patients with a sufficient number of blankets to keep them warm. Allow fresh air to surround them. When typhus fever manifests itself, it can readily be understood how important it is that the guardians of the poor should not only enforce cleanliness, but that they should feed the poor better than at other times. If cleanliness is observed, the dwellings thoroughly disinfected, and the poor well fed, the most virulent epidemic can soon be stayed. The effects produced by such measures are sometimes wonderful. In the year 1861, at the commencement of the epidemic (as has been stated), the first case occurred in a tenement-house in a down-town street, in New York City; it was six weeks before it spread from that locality. The spread of the fever should have been stopped at that point; but very little attention was paid to it, and it began to spread from one point to another, until some six or seven thousand cases were developed. Many prominent citizens sickened with the fever and died. This epidemic could have been prevented had measures been taken early to prevent the spread of the disease. It seemed to me that the authorities of New York City were responsible for a large proportion of the deaths which occurred during the prevalence of that epidemic.

Medicinal treatment is powerless either to arrest the progress or shorten the duration of this fever, but it can undoubtedly save lives that would otherwise be lost, and hasten convalescence. The first point under this head relates to neutralizing the poison. I have found no medicinal agent which can neutralize or destroy typhus poison, or which has power to arrest the progress or shorten the duration of this fever. Different agents have been proposed for the accomplishment of this result, according to the views held in regard to the nature of the typhus poison and its effects upon the system. At one time the mineral acids were supposed to possess this power, and were administered for that purpose, but have now fallen into disuse.[1] The internal use of carbolic acid, chlorine water, creasote, and, more recently, salicylic acid has been recommended for the same purpose. The inhalation of oxygen gas has also been thought to be of service in arresting the blood-changes, and thus preventing the poison from having its customary effect upon the system. By the stimulation which it produces, the patient may be brought out of an apparent state of coma, and revive in a marked degree; but the relief is only temporary. For a time the patient may improve, his consciousness return, and his appearance indicate that convalescence is established; but his unfavorable symptoms will return, and it will become quite evident that the oxygen has not neutralized the typhus poison.

Fresh air is the only thing which I have found to have power to neutralize the poison of typhus fever. It certainly possesses this power when external to the body. For example: place a patient sick with typhus fever in a well ventilated board pavilion, or in a tent, where an abundance of fresh air can circulate about him, and it is almost impossible for him to communicate the disease to a healthy person. Again, place a patient in a closed room, perhaps twelve by fourteen feet square, let a healthy person remain with him a single night—probably a much shorter time will be sufficient—and the latter will be almost certain to contract the disease. Why is the disease more readily communicated in the one case than in the other? Certainly the fresh air which circulated about the typhus fever patient must have prevented contagion. Fresh air, when inhaled, produces to a greater or less extent the same effect. How do we know this? As a clinical fact, I have seen a typhus fever patient, who was apparently overwhelmed by the poison—who within forty-eight hours from the commencement of the attack was in a state of coma, with high temperature, a rapid pulse, etc., and all symptoms indicating that he was fast succumbing to the disease—when brought from a crowded tenement house and placed in a tent where he could inhale plenty of fresh air, within four or five hours from the time of admission begin to rally, and go on to recovery. Fresh air was the only remedial agent employed.

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[1] Though a very recent work (*Wilson on Fevers*) says nitro-muriatic acid, alternating with turpentine is preferred in the United States, and that mineral acids occupy the highest rank.

If fresh air does not neutralize the poison, it certainly has some effect in eliminating the poison, and thus mitigating the severity of the fever, and, perhaps, shortening its duration. It may be regarded as a remedial agent, for it certainly is of greater value than any so-called remedial agent at our command. To accomplish the best results, place three or four patients in a tent twenty feet square; the fly of the tent should be thrown up, and if the weather is cold, the patients should be well covered with blankets. By this means all the advantages of free ventilation will be insured. The question arises: what therapeutical agents can be employed with advantage in order to accomplish the desired results?

It is of the greatest importance to reduce temperature and to sustain heart-power. The former is of as great importance in typhus as in typhoid fever, and the same rules should govern one with regard to the agents to be employed, and the mode of their employment.

By the beginning of the second week, if not before, there will be evidence of heart failure, and the question presents itself, Shall alcoholic stimulants be administered? Most writers have regarded a frequent feeble pulse, with feeble cardiac impulse, even though cerebral symptoms may be present, as certainly indicating the administration of alcoholic stimulants. No limit was given as to the quantity to be administered. The object to be accomplished was to control the pulse. This could, in most cases, be done for a time; but as the disease advanced and the patient became more and more overwhelmed by the typhus poison, alcohol lost the power of giving force to the pulse.

That stimulants will control the pulse and sustain the heart's action for a time, there can be no question; but I have found that in all severe cases there came a time when alcohol, in however large doses it was given, ceased to have power.

Typhus-fever patients under twenty-five years of age rarely require or are benefited by alcohol, unless they were of intemperate habits prior to the attack. To the old and feeble its occasional administration may be of great benefit, and at times be the means of saving life. A copious dark eruption, with coldness of the extremities, especially indicates the use of alcohol. As a rule, delirium, headache, scanty urine, and intense surface heat, contra-indicate the use of alcohol. In any case when it is decided to administer spirits, carefully watch the effect of the first few doses; the same rules govern here that were laid down for the administration of stimulants in typhoid fever. It is impossible to give any positive rule as regards the quantity of stimulants required in each case. It is very rarely necessary, at any time during the fever, to give more than eight ounces of brandy during twenty-four hours. If this amount will not sustain the heart-power, I am confident larger quantities will fail to do it, and also that such administration has hastened the fatal issue. As soon as the symptoms on account of which the alcohol may have been resorted to are relieved, the quantity must be reduced, or its administration altogether stopped.

I do not altogether condemn the use of stimulants in typhus fever, but I do so as regards stimulants as a "plan of treatment;" and where the patient can be freely exposed to fresh air, I doubt if their use is often required. To diminish the frequency of the pulse, cardiac sedatives have been employed, such as veratrum viride, aconite, and digitalis.

The rapid pulse in typhus fever, after the first onset of the disease, is due to the failure of heart-power; when such is the case, digitalis should be employed. From four to six drachms of the infusion of digitalis may often be given with benefit during twenty-four hours. If the heart-power cannot be sustained by the moderate use of stimulants and by digitalis (given as indicated), no more can be done so far as remedial agents are concerned. The treatment of the special symptoms of typhus fever requires only a passing notice. The headache, when intense, is best relieved by cold applications in the form of ice-bags. If it is accompanied by intolerance of light, a blister on the back of the neck will be found to give relief.

Sleeplessness, in any stage of the disease, if it continues for two or three days, must be relieved, for it is of itself sufficient to cause a fatal termination. If sleep does not follow the application of cold to the head, opiates may be administered in full doses. I have seen typhus-fever patients that had not slept for forty-eight hours drop into a quiet sleep within a few hours after they had been exposed to free ventilation. Great care should be exercised that their apartments are kept perfectly quiet and darkened. When delirium and cerebral symptoms are associated with sleeplessness, hydrate of chloral may be carefully employed. Stupor is to be counteracted by promoting the action of all the excreting organs, applying external stimulants, and administering diffusible stimulants, the most serviceable of which are black coffee, musk, and camphor. In the early stage of the disease the cold douche may be employed.

Two remedies have been recommended for the coma of typhus, namely: valerian and phosphorus; neither of these remedies has seemed to me to be efficacious. When there are evidences of great prostration in connection with any of these special symptoms to which reference has been made, the moderate administration of stimulants may be resorted to, and if relief follows the first few doses their use may be continued. In the treatment of the complications which are liable to occur during the course of typhus fever, one must be guided by general principles and by the symptoms in each individual case, it being always remembered that the primary disease has a tendency to induce great nervous prostration and depression, and that the heart's action forbids the use of all depleting remedies, and indicates a supporting plan of treatment. The pulmonary and laryngeal complications, as well as erysipelas, bed sores, and gangrene, are to be managed in the same manner as was proposed when they occur as complications of typhoid fever.

The diet is of primary importance. Though the patient refuse all nourishment, if possible he must be required or even compelled to take it. As the digestive powers are impaired, great care is required in selecting and administering the proper nourishment, and it must be given at stated intervals, varying from one to two hours. Care must be taken not to overfeed—much harm may be done in this way. When the patient clinches his teeth and obstinately refuses all food, or is unable to swallow, his life may sometimes be saved by pouring liquid nourishment into the stomach by means of a long tube passed through the nose. Milk best serves the purpose as an article of diet. It may be given ice-cold, if desired, and in such quantities as the stomach can receive and digest. If more concentrated nutrition is desirable, the yolk of eggs may be beaten up and added to the milk.

The management of patients during convalescence from typhus fever is a matter of very great importance. As soon as the fever ceases, most patients convalesce rapidly unless there is some complication, and the chief duty of the physician is to prevent premature exertion and exposure to cold, and to restrain the patient in the gratification of an inordinate appetite. At this time porter or ale may be taken with benefit. The mineral acids, Peruvian bark, and iron may also be given as tonics; these are particularly called for when the pulse is slow and feeble. It is important to guard against any sudden physical effort during the early period of convalescence, as it may lead to coagulation of blood in the veins. An opiate or hydrate of chloral is sometimes required to produce sleep during convalescence. In all cases great benefit will be derived from a temporary change of residence and daily exercise in the open air.

RELAPSING FEVER.

This has been called *famine* and *seven-day* fever, *synocha, typhinia, mild yellow fever, typhus recurrens, dynamic* fever. The French call it "Fièvre à Rechute;" and the Germans, "Hungerpest."

Relapsing fever is no new form of disease. It was described more than a century ago by Dr. Rutty,[1] and since that time has prevailed as an epidemic disease in most of the countries in northern Europe.[2] There is no reliable history of its occurrence as an epidemic in this country until 1872-3, when an epidemic prevailed in New York City. It has been reported that in the year 1844 a vessel landed in Philadelphia passengers ill of relapsing fever. At one time, while typhus fever was prevailing in Buffalo, some twelve or fourteen cases of relapsing fever were reported; but it is altogether probable that they were cases of irregular typhus fever, for when relapsing fever has been introduced into a locality it is not limited to *one or two dozen* cases.

Morbid Anatomy.—In this disease there are no pathological lesions that are characteristic. There are changes present in some of the organs which very closely resemble those met with in typhus.

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[1] John Rutty, "A Chronological History of, etc., in Dublin, from 1725 to 1765" London, 1770.

[2] Accounts in Hippocratic writings leave no doubt but that it prevailed 2,000 years ago in islands of Thrace.

Spleen.—In the majority of autopsies, if death has occurred in the active period of the disease, the spleen will be found increased in size, its capsule thickened, smooth, tense and slightly clouded, the trabeculæ of the organ increased, and the Malpighian tufts more prominent than normal. In some cases the spleen will be found enlarged, soft, flabby, and even diffluent. There is no uniform change in its substance, although it is always increased in size during the active period of the disease. After this period has passed, it will be found diminished in size, and its surface will present a shrivelled appearance, with the capsule rolled into folds. Infarctions (not embolic in origin) are often found. In many cases a number of rounded or irregular miliary masses, of a dull yellow color, will be found, containing granular detritus, cell-elements, and free nuclei.

Liver.—During the active period of the fever, the liver will also be found enlarged. The gall-bladder is generally distended with dark yellow and viscid bile.

Kidneys.—The kidneys are increased in size, the increase being due to congestion of the cortical substance. There is a granular infiltration of the epithelium of the uriniferous tubes, a change similar to that noticed in other fevers. Small hemorrhages stud the whole organ in severe cases.

The urine often presents a cloudy appearance.

Intestines.—Usually enlargement of the glandular follicles of the intestines will be found. The solitary glands are more commonly affected, but even the Peyerian patches may present the "shaven-beard" appearance. The mesenteric glands are slightly enlarged in severe cases. Their appearance is similar to that noticed in typhus.

Mucous Membranes.—In the majority of cases, small spots of blood-extravasation will be found upon the mucous surfaces, especially the membranes of the stomach and intestines, and they may be found on the mucous membrane of the bronchial tubes. The stomach shows small blood-extravasations when vomiting has been severe during life, or when there has been black vomit. These spots of ecchymosis are perhaps as constant as any pathological lesions of the disease.

Blood.—The blood coagulates imperfectly, as in typhus. Spirilli are discoverable, oftentimes, provided death occurs in an active stage. The heart presents no constant changes. In some cases fine granular infiltration or vitreous degeneration of the muscular fibres has been observed. This same granular infiltration is also sometimes seen in the voluntary muscles. Coagula are rare.[1] Diffuse or circumscribed changes in the marrow of the bones occur, according to Ponfick. The lymphoid elements increase, and large cells, filled with numerous oil globules, appear along the track of vessels. Necrotic softening of the marrow has been seen in severe cases.

Etiology.—There have been differences of opinion and much discussion in regard to the etiology of this disease. At the present time it seems to be the unanimous opinion that it is a contagious disease, and that it is a distinct type of fever. Although it presents many phenomena which ally it to typhus, and many other phenomena which ally it to malarial fever, it is neither typhus nor malarial, but is a distinct type of fever having a distinct poison.

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[1] Ponfick, in Virchow's Archiv, B. 60, p. 153, 1874, states that the *cardiac degeneration* may be so extensive as to cause death, *resembling the heart* of phosphorus poisoning.

From observations which have been made upon the blood of patients suffering from this fever, organisms which have the power of developing the fever have been found. This parasitic organism (*spirillum Obermaieri*) is a spiro-bacterium, unlike that sometimes found in water and in mucus from the mouth. But Billroth and Manassein have found them in fluid from *noma* and in a cyst of the *antrum*. Several German observers, Cohnheim among them, have given drawings of these organisms, which seem to be little spiral lines that are constantly undergoing a twisting, rotary, rapid motion, and these observers tell us that they are distinctive of this form of disease, and are always present during its active period.[1] They are absent in the interval between the primary attack and the relapse, but are to be seen as soon as the relapse occurs.

Clinical experience has shown that relapsing fever is a contagious disease, and can be propagated by personal contagion. The disease is not necessarily accompanied by starvation, for it is developed among those who are well fed as well as among those who are badly nourished. As in typhus fever, there is a connection between the development of an epidemic of this fever and imperfect ventilation and bad hygiene.

I had never seen a case of relapsing fever until 1870, when the epidemic prevailed in New York City. At that time patients were brought into my wards in Bellevue Hospital with a fever differing from typhus fever by the absence of an eruption; from intermittent, in the order of its development, and not closely resembling remittent fever. It seemed to me an irregular form of malarial fever, differing from any with which I was acquainted. Eight cases were brought in. From these my house physician contracted the fever, and during his illness I reached the diagnosis of relapsing fever. Subsequently we had large numbers of relapsing fever patients, and a hospital was established for their reception on Hart's Island. In every case that occurred at that time, where the origin of the fever could be traced, it was found that there had been direct exposure, and it was established beyond doubt that the first cases were brought from Ireland. The contagious character of the affection was also established by the fact that all the nurses and all the physicians who were in immediate attendance upon the sick contracted the fever. If a patient was placed in a bed previously occupied by a person sick with relapsing fever, before it had been cleaned, he was almost certain to contract the disease. The closer the contiguity the more certain is the individual to contract the fever.

At the time of this epidemic we found no evidence that the fever was conveyed by clothing, although some British writers have claimed that it can be done. When our patients were admitted into the hospital, their clothing, as it was removed, was simply washed, not disinfected in any special manner, then packed away, and not a single person who was thus brought in immediate contact with the clothing contracted the disease. There is no immunity from a second attack. The period of incubation ranges between five and seven days, rarely nine.[1]

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[1] Vide Centralblatt, 1873, and Virchow's Archiv, Bd. 47. Also Obermeier's article in Berliner Klin. Wochen., No. 35, 1873. It is from two to six times as long as a red blood disc, and no thicker than the finest fibrin-fibril. It is readily destroyed by nearly every reagent.

Symptoms.—The symptoms which usher in relapsing fever are usually well marked. If there are any prodromes, they are the same as in typhoid fever (*q. v.*).

It is sudden in its advent. This is marked by a severe rigor or by a distinct chill. Accompanying the chill there is frontal headache, vertigo, pain in the limbs and joints, more or less pain in the back, nausea and not infrequently vomiting.[2] A rapid rise in temperature follows the chill, and with the pyrexia the headache increases, as does also the pain in the limbs, especially about the joints. Sweats may, at first, follow the rigors. There is vomiting, at first only of the simple contents of the stomach, afterwards of yellowish material. This may be followed by the ejection of a dark-colored material, which very closely resembles the black vomit of yellow fever. In this disease the rise in temperature is always rapid, and usually attains its highest point within the first twenty-four hours; during this time it may rise to 104° F., or even as high as 109° F. From this time, for two or three days, there is usually very little variation. With the occurrence of the chill and fever there is also a rapid increase in the frequency of the pulse. In no disease does the pulse so quickly become rapid as in relapsing fever. It is not uncommon for it to reach 140, 150, or even 160 beats per minute within the first twenty-four hours. It is usually small and compressible, sometimes dicrotic. The mind is clear. There is nothing peculiar about the countenance of the patient, but it presents the ordinary appearance noticed in an active febrile excitement. Sleeplessness is often present on account of the severe pains in the limbs. As the disease progresses the patient becomes more and more prostrated ; by the second day he may be unable to turn in bed. The arthritic pains increase in severity and often become the most distressing symptoms of the fever.

As early as the second day, patients begin to complain of a feeling of weight and uneasiness in the upper part of the abdomen, more severe in the *left* than in the right hypochondrium. Profuse sweats are common about the second day, but they afford no relief to the urgent symptoms. Usually, there is considerable enlargement and tenderness of the liver. The spleen, also, becomes rapidly enlarged, and its enlargement is attended with quite severe pain and tenderness. Moderate meteorismus is not uncommon. The muscles of the body are, however, the seat of the most severe pain, which is increased by movement and by pressure; the pain is piercing and lancinating in character. On account of this pain, the patient usually lies perfectly quiet; he is not restless but sleepless. Delirium is not an infrequent symptom, and is sometimes very active ; yet in the majority of moderately severe cases the mind remains undisturbed. There may also be present irregularities of the pupils, photophobia, and other symptoms which might lead one to the diagnosis of meningitis were it not for the character of the pulse. The muscles of the eyes are often stiff and immovable; the conjunctivæ are reddened and the eyelids are swollen.

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[1] In some cases the fever begins a few hours after exposure. Lebert found 75 per cent. of cases to have an incubatory period within seven days, and of these more than one-half sickened within three days after exposure.

[2] This headache persists till the remission ; it is unvarying and intense ; the vertigo is so severe that the patient has to take to his bed as much from giddiness as pain.

As the disease progresses, in a certain proportion of cases, jaundice is developed; this is usually accompanied by vomiting and severe diarrhœa; and these symptoms seem to ally the disease to some forms of malarial fever ("bilious typhoid"); not infrequently, especially in children, there is epistaxis. The skin may be covered with herpes or sudamina. The great prostration and rapid rise in temperature ally it to typhus fever, but the rise is more rapid and reaches a higher point within the first twenty-four hours than it does in typhus fever. There is sometimes a slight rose-colored eruption resembling roseola, but having none of the characteristics of typhus eruption. The patient goes on from day to day gradually getting worse; the fever becomes more and more intense; loss of strength and emaciation are progressive, and the muscular pains are more severe. In some cases the patient rejects everything taken into the stomach. The pulse reaches 160 per minute, the tongue is brown and dry, extreme nausea and bilious vomiting are present, and the severity of the symptoms indicates that death may speedily occur; when, on the seventh or eighth day of the fever, a remission suddenly occurs, attended by a profuse perspiration, by a critical diarrhœa, or, rarely, by bleedings from mucous surfaces.

With the occurrence of the profuse sweating the temperature falls; in a few hours it may fall five, six, or even seven degrees; the pulse becomes less frequent; the respirations, which have been hurried and difficult, become regular; the pains in the head and limbs pass away, the thirst disappears, the tongue becomes moist; the engorgement of the liver and spleen rapidly diminishes, as is shown by the rapid diminution in the size of these organs as determined by percussion. The bowels are constipated. Within twelve hours from the commencement of the remission, the temperature may fall to less than 100° F., perhaps below the normal standard, and the pulse may fall to 80 or 90 beats per minute.

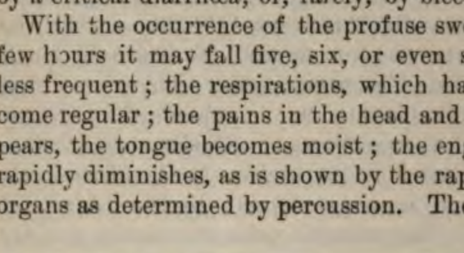

FIG. 164.

Temperature Record in a case of Relapsing Fever. Recovery.

As soon as the remission occurs, the patient feels perfectly well, except a sense of weakness. He gets out of bed, and, if he is in a hospital, perhaps insists upon his discharge; his appetite begins to return, and he appears to be rapidly convalescing, but in many the pulse at this period is as slow as 40 to 60 per minute, and the first sound of the heart is very faint, the second being intensified.

His apparent convalescence is of short duration; sometimes in three or four days, usually at the end of a week, certainly by the twelfth or fourteenth day of the disease, all the phenomena of the primary fever are suddenly developed, or what is termed the relapse occurs.[1] Sometimes the relapse occurs in the morning, sometimes in the afternoon, but more frequently it comes on at night. The relapse may be ushered in by a chill, or it may occur without a chill. The pulse may begin to increase in rapidity, and in twelve hours reach 140 per minute. With the rapid pulse, the temperature rapidly rises to 106° F. Usually the fever which attends the relapse is more intense than the primary fever, the liver and spleen becoming as enlarged as during the primary fever. The relapse usually lasts three or four days. In a few cases I have seen it last six or seven days, and in some it does not continue more than forty-eight hours. After it has continued a certain period, a second remission is developed; this, like the first remission, comes on suddenly, is accompanied by a profuse perspiration, and in twenty-four hours from its commencement the pulse and temperature have reached their normal standard. From this period, the patient usually goes on to complete recovery.

As many as three or four relapses may occur, but ordinarily the convalescence becomes complete after the second remission. Convalescence from relapsing fever is usually slow, the patient for a long time remains in a weak condition, suffering more or less from arthritic and muscular pains. The appetite returns slowly. An anæmic murmur, which is often very distinct during the active period of the fever, is heard for two or three weeks after the commencement of convalescence. Œdema of the feet, due to general anæmia, is often quite marked during convalescence. The period of convalescence is usually as long as both the period of fever and remission; not infrequently six or eight weeks elapse before relapsing fever patients are able to resume their accustomed avocations. At the commencement of convalescence, the decrease in the size of the spleen is rapid, but frequently it is a long time before the organ reaches its normal size.

Complications.—Few complications have been noticed during the course of relapsing fever. In some epidemics pneumonia has occurred quite frequently; at other times it has been exceedingly rare. When it does occur, it is often double, and terminates in gangrene in a number of cases.

Sudden collapse may occur as a complication of relapsing fever, either during the primary fever or during the relapse. The pulse suddenly becomes small, irregular, or intermittent, sometimes imperceptible. The cardiac impulse is feeble, the heart sounds are lost, and the patient rapidly passes into a condition of collapse and dies. The collapse may come on suddenly in cases previously mild; with fatty heart, death in relapsing fever is nearly always from this cause.

Post-febrile ophthalmia is another very remarkable complication or sequela of this fever. It has been observed in most epidemics. It presents two distinct stages, the amaurotic and the inflammatory. During the first stage the patient complains of impaired vision, with motes and luminous circles floating before the eyes. The inflammatory stage is characterized by intense circumorbital pains and lachrymation, without injected conjunctivæ or marked constitutional disturbance. Recovery is tedious, and, unless the case is carefully treated, may end in complete loss of sight. Both eyes are rarely attacked; the right eye is most frequently affected. Iritis, choroiditis, and retinitis are not uncommon.

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[1] It is very rare for the disease to end in one single paroxysm.

Diarrhœa and *dysentery* are common complications, and in some epidemics they are the chief cause of death. They are most likely to come on during the relapse. In our epidemics the most frequent complication is hemorrhage from the mucous surfaces, especially from the stomach and intestines. In two cases that came under my observation hemorrhagic pachymeningitis was the cause of death. In very rare instances, abscess of the spleen, accompanied by pyæmic symptoms, has occurred during the relapse and convalescence. Pregnant females, no matter at what stage of pregnancy, usually abort during an attack of relapsing fever.

Differential Diagnosis.—The diagnosis of relapsing fever is not difficult if one has the entire history of the case; but at the commencement of an epidemic, during the primary fever the diagnosis will necessarily be doubtful. The diseases with which it is possible to confound relapsing fever are *typhus, typhoid, remittent, yellow* and *dengue fever, small-pox* (before the eruption), and *measles.* It differs from all but typhus in the suddenness of its invasion, in the short duration of the primary fever, in its termination in a crisis, and in the almost uniform occurrence of a relapse between the third and fifth days. Then the muscular and arthritic pains, which are such constant attendants of relapsing fever, distinguish it from the other forms of fever.

In *typhus,* the dusky face, contracted pupils, absence of all abdominal pain, peculiar smell, *stupor,* apathy of mind, and the pathognomonic eruption on the fifth or seventh day will be sufficient to distinguish it from relapsing.

In *typhoid,* the slow invasion, the "step-ladder" rise in temperature, the eruption, the characteristic diarrhœa, and the continuance without remission or intermission, will enable a diagnosis to be reached.

A severe form of relapsing fever, attended by jaundice, resembles very closely, in its general appearance, *yellow fever.* In yellow fever the pulse is rarely over 110, the *spleen* is normal; but the high temperature and rapid pulse which attend the development of the former readily distinguish it from the latter; besides, when the intermission comes on, there can no longer be any question as regards diagnosis, for yellow fever is a disease in which only a remission occurs, not an intermission.

Small-pox simulates relapsing fever only during the period of invasion. One need make no doubtful diagnosis after the third day, when the red spots appear along the edges of the hair.[1]

In *measles* the eruption following the symptoms of a common cold and a bronchitis will suffice for a diagnosis.

Prognosis.—The prognosis in relapsing fever is always good. During our epidemic about three per cent. of all the cases treated in hospitals terminated fatally. This is a lower rate of mortality than we have with measles. Usually deaths from relapsing fever occur, not from the disease, but from some complication. During the epidemic in this city, syncope during relapse was the most frequent cause of death. Relapsing fever patients may die of hemorrhage from some of the mucous surfaces. A fatal termination may occur from bronchitis, pneumonia, or other pulmonary complications. Diarrhœa and dysentery occurring during convalescence sometimes cause a fatal termination; purpura also. Sudden suppression of urine, dependent upon renal congestion, may give rise to acute uræmia, and thus cause death. My own experience leads me to the belief that the greatest danger in this disease arises from sudden syncope. I remember one very marked case, that of a young physician who seemed to be doing well in his second relapse, when suddenly he passed into a state of syncope and died. At the post-mortem examination no condition of the internal organs was found which would account for his death.

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[1] In dengue fever the pains in the joints are severe; there are glandular swellings not found in relapsing; the paroxysm is shorter (three days) than in relapsing (seven days); and there is an eruption (like scarlatina) on the palms and neck.

Treatment.—Dr. Rutty stated more than a century ago that all those cases of relapsing fever which were abandoned to *whey* and the good providence of God recovered. The experience of a century has furnished no accepted plan of treatment. The profession are still unsettled as to the best course to be adopted in the management of this disease. When this fever appeared in our midst, we thought we could control it by large doses of quinine, but we soon found that quinine was of no service in its treatment. Then arsenic, aconite and veratrum were employed in full doses as antipyretics, but after a time these were abandoned as useless. Cold baths were resorted to, as also was sponging of the surface in order to reduce the temperature, but in their use we were disappointed. The temperature was reduced while the cold was being applied, but rose again very soon after the patients were removed from the baths, and there was no evidence that it diminished the severity or shortened the duration of the primary fever, or prevented the occurrence of the relapse. Opium in full doses was then tried, but with equally unsatisfactory results, although its free use was found to give more comfort to the patients than did any other plan. In some cases stimulants were administered quite freely, but without any apparent beneficial results.

The conclusion arrived at was, that relapsing fever patients were as well without as with medication. I would insist that relapsing fever patients should be kept quiet in bed during the primary fever, and should not be allowed to leave their rooms until the period of relapse, shall have passed and that the greatest care should be exercised to guard against the occurrence of syncope. If there is any evidence of heart-failure, digitalis and stimulants should be administered according to indications. Beyond this I have nothing to suggest. My experience leads me to place relapsing-fever patients under the best hygienic management, with free ventilation and a milk diet, and then carefully watch lest some complication should occur.

SMALL-POX.

(*Variola.*)

There are three recognized types of variola, viz.,—"variola discreta," "variola confluens," and "variola hemorrhagica."[1]

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[1] Small-pox is a very ancient disease. Before the Christian era a Goddess had been worshipped in India as a protectress against it. The Arabians gave the first detailed account of variola. During the thirteenth, fourteenth, and fifteenth centuries it prevailed in Europe, and two centuries later it appeared on the American continent. During the eighteenth century one-sixth to one-twelfth of the total mortality in Europe was caused by small-pox.

Morbid Anatomy.—Besides those anatomical lesions which occur upon the mucous membranes and skin, there is more or less intense congestion of the lungs, brain, liver, spleen and kidneys.[1] In the hemorrhagic form of small-pox small hemorrhages occur in nearly all the viscera, with ecchymoses in the serous membranes and blood-stained fluid in the serous cavities. The mucous membrane of the *stomach* and rectum is oftenest the seat of these extravasations.

The characteristic anatomical lesion of small-pox is to be found upon the mucous membranes and upon the skin. This lesion is usually spoken of as the *eruption.* It does not differ essentially in the different varieties; the modifications which are met with are due rather to its duration and the order of its development. These surface lesions pass through regular stages of development and decline.

The *first* step in the formation of a small-pox pustule is congestion of the skin in discrete spots ; the vessels of the corium are dilated and tortuous, and the connective-tissue of the papillæ, in the centre of the congested zone, shows more or less œdema. The non-elevated red spot (looking at first like a flea-bite) is a *macule.* Next, the skin is elevated at these (macular) points and a *papule* forms, from changes in the cells of the rete Malpighii. Soon the papule becomes a *vesicle ;* in its centre the epidermis becomes distended with serum and cells. As the effusion increases the cells change ; the horny layer above is raised, and the summit of the papule becomes the centre of the vesicle. The changed cell elements are pressed, separated, and massed into groups from pressure of the effusion, and a stringy mesh-work is formed in the vesicle. Meanwhile proliferation of the adjoining cells forms a peripheral wall for the vesicle, the contents of which soon become turbid.

Umbilication of the vesicles now occurs. Trabeculæ slowly spread from roof to floor of the vesicle, and hold down its centre, while marginal cell proliferation and the accumulation of serum bulge out its periphery.[2] After the vesicles are fully formed, pus-cells appear in them, and as a result the vesicles change in color, and become *pustules.* At the same time an inflammatory process, more or less extensive, is going on in the walls of the pustule, and in the surrounding cellular tissue, which terminates in a destruction of tissue at the point where the papillary congestion first occurred. If only the superficial layer of the skin is involved, the infiltration of pus-cells into the vesicle and the formation of the pustule may take place without extension of the inflammation into the cellular tissue beneath, and necrosis or death of the part will not follow ; but if the inflammation extends into the deeper tissues, a slough will be produced which necessarily will be followed by a cicatrix and pitting. After the pustule is formed the inflammatory products begin to dry down, and a *crust* is formed which contracts in the central portion, and the same umbilicated appearance is presented that is seen in the umbilicated vesicle. The incrustation begins at the centre. The crusts are made up of dried pus-cells and detritus. After a time these crusts are separated by the ordinary changes which occur in the subsidence of an inflammatory process, and recovery is complete, except that there is left behind a cicatrix which undergoes the same changes as does a cicatrix formed under any other circumstances. These pustules may be formed upon any mucous membrane. They occur oftenest in the nose, mouth, trachea, bronchial tubes and larynx.

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[1] Enlargement of the spleen is rather an infrequent event in small-pox. Weigert states that the blood-vessels of the lymphatic glands and abdominal viscera are often filled with micrococci, that necrosis of the cells about these colonies induces pus accumulation, the direct result of "coagulation necrosis" (see Inflammation) : but that abscesses rarely form ; some find an analogy between these and the skin diseases.

[2] Some explain it by saying that each papule and subsequent vesicle holds imprisoned at its centre either a hair-follicle or the duct of a sweat-gland, and that when this epidermidal layer of the papule is elevated by the serous exudation or infiltration, the portion immediately about the hair-follicle or the sweat-duct is held down, and a depression is produced by the exact point where the hair-follicle or duct of the gland may be situated ; but since umbilication is present when neither structure is found, this view cannot be accepted.

There is nothing specific or essentially different in the development of the pustules in *hemorrhagic small-pox*, except that they contain blood instead of serum or pus. In the hemorrhagic variety, larger or smaller hemorrhages take place into the cellular tissues and into the cutis; in the milder forms they take place only in the layer beneath the papillæ; while in the severer forms they take place beneath all the cutaneous layers; even the subcutaneous fat may be infiltrated with blood. No changes in the walls of the vessels have as yet been discovered which will account for these hemorrhages. These extravasations more frequently occur in those cases in which death takes place before the period of pustulation is reached. In hemorrhagic variola blood extravasations occur into the substance of all the organs, the marrow of the bones, and on mucous and serous surfaces, and infarctions in the lungs are the rule. Hyperæmia and œdema of the brain sometimes occur.

Etiology.—The disease is propagated only by contagion; it is a disease which can only be produced by its own specific poison, and is communicable only to persons who are not protected from its influence. There has been considerable question as to *where* the virus of small-pox is located. Some claim that it is exclusively in the pustule, and that it is not possible for a person suffering from small-pox to give the disease to an unprotected individual unless some of the virus from the pustule is brought in contact with a cutaneous or mucous surface. This is a mistake. That small-pox can be conveyed by means of virus taken from a pustule there can be no question,—"contagion by inoculation,"—but the cutaneous surface of an unprotected person may be rubbed with pus taken from a small-pox pustule, and unless there is an abrasion of the surface the person will not become inoculated with the disease; but if the virus is brought in contact with a mucous surface of an unprotected person he will almost certainly contract the disease. It is equally certain that the disease can be communicated from one person to another by means of the breath and exhalations from the skin.

There is no evidence that the disease can be conveyed by the discharges from the bowels. Perhaps if a pustule should be developed somewhere along the line of the intestine the discharges might become so contaminated as to have the power of communicating the disease. Small-pox can also be conveyed from one individual to another through the atmosphere. In the open air the distance of contagion is about two and one-half feet.

In a small room the atmosphere may be so contaminated that an unprotected person will contract the disease upon a single entrance into the room. The disease can be conveyed in clothing, and the poison will remain for a long time in clothing unless it has been exposed for a considerable time to the air. In other words, there is no doubt but that it is a portable disease. In order that the disease may be transferred by means of the clothing or merchandise, it is necessary that the clothing or merchandise contain the pus or crusts from the small-pox pustules; how long a time may elapse before the virus loses its vitality is not known. There are well-authenticated cases in which it has retained its virulence for more than a year.

No period of life is exempt from the contagion of small-pox; even intra-uterine life is in danger from infection. Rarely does an individual have a second attack. I remember one exception, that occurred in the person of a young Swedish woman, who, under my observation, passed through three well-developed attacks of the disease; the last attack was the most severe. Concerning the exact nature of the small-pox virus nothing definite is known.[1] Some claim that the earliest period at which one suffering from this disease can infect the unprotected, is the period of suppuration; others, that the infecting period is during the stage of desiccation. There are well-authenticated cases, however, which prove that infection may take place during any stage of the disease, even during the period of incubation. There is little doubt but that the suppurative stage is the most infectious period.

There are many views as to the manner in which the small-pox poison gains entrance into the system; the most probable of these views is, that it is principally absorbed by the mucous membrane of the respiratory tract during respiration, and it is also probable that exceedingly fine particles detach from the pustules and crusts, which are suspended in great numbers in the air surrounding small-pox patients, and that these convey the contagion. There are no facts to sustain the views as to the parasitic nature of this contagion.

The length of time which elapses after exposure to, and reception of, the variola contagion before the disease is developed varies from five to thirty days, giving the extremes. This is called the period of *incubation*, during which the recipient of the poison usually presents no abnormal symptoms. If the poison is introduced into the system through inoculation, only forty-eight hours elapse before the characteristic phenomena of the variola are manifested. It is not known what change takes place in the body of the infected person during this period of incubation. Usually, twelve to fourteen days after exposure, one who has contracted small-pox begins to feel chilly; this feeling of chilliness increases until he has a distinct chill. This has been termed the initial stage, or the stage of initiatory fever. *Measles* and *small-pox* poisons may be latent at the same time in the same individual; also *scarlet fever* and *small-pox*.

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[1] Cohnheim and Weigert state that the micrococci in the vesicles are the contagious, specific elements.

Symptoms.– The transition from the stage of incubation to that of the initiatory fever is sometimes abrupt and sometimes gradual ; usually it occupies two days, and is followed by the eruption. In this stage there is greater variation in the intensity than in the duration of the symptoms. The intensity of the symptoms bears no relation to the severity of the attack. Not infrequently, the most violent symptoms in the initial stage are followed by a mild attack of variola; while mild symptoms in the initial stage may be followed by the gravest form of small-pox. The headache, which usually precedes the fever, grows more intense, only subsiding as the eruption appears. With the chill, which may be more or less severe, there is pain in the head and back, especially in the middle of the back and loins,[1] with this pain there will be rapid rise in temperature. The chill is more severe than in any other exanthem. During the first day the temperature may rise to 104° F., during the second day to 105° F., and by the third day it may reach 106° F. or 107° F.; in some cases it has been said to have reached 109° F. Sweating begins with the first rise in temperature, and continues till the period of eruption. With this rise in temperature there will be an acceleration of pulse ; it may reach 100 or 120 beats per minute. In the strong and robust person, the pulse will be full and not easily compressed. In females, and in the weak and feeble, the pulse has less volume and usually is more frequent ; it may reach 140 beats per minute. In children, 160.

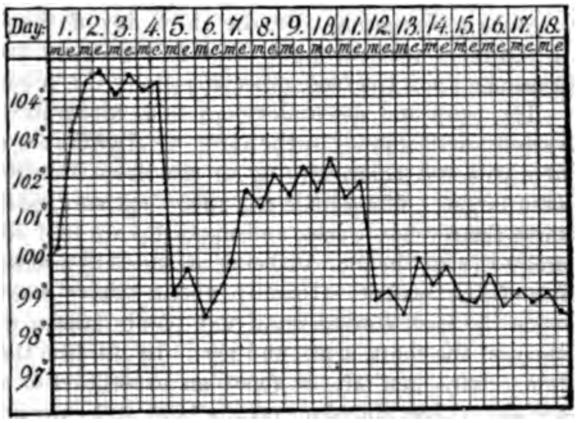

Fig. 165.

Temperature Record in a case of Discrete Small-pox.

At the onset, there is usually more or less nausea and vomiting, and soreness of the throat. This soreness of the throat may have preceded the chill by twenty-four hours, but now in many cases it will be quite severe, and the patient will complain of more or less dysphagia, and pain in the pharynx. The extent of the throat symptoms will depend upon the severity of the attack. In the severer forms of the disease, by the third or even before the end of the second day, there may be delirium. In all cases, the face will be flushed, the conjunctivæ congested, and there will be throbbing of the carotids. With these symptoms, there will be great restlessness, and an anxious expression of countenance, with somnolence. The respirations will be short, frequent, and labored, many complaining of dyspnœa in whom there are no lung complications. Many suffer from extreme vertigo, and in children convulsions are not infrequent. By the evening of the second, or morning of the third day, swelling and diffuse redness of the tonsils and soft palate are present; not infrequently the swelling and redness of the mucous membranes extend into the larynx, causing hoarseness and huskiness of the voice and a stridulous cough.

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[1] Incomplete paraplegia has occurred, disappearing, however with the appearance of the eruption.

During the fever of invasion patients are languid and weak in proportion to the severity of the fever.[1] Frequently within twenty-four hours after the ushering-in chill, the strongest and most vigorous will be unable to get out of bed. Paralysis of the bladder may occur in this stage. The tongue is coated. There is epigastric pain and tenderness. If vomiting occurs it is present at the very beginning, and continues with great obstinacy throughout its entire course. In the hemorrhagic variety the matters vomited may contain blood. There is constipation, but diarrhœa is not infrequent in children.

Stage of Eruption.—By the third day of the disease, at least after the initial fever has continued three full days, an eruption will make its appearance upon the face, especially along the edges of the hair.[2]

The eruption, as it develops in a moderately severe case of discrete variola, first appears in the form of slightly elevated maculæ. These are of a pale red color, varying in size from a millet-seed to a pin's head, or even larger. These little red spots look very much like flea-bites. In most cases the forehead, nose, and upper lip are first covered; they gradually increase in size, the increase being attended by a sensation of itching and burning of the surface. Usually, about twelve hours after their appearance upon the face, similar small red points appear upon the neck and wrists, then on the chest, arms, and legs. In children they may first appear on the loins, nates, genitals, or about an excoriated or a blistered surface. They are always less abundant on the body and extremities than on the face.

On the second day of the eruption, these spots assume a darker red color, become elevated, and have a distinctly papular feel, like shot under the skin. In a majority of instances, as they enlarge a depression is formed, which gives them an umbilicated appearance. The appearance of the eruption is attended by a subsidence of the febrile symptoms, the patient no longer complains of pains in the head and back, the temperature falls two or three degrees, and the pulse diminishes fifteen or twenty beats in frequency, sometimes to normal. Vesicles are also seen in the mouth, pharynx, upper part of the larynx, etc., etc.

Stage of Suppuration.—About the sixth day of the eruption the contents of the vesicle, from the admixture of pus-corpuscles, gradually become turbid, and by the eighth day the pustules become fully formed, and the disease enters on the stage of suppuration. The integument in the immediate vicinity of the pustule now becomes red, œdematous, and tumefied, each pustule being surrounded by a broad red base, the "halo," and where they are thickly set they become confluent. The face swells to a shapeless mass, and the patient becomes frightfully deformed. The eyes are closed, and the hands and feet look like round balls. The itching now becomes almost unbearable and causes the patient to scratch himself, thus causing ultimate disfigurement. During this period a characteristic sickish odor is emitted. The eruption passes through its stages two or three days later on the extremities than it does on the face ; consequently, suppuration may be complete on the face while it is incipient on the extremities, and the eruption may be perfectly discrete on the trunk while it is confluent on the face. About the eighth or ninth day of the eruption the pustule is fully formed ; the stage of suppuration is complete. Then commence the retrograde changes. The pustule either ruptures, discharges its contents, dries up and forms a yellowish crust, or it shrivels and dries up without rupturing ; this is the period of *desiccation.*

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[1] Trousseau and many others state that the longer the skin manifestations are delayed, the more harmless the disease, and the more rapidly the eruption comes on the more dangerous is it.

[2] *Prior to the eruption* a diffuse scarlatina-like redness sometimes covers all the body, and a few sudamina may appear in the erythema. At this point haste may lead to a diagnosis of scarlet fever or measles. Petechiæ and ecchymoses are less frequently seen; they are not necessarily followed by variola hemorrhagica.

Stage of Desiccation.—Desiccation commences in those parts in which the eruption first appeared, and commonly on the twelfth day of the disease. As the drying down of the pustules takes place, the redness, tenderness and œdema of the skin lessen, and the countenance begins to assume a more natural appearance. At first the crust adheres quite firmly to the surface, but about the fourteenth day of the eruption it becomes separated and falls, leaving a stain of reddish-brown color, with elevated edges and depressed centre, which remains visible for five or six weeks. These spots gradually become lighter in color, until finally, if there has been destruction of the cutis, and if excoriation, ulceration and renewal of the scab have occurred, a pit will be formed of greater or less depth, of a white color, giving to the face a "pock-marked" appearance, which will remain during the life of the individual.

On the eighth day of the eruption the secondary fever comes on. It often commences with a distinct chill. The fever is highest in the evening; it is of a distinctly remittent type, the pulse becomes frequent, the temperature rapidly rises, perhaps reaches a higher elevation than it did during the initial fever, sometimes rising as high as 108° or 109° F. ; it reaches its maximum when suppuration is at its height. As desiccation commences, the temperature begins to fall, and by the time the crusts are fully formed the temperature reaches very nearly a normal standard. If the temperature rises again, its rise is due to some complications such as erysipelas or some phlegmonous process. With the fall of the crusts, the patient's appetite returns, and he is able to sleep; convalescence is now fully established.[1] The dividing lines between the different varieties of small-pox are not sharply defined ; one variety gradually passes into another. It is unnecessary to consider all the forms into which this disease has been divided by some writers ; frequently the basis of the division is merely arbitrary. Our attention will therefore be confined to the more common and well-recognized varieties.

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[1] The menses appear in the initial stage in the large majority of women with small-pox, even though it be not the proper time. (Quincke, Leo, Knecht, Curshmann, Buck, Obermeier, and others.)

Confluent Small-pox, or Variola Confluens.—This is a much more severe form of the disease than *variola discreta.* It develops far more rapidly and is much more fatal in its results. The fever of invasion is usually much more severe, and of shorter duration, frequently not lasting more than forty-eight hours.[1] The eruption spreads rapidly over the entire body, often appearing simultaneously on the face and the other portions of the body. The red dots which mark the first appearance of the eruption are very numerous, especially on the face and hands; on the first day of their appearance they are almost confluent. The conjunctivæ are early involved and suppurative keratitis is not uncommon in this variety;—the whole eye may be converted into an abscess. On the second day the skin is intensely red and swollen, and so thickly studded with large flat vesicles that they rapidly unite, suppuration speedily follows, and flattened, yellowish-colored confluent patches are formed upon a dark, reddened, swollen skin. Gradually these patches run together over a still larger surface, and the epidermis is elevated in the form of large, flat bullæ, which are filled with a seropurulent fluid and are tense and elastic. In this way the entire skin of the face is covered by an immense bulla, and the patient is as unrecognizable as though he wore a mask. While the eruption may be completely confluent on the face and hands, on other parts of the body it remains discrete, and never becomes confluent except over limited spaces.

The period of desiccation is slowly reached. Large concentric crusts are formed over the confluent patches; these adhere firmly to the skin, while beneath them suppuration of the papillary layer continues. The true skin is more or less extensively destroyed, and when the crusts have fallen, there is left extensive loss of substance in the cutis, giving rise to pits and ugly scars, which have a tendency to contract, often producing permanent and unsightly disfigurements. In this variety of small-pox, the eruption is often confluent upon the mucous membrane of the mouth and throat; it may involve the mucous membrane of the posterior nares and extend into the larynx. In some cases the attending pharyngitis is so severe as to render deglutition impossible. The pharyngeal inflammation is submucous, and is frequently accompanied by more or less enlargement of the parotid and sublingual glands. When this condition exists there is danger of the sudden development of œdema glottidis, for the occurrence of which one should be on the watch.[2]

In confluent small-pox hemorrhage may occur in the pustules; this is not variola hemorrhagica, but a hemorrhagic pustular confluent small-pox. In confluent variola the skin may exhibit erysipelas, phlegmon, gangrene or multiple abscesses. In confluent small-pox the severity of the constitutional symptoms corresponds to the severity of the local manifestations.

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[1] The thermometer not infrequently shows a fever of 106° to 110° F. for a short time, which sinks to 103° to 104° till suppuration, then rising even higher than before.

[2] During the year that I had charge of the Small-pox Hospital, there were three cases in the hospital of œdema glottidis: one case terminated fatally before I reached the patient; life was saved in the other two cases by the performance of laryngotomy.

The temperature during the initial fever often reaches 106° F. or 107° F., and in very severe types of the disease it may rise as high as 110° F. The pulse is correspondingly frequent and feeble. After the appearance of the eruption the temperature falls slowly to 103° F. or 104° F., where it remains until the stage of suppuration is reached; then it again rises, in some cases even higher than during the period of invasion. Violent delirium is very frequently present during the fever of invasion, as well as during the period of secondary fever, and not infrequently patients pass quite suddenly into a state of coma. Uncontrollable vomiting and obstinate diarrhœa are not infrequent, coming on during the fever of invasion and continuing throughout the course of the disease.

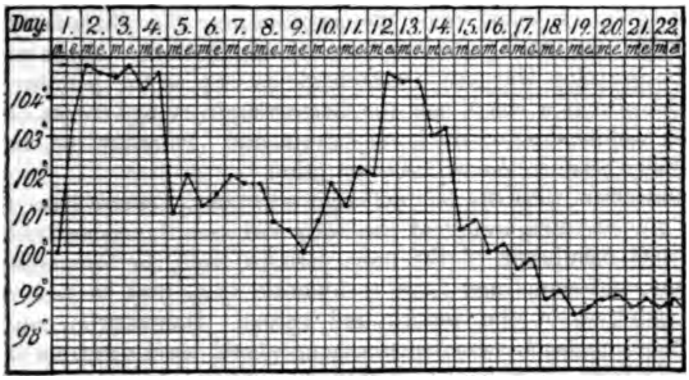

FIG. 166.
Temperature Record in a case of Confluent Small-pox.

In *all severe* cases typhoid symptoms manifest themselves soon after the appearance of the eruption, and patients often lie for days in a semiconscious state, with dry, brown tongue, subsultus, a low muttering delirium, and all the attendant phenomena of intense nervous depression. In such cases albumen appears temporarily in the urine. Complications occur much more frequently in confluent than in discrete small-pox. Inflammations of the serous membranes, especially pleurisy and pericarditis, are the most common. Croupous and catarrhal pneumonia and acute laryngitis frequently complicate the severe bronchial inflammation from which so few patients with confluent small-pox escape. Permanent alopecia often follows confluent small-pox.

Variola hemorrhagica[1] is a form of small-pox which can hardly be regarded as a distinct variety, but rather as a modification of other varieties, called the black or malignant small-pox. It differs from the varieties already described, not in the manner of its development as far as the initial fever is concerned, but in the appearance of the eruption. This hemorrhagic tendency is often manifested as early as the first appearance of the eruption, by the dark color which the eruption assumes. Sometimes the papules become hemorrhagic from the very moment of their development; at other times they first become vesicles, and then become hemorrhagic. Again, at other times, the hemorrhage first shows itself after the vesicles become pustules. In some cases the eruption over the whole body becomes hemorrhagic ; in other cases, it is hemorrhagic in spots. In the majority of cases, the eruption becomes hemorrhagic as soon as the papules have attained the size of a lentil, and the hemorrhagic change comes on slowly, generally commencing on the lower extremities. Petechiæ and ecchymoses often appear between the points of eruption.

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[1] Zulzer found that in eighty-five to ninety per cent. of his cases of hemorrhagica, the period of incubation was only six to eight days, *i. e.*, half as long as in simple small-pox.

In connection with the hemorrhagic eruptions, hemorrhages from the various mucous membranes of the body will simultaneously occur—from the mucous membrane of the nose, perhaps from the bronchial mucous membrane, and sometimes large ecchymotic spots may be seen upon the mucous surfaces of the mouth and throat. Hæmaturia, conjunctival hemorrhages, melæna, hæmatemesis, hæmoptysis, bleedings from the gums, and particularly epistaxis are met with. It is rare for this form of small-pox to reach the stage of suppuration, for before this stage is reached patients die. During the initial stage of this variety of small-pox, the constitutional symptoms do not differ from those which attend the development of the other forms of this disease. It is impossible, from their character and intensity, to predict, with any degree of certainty, the subsequent development of hemorrhagic variola. It has been said that the pains in the back and limbs are more severe ; but these are not characteristic. Frequently the fever of invasion is exceedingly violent, while during the eruptive period, and during the entire subsequent course of the disease, the temperature is comparatively low. In cases in which extensive hemorrhages have occurred, the temperature often falls below the normal, while the pulse ranges from 140 to 160, and is exceedingly feeble in character. Only when comparatively few of the vesicles become hemorrhagic does the case terminate in recovery.

Differential Diagnosis.—The first question that arises is : how early can small-pox be recognized ? One who has seen very many cases of the disease may be able to reach a diagnosis on the third day, that is, the first day of the eruption, although at that time there is nothing characteristic about the eruption or the ushering-in symptoms. It is, however, better and safer to wait until the second or third day of the eruption before making a positive diagnosis, for there is little to be feared from infection until the vesicles are fully formed.

The eruption of *measles,* in its early stages, is liable to be taken for small-pox. If one defers making the diagnosis until the vesicles are fully developed, no such mistake will be made. In measles there is coryza, a cough, sneezing, redness and suffusion of the eyes. These symptoms are not present in small-pox. The range of temperature is two to three degrees higher in small-pox than in measles. In these respects the two diseases differ sufficiently to enable a differential diagnosis to be made. Again, if one waits until the vesicles become umbilicated, it will be impossible that a mistake in diagnosis should be made.

During the period of initial fever it is possible to mistake small-pox for *typhus fever*. In both diseases there may be delirium, pain in the head, vertigo, high temperature, and evidence of great disturbance of the nervous system. There is no system which will enable a positive diagnosis to be made during the very early period of the disease. Of course, if typhus fever is prevailing, or if small-pox is prevailing, and the patient has been exposed to either one of these contagions, one will be able to make a diagnosis without difficulty. Usually there is greater loss of muscular power in typhus fever than in small-pox, but this symptom is not always well marked. By the third day, the appearance of the eruption upon the face, where it is first seen, settles the question of diagnosis. The eruption of typhus fever is first seen upon the abdomen, and may extend over the whole body without appearing on the face. It rarely appears before the fifth day of the fever. Therefore, the differential diagnosis between small-pox and typhus fever can be readily made as soon as an eruption appears. The temperature falls as soon as the eruption occurs in small-pox, and does *not* in typhus.

Meningitis is another disease which small-pox, in its initial stage, resembles. There is always considerable cerebral disturbance and a full, hard, bounding pulse in the initial stage of small-pox. Photophobia, intense pain in the head, nausea and vomiting may be present in both diseases. Unless it may be the expression of the face, there is often no distinguishing mark between the two diseases in their early stages. In meningitis, there is usually a pale, anxious expression of countenance, whereas early in small-pox the face is flushed, and day by day the flush deepens until the eruption appears. The fever in meningitis is lower than in small-pox by 2° to 3° F., the pulse is smaller, less compressible, and not as rapid as in variola; and the vomiting is projectile in meningitis, while it is retching in character in variola. On the appearance of the eruption, the differential diagnosis between these two diseases is readily made.

Prognosis.—The prognosis in any case of small-pox depends upon the amount of the eruption; the more abundant the eruption, the greater the danger to life. The prognosis also depends upon the type of the disease. Unless some complication arises, most cases of discrete small-pox recover; while of confluent small-pox nearly one-half the cases prove fatal.[1] The best record obtained in the small-pox hospital on Blackwell's Island was one death in every five cases. Only a very few cases of the hemorrhagic variety recovered, and when recovery did take place it was only reached after the patient

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[1] In twenty years the "London Small-pox Hospital" gives the following definite statistics:

	No.	Mortality.
Patients admitted with small-pox	4,879	
A. With 1 vaccine scar	2,001	7 7-10 per cent.
B. " 2 " scars	1,446	4 7-10 " "
C. " 3 " "	518	1 9-10 " "
D. " 4 or more scars	544	1-2 " "
E. Said to have been vaccinated, but no scar visible	370	23 1-2 " "

In the "London Small-pox Hospital" the mortality is: 4 per cent. of discrete, simple variola; 8 per cent. of semi-confluent variola; and 50 per cent. of confluent variola.

had passed through an apparently fatal condition of coma. The ratio of mortality is always lower at the end than at the beginning of an epidemic. The disease is more fatal in the summer than in the winter.

The age of the patient greatly influences the prognosis. In infancy and old age the ratio of mortality reaches its maximum. Among adults the prognosis is worse in females than in males. In the intemperate the prognosis is always bad, for with this class of persons the disease is liable to assume a hemorrhagic type. The intemperate die in discrete small-pox when the temperate would almost certainly recover. In the overworked and badly-nourished the prognosis is bad. The robust and healthy pass through a severe type of the disease much more safely than those enfeebled by chronic disease. The severity of the fever of invasion is not a safe guide in prognosis. Sometimes a severe initial stage precedes a mild form of the disease; sometimes patients with this disease pass into a state of complete unconsciousness, remain in that condition for some time; then the eruption begins to change in color, and finally recovery takes place. Such cases, however, are exceptional. However well-developed the eruption may be, or however well-filled the vesicles, it is to be remembered that the eighth day is the commencement of the suppurative fever, which is the period of the greatest danger. Upon this day the patient may pass into a state of collapse, the result of the depressing influence upon the nervous system produced by the large extent of surface involved in the suppurative process. In most cases in which patients do not die until the second week of the disease, the fatal result is due to exhaustion, although death may occur from complications. Usually they pass into a typhoid condition, the result of the excessive drain upon the system by the suppurative process.

Pregnancy is a bad complicating condition; in the confluent, the *absorption* that is so liable to occur is likely to be attended by fatal *bleeding*. The most frequent complications which cause death are those which occur in the throat and air-passages. In some instances swelling of the glands of the neck and mucous membrane of the throat takes place to such an extent as to seriously interfere with deglutition and respiration. When this occurs it becomes an element of great danger, and materially affects the prognosis. The tongue may become swollen to such an extent that the patient will be unable to protrude it, or, being able to protrude it, will not be able to retract it. Under such circumstances deglutition is almost impossible. There may be laryngeal ulcers, and ulcers occurring in the trachea and in the bronchial tubes.[1] Whenever, in the course of the disease, the urine becomes scanty and high-colored, but especially when it becomes so at the commencement of the secondary fever, it is certain that kidney complication exists. Under these circumstances the patient may pass into a condition in which convulsions will be developed, and coma and death ensue.

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[1] Keratitis, choroiditis, iritis, conjunctivitis, inflammation of the middle ear, ulcers in the nose, acute arthritis (of the large joints), pericarditis, ulcerative endocarditis, pyæmia, and erysipelas—these are all occasional complications. Diphtheria is a common complication of hemorrhagic variola. *Cerebral hemorrhage* is not an infrequent complication of small-pox; *aphasia* may also occur, and thrombosis of the basiliar artery may induce a "dementia-like" condition. (Collie.) Boils, abscesses, and phlegmons of the skin are frequent sequelæ of small-pox. Blindness and deafness also not infrequently follow, as also paralysis of the bladder and paraplegia, due (according to Westphal) to acute disseminated myelitis that has complicated the fever.

Treatment.—In vaccination, properly performed, we undoubtedly possess a means by which we may prevent one from contracting the disease when exposed to its infection. But the question arises, have we any power to arrest the development or mitigate the severity of the disease after the initial fever is established? No reliable affirmative answer has been given to this question. It has been proposed to accomplish this by blood-letting, emetics, diaphoretics, purgatives, cold baths, and more recently by the subcutaneous injection of the vaccine virus. All of these means have been tested, and have failed to accomplish the desired result. The assertion that large doses of quinine, given during the stage of invasion, will shorten the duration and modify the course of the disease, is verified only by the experience of its author (Stiemer). Quite recently it has been claimed that carbolic and salicylic acids destroy the septic poison of the variola, and thus shorten and modify the course. My own experience as regards their use has not been sufficient to decide the question, and I am unable to find any statistics which sustain such an assertion.[1]

During the fever of invasion all that can be done is to treat special symptoms. Place the patient in bed in a large well-ventilated apartment; if possible, keep the temperature of the room below 60° F. I remember that, in the Small-pox Hospital, those patients did best who were placed in barracks, which were so open, that frequently, during the winter months, when I made my morning visit, I would find little snow-drifts on the floor between the beds. When the body temperature ranges as high as 107° F. or 108° F., it is recommended to employ cold to the surface, and to give antipyretic doses of quinine to reduce the temperature. If the headache is severe and the face flushed, iced compresses and ice-bags to the head will usually afford relief. If the vomiting is severe and constant, iced carbonic acid water may be given, and if the vomiting is attended by great restlessness, hypodermic injections of morphine are indicated. Administer such food as can be readily assimilated. I have found nothing better than iced milk and seltzer water. If the bowels are constipated, it is well to relieve them by enemata of cold water. In those cases in which the eruption is tardy in making its appearance, and the temperature is higher, sometimes, if the patient is kept in a warm bath for fifteen or twenty minutes, the development of the eruption is hastened.

When the eruption has appeared, the measures to be employed will vary with the character of the eruption. The milder forms of discrete variola require no interference. In the severer forms the attendant symptoms will decide the means to be employed. Sooner or later, sometimes very arly in the severer forms of the disease, the patient will be found sinking rom the depressing effects either of the small-pox poison or of the supurative process which is taking place upon the surface of the body. Under such circumstances stimulants are indicated. There is no question but that the free use of stimulants for a few days, just at the period of suppuration, in very many cases does much to save life. At this time the patient has a dry tongue, a frequent, feeble pulse, blue lips and finger ends, giving evidence that he is rapidly passing into a state resembling that met with in the later stages of typhoid fever. Active delirium is frequently present; the patient insists upon getting out of bed. Under these circumstances, life will often be saved by the judicious use of stimulants. If the delirium is excessive, hypodermics of morphine may be combined with the administration of stimulants.

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[1] Zulzer (one of the authors in Ziemssen) states that xylol given internally coagulates the contents of e pustules and cuts short their *development.*

During the stage of desiccation, warm baths employed every day or every other day give great comfort, and assist in the removal of the crust. After the baths the surface should be freely oiled. Complications will be treated according to the general rules which govern their treatment. If abscesses occur in the subcutaneous tissue, they should be freely opened at once. We are powerless when we come to deal with the hemorrhagic form of small-pox. Although tonics and stimulants have been highly recommended, they do little good. Transfusion has been proposed and practised with no definite results. If the mouth and pharynx are very much involved, and there is difficulty in deglutition, ice-cold carbonated water with a weak solution of the muriated tincture of iron used as a gargle will often give great relief. Sometimes the stronger antiseptic gargles, such as carbolic acid and the permanganate of potash, will be of service.

There is still one point in the treatment of small-pox which is deserving of attention, and that is, what means may be employed to prevent the pitting, especially upon the face, which is so frequent a result. The eruption first makes its appearance upon the face; there it is usually most abundant, and is most liable to be followed by pitting, and there it passes more quickly through all its stages than upon any other part of the body. In order to prevent pitting it has been proposed by some to exclude light and air from the surface covered by the eruption. For this purpose a great many substances have been employed, such as collodion, gutta-percha, certain forms of plaster, liquid paper, etc., etc. All these substances are to be so applied as to form a mask for the face, which completely excludes light and air from the surface.[1] The pitting is due to the formation of a slough, and the slough is seated in the areolar tissue; if by any means you can so interfere with the inflammatory process as to prevent the formation of a slough, you will prevent the pitting. It was claimed by those who advanced the theory that excluding light and air prevented the pitting, and that it did this by preventing the occurrence of sloughing.[2]

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[1] Gold leaf, mild mercurial ointments, bismuth, chalk and sweet-oil, linseed meal poultices, collodion, carbolic acid and white lead paint have all been extensively used.

[2] When I had charge of so many small-pox patients, I took pains to test all those applications which at that time had been and are still recommended for the purpose, and I satisfied myself that about the same results were obtained in the use of every remedy, and in no case was pitting prevented. Certain patients were much more scarred than others, but that was the natural result of the disease. Some have proposed to coagulate the serum in each vesicle by nitrate of silver, and to paint each papule with iodine, and so arrest the inflammatory process and prevent pitting. But the use of these means has been

INOCULATION AND VACCINATION.

There are two recognized methods of protection against the infection of small-pox: inoculation and vaccination. Inoculation was first introduced into England by Lady Montague, who first practised it upon her own child.[1] Subsequently it was quite generally practised throughout Great Britain. Pus from a small-pox pustule was introduced beneath the epidermis of one who had been prepared by diet and general hygienic measures for the safe development of the disease. It was claimed that the disease resulting from inoculation was a modified small-pox, differing from the original disease in that it ran its course more rapidly, was attended by few pustules, perhaps no more than twenty or thirty, and was said to rarely terminate fatally, the ratio of mortality being about one in one hundred. Those who were inoculated were as fully protected from small-pox as those who had the disease in the ordinary manner. The disease developed by inoculation passed through the regular stages of small-pox.

Early in 1776 Edward Jenner observed that in some of the northern counties of England persons employed in dairies, who suffered from a certain form of ulcer upon their hands, did not contract small-pox when exposed to it.[2] He also found that these ulcers upon the hands resembled pustules found upon the udder of the cow, and seemed to have been caused by contact with them. Jenner made a thorough investigation of the subject, and arrived at conclusions sufficiently satisfactory to himself to warrant the experiment of taking matter from one of these pustules found upon the udder of the cow and introducing it into the arm of the individual who was supposed to be unprotected from the contagion of small-pox. After the sore upon the arm had run its course, he exposed the individual to the infection of small-pox, and in this way he established its protecting power. In 1796 he made his first vaccination on man. In 1798 he published his first paper on the subject.[3] Vaccination was introduced into this country in the year 1799, by Waterhouse of Boston, and very soon became the practice of the profession. In 1800 it was first practised in France. At the pres-

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attended by the same unsatisfactory results. The only means which I found of certain value was a simple cold-water dressing applied over the face, after having ruptured each vesicle before it became a pustule. In this way, I was able to diminish the intensity and extent of the inflammation. This plan of treatment I adopted in twenty cases of confluent small-pox, and it not only gave the patients very great comfort, relieving them to a certain extent from the intense itching, thus avoiding rupture of the vesicles by scratching, but not in a single case that recovered was there bad pitting. In the treatment of small-pox, the prevention of pitting is of greatest importance to certain patients, especially young unmarried females.

[1] In 1717 Lady Montague, writing from Adrianople, in Turkey, where the practice of inoculation was in vogue, says: "They take the small-pox here for diversion; I have tried it on my dear little son; I am going to bring this useful invention into fashion in England." In 1718 it did become the fashion.

[2] In 1771 a Holstein schoolmaster vaccinated three pupils, and in 1774 an English farmer vaccinated his wife because of his belief in the power of bovine virus as seen in his dairymaids.

[3] During six years no member of the profession ever received more anathemas or more scurrilous abuse than Jenner. He was attacked by the leading physicians and surgeons of Great Britain, and persecution and ridicule so followed him that placards with caricatures of Jenner were posted throughout the streets of London and the principal towns of Great Britain; Jenner kept steadily at work and repeated his experiments, until he became fully convinced that by vaccination perfect protection could be obtained against small-pox. Within the short space of six years Jenner compelled the profession to admit his statements and adopt his practice, and within the five or six years following its first recognition, the practice of vaccination became generally recognized and practised.

ent time there is no question among the intelligent portion of the profession but that vaccination, properly performed, is a perfect protection against the infection of small-pox; if persons contract small-pox after they have been vaccinated, then it has not been properly performed.

There are two methods of performing vaccination. One method is to take the *virus* directly from the cow; this is called *bovine* virus; the other method is to take the *virus* from a vesicle developed upon the human body,—perhaps a vesicle removed from the original by several vaccinations,—this is called *humanized* virus. To-day good humanized virus is warmly advocated; first, because it is more successful (98 per cent.) than bovine virus (only 70 per cent.); and secondly, because it is a surer safeguard. Jenner found that there were several pustules developed on the udder of the cow which closely resembled each other, but that only *one* contained the virus which afforded protection from small-pox. In obtaining bovine virus it is of the greatest importance that the genuine vesicle be selected. In order to make the selection, it is necessary one should be familiar with the peculiarities of each variety. If humanized virus is used, there is danger of introducing into the system the infection of other diseases. I have in my possession facts which prove beyond the possibility of a doubt that syphilis can be conveyed from one person to another by vaccination. Cutaneous eruptions may also be conveyed by humanized vaccine virus, which cause the development of very extensive and serious cutaneous diseases. Again, if any chronic or acute skin disease exist at the time the vaccine vesicle is running its course the protective power of the vaccination will be altogether destroyed or very greatly modified.[1] The vaccine virus is usually introduced by scarifying the surface so as to redden it, scarcely drawing blood; then the surface of the quill containing the virus is applied to the scarified part, or the lymph is conveyed from one to the other by direct transmission.

Any irregularity in the development of the vesicle destroys in a greater or less degree its protecting power. When an individual has been once vaccinated, a second vaccination is liable to run an irregular course.

A primary vaccination, such as the first vaccination of a child, *should pass through the following regular stages, and if it does not it fails to give protection:* upon the third day after the introduction of the virus there will be noticed at the point whêre it was introduced a little red spot,—a papular elevation. By the fourth day this little red spot will be occupied by a bluish-white vesicle, and at the commencement of the fifth day there will appear around the vesicle a little yellow margin. This vesicle goes on increasing in size up to the eighth day, when it will become umbilicated and there will appear around it a distinct areola; about the seventh day there has been a trifling areola present; on the eighth or ninth day it becomes very distinct. Now a change is to take place in the vesicle, and by the next day it will be noticed that the areola has extended, perhaps so as to measure an inch in diameter; this areola goes on extending itself through the ninth, tenth and eleventh days, when it will have reached its maximum extent, which may be one or two inches from the vesicle in all directions. It is now a deep red color. The part over which the areola has spread is more or less elevated, the arm is considerably swollen and painful, and the adjacent glands more or less enlarged and tender to the touch. The extent of the enlargement of the gland adjacent to the vaccine vesicle,—the axillary gland, if the vesicle is upon the arm, the inguinal, if it is upon the thigh,—varies considerably in different persons.[1] In some it is very great, in others it is scarcely noticeable. The maximum degree of inflammation in the vesicle has now been attained, and there is a distinct infiltration of the tissues about it.

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[1] In obtaining vaccine virus for use, both the bovine and the humanized virus should be taken from the vesicle on the eighth day. The lymph should be taken from the vesicle before the inflammatory process has commenced which is to change it into a pustule. Jenner's "Golden Rule" was, any vesicle which manifests *an areola* must be discarded in the matter of withdrawing lymph. A few years ago it was the common practice in this city to use the vaccine crusts, but this practice has fallen almost entirely into disuse because of the great danger of thereby transmitting other diseases. I prefer bovine virus when it is possible to obtain it. If compelled to use the humanized virus, use the lymph. The vesicles must be punctured in such a manner that the lymph cannot be contaminated by the blood; this is best done by introducing the instrument parallel with the arm. The vesicle must be tapped in several places. The lymph which spontaneously flows from such a puncture can be preserved upon the convex surface of a piece of quill, and conveyed from one individual to another. Vaccine virus secured from the human arm in this manner is less liable than any other form of humanized virus to do permanent harm to the vaccinated individual.

On the twelfth or thirteenth day the pustule ruptures, and the contents escape. The rupture belongs to the natural course of the vaccine vesicles, and is independent of mechanical violence. From this time the inflamed areola becomes less and less distinct, and by the fourteenth or fifteenth day the crust has assumed a dark, brownish appearance, which goes on deepening until on the seventeenth day a deep-brown crust is formed having a central depression and no areola of inflammation. It may be attached to the surface only in one or two places, and can be readily removed. If permitted to remain, it usually falls off on the eighteenth to the twenty-first day. This is the course pursued by a perfect vaccine vesicle. The shape and size of the crust will correspond to the shape and size of the vesicle. If the eighth day a pustule is formed instead of a vesicle, it is evident that the regular development of the vesicle has been disturbed, and that it will not afford complete protection.

The inflammatory process around the vesicle is usually more active when the *bovine virus* is used, than when the *humanized virus* is introduced, and there is more constitutional disturbance. Ordinarily, during the development of the vaccine vesicle and pustule, there is but little constitutional disturbance; this is usually self-limiting, and not sufficiently severe to require treatment. In children, eruptions, transitory in character, are liable to occur about the eighth or tenth day. About the eighth or ninth day the person vaccinated may feel a little chilly, and have severe headache; in most cases there is a slight rise in temperature.

The regular course of the vaccine vesicle may be interfered with by the occurrence of an erysipelatous inflammation, and if such an inflammation does occur during the course of its development, it entirely destroys the protecting power of the vaccination. Again, if a large quantity of pus has been discharged, and healing of the ulcer does not take place for two or three months, it is probable that something besides genuine vaccine virus has been introduced into the arm, and that the vaccination is not protective. As I have already stated, the presence of a vesicular eruption upon the surface at the time vaccination is performed will interfere with its development, therefore I would advise never to vaccinate one who has an eczematous eruption upon any part of the body, unless he has been exposed to the contagion of small-pox, for it is very probable that the vaccination will not be a protective one. It is better never to vaccinate a person having any form of skin disease, especially if the eruption is vesicular in character. The best time for the first performance of vaccination is in infancy, between the third and fifth months.

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[1] The axillary swelling is sometimes so intense that abscess results.—*Quain's Dict.*

Revaccination should be performed after puberty, and always after or preceding a new exposure to the contagion of small-pox, for the period during which revaccination will afford complete protection is not the same in every individual. In some cases a single vaccination will afford complete protection for a lifetime. In other cases it is necessary to frequently repeat the vaccination, perhaps every two years, in order to secure the desired protection.[1]

VARIOLOID.

During every epidemic of small-pox there is a certain number of cases concerning which there will be doubt as to whether they are cases of variola or varioloid. Certain persons who have never been vaccinated may, through a naturally slight susceptibility to the infection of small-pox, have so mild a form of variola that it is difficult to distinguish it from varioloid.

Varioloid differs from small-pox in the rapid development and decline of the symptoms, in the small number of the pustules, and in the short time required for the formation and separation of the crusts. The entire period of the eruptive stage often does not last more than a week. Rarely are cicatrices or pits left after the eruption. In varioloid the period of incubation is about one-half as long as in variola, hence the onset of the graver disease may be anticipated by vaccinating one who is known to have been exposed, and who, otherwise, would go on and have the unmodified disease.

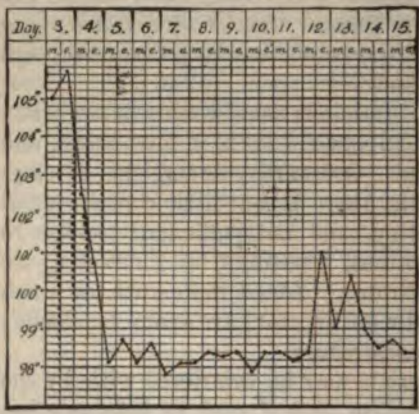

Fig. 167.
Temperature Record in a case of Varioloid.

In varioloid and variola the pustules pass through similar stages. We first have the small red spot, then vesicles form, often within twelve hours after the appearance of the erupiton. These vesicles rapidly increase in size ; sometimes they are umbilicated ; by the end of the third day their contents sometimes become purulent, without any tumefaction of the surrounding skin. Many vesicles abort; they do not become pustules. On the fifth day desiccation commences, which is often complete by the seventh day. The majority of the pustules simply dry up, without previously bursting, and form brown crusts which are thinner and smaller than those of variola. In varioloid there is no regular period of development as in variola. In variola there is the period of eruption, during which the vesicle is perfected; this is succeeded by the period of suppuration, then by desiccation, about fourteen days being required to complete the process; while in varioloid the course of the eruption is irregular, and is usually completed within one week. Secondary fever is slight or absent. Again, in varioloid there is but little constitutional disturbance after the appearance of the eruption. By the end of the first or commencement of the second day the temperature is usually normal. It resembles variola in the severity of the symptoms during the period of invasion, but as soon as the eruption appears there is an entire cessation of all the active febrile symptoms. During the period of invasion varioloid may be said very closely to resemble variola. When an unprotected individual is exposed to varioloid, the most severe confluent small-pox may be the result. This fact proves that varioloid is a modified form of small-pox. Varioloid is small-pox having a shorter duration and a milder course than usual.[1]

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[1] The best plan is to vaccinate at intervals until the individual *has four good scars.*

Prognosis.—Usually the prognosis is good. The rapidity with which the vesicles are developed, their shorter duration, the subsidence of the fever, and the appearance of the eruption, together with the usual duration of an attack, are sufficient to distinguish it from variola.

Treatment.—The treatment for varioloid is the same as for a mild or modified form of small-pox. The patient should be placed in a large, well-ventilated room, and quarantined the same as though suffering from variola. If the form of invasion is severe, saline cathartics may be administered. When delirium is present, and the pain in the back is very severe, the moderate use of opium is admissible. As soon as the eruptive period of varioloid is reached, no further treatment is required; the patient passes on to a rapid and complete convalescence.

CHICKEN-POX.

(*Varicella.*)

Varicella is an acute contagious febrile disease accompanied by a vesicular eruption, which chiefly affects children. It has been called "spurious variola," swine-pox, etc., etc.

Morbid Anatomy.—The only lesion of this disease is the eruption, which consists of small slightly elevated rose-spots, varying in number from twenty-five to two hundred, which in from ten to twenty-four hours become small vesicles with clear contents. They vary in size from a pin's head to a pea. They are usually discrete, but may run together and form bullæ three-fourths to two inches in diameter. They rest on a hyperæmic zone of skin. In many cases the areola is absent. As the vesicles enlarge, they become globular or ovoid in shape and their contents are translucent, glistening and opalescent, never acid as in sudamina. Sometimes the vesicles are divided into compartments. On the third day pustulation of a few vesicles may occur. On the fourth day the vesicles commence to dry up; on the sixth crusts are formed. One crop occupies rarely more than six days, and as a second crop appears or starts also on the second and third day of the first crop, the whole number of days of the eruption is from seven to nine.

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[1] It may be said that we modify small-pox by inoculation. We do not: we only modify its intensity. There is the same regular development of the disease after inoculation that we have in the ordinary form of small-pox; while by vaccination we not only lessen the severity of the disease but we are able to so modify the stages of its development as to shorten its duration.

According to the shape of the vesicles, varicella is called lenticular, globular, conoidal,[1] etc. Pitting rarely occurs; should cicatrices remain, they disappear in two years.

Etiology.—Opinions are still divided as to the identity of variola and varicella. Hebra claims that there is one poison for the two diseases. Senator, Thomas, and others regard it as a specific disease. It occurs sporadically and epidemically. Inoculation has given negative results. The period of incubation varies from eight to seventeen days.

Symptoms.—Twenty-four hours preceding the eruption there is usually lassitude and a feeling of malaise. The eruption appears first on the back or cheek, and then on the face or scalp. It spreads irregularly to the abdomen and extremities. About the second day vesicles may appear upon the tongue, lips, cheeks, palate, and on the mucous membrane of the genitals. On the second day after the first crop of the eruption a new crop appears, and in many cases there is a third crop on the following day. The temperature rarely rises over 100° or 101° F.

Differential Diagnosis.—The points of differential diagnosis between *varicella* and *variola* are as follows:—varicella runs rapidly through its stages; small-pox has three distinct periods—the papular, the vesicular, and the pustular. The eruption of varicella is complete by the third, while the eruption of variola is never complete until the ninth day. In both natural and modified small-pox prodromata occur before the eruption appears, and then the temperature falls; in varicella there are no prodromata, and a *rise* in temperature *follows* the eruption. Varicella spreads irregularly. Small-pox vesicles are umbilicated and multilocular; those of chicken-pox are globular or pointed, unicellular, and collapse on pressure. Small-pox is inoculable, varicella is not.[2] The stage of incubation is much longer in chicken-pox than in small-pox, and vaccination does not protect against it; and during its progress a child can be successfully vaccinated. It is very doubtful whether varicella ever attacks the same individual twice.

Prognosis.—The prognosis is always good.

Treatment.—The treatment is rest in bed, cleanliness, a non-stimulating diet, and cooling drinks.

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[1] Conoidal is also called swine-pox.

[2] Small-pox and vaccinia are often early followed in the same individual, say within two or three years, by chicken-pox, or vice versâ. Chicken-pox, vaccinia, and small-pox have been known to follow in immediate succession in the same individual.

SCARLET FEVER.

Scarlet fever or *scarlatina* is a contagious disease accompanied by an inflammation of the tegumentary investment of the entire body, both cutaneous and mucous. This name has been given on account of the bright red appearance of its eruptions. It is a disease of childhood, but may occur at any age.

Its development and course are divided into three periods: *first*, the *period of invasion*, which lasts from twenty-four to forty-eight hours; *second*, the *period of eruption*, which lasts from five to seven days; *third*, the *period of desquamation*, during which the entire epithelial surface is removed. Some classify the disease according to its severity; others according to the prominent organs of the body which are involved; others according to the prominent phenomena which attend its development. The more common classification, and certainly the simplest, is that which divides it into *scarlatina simplex*, *scarlatina anginosa*, and *scarlatina maligna*. I shall adopt this classification.

Morbid Anatomy.—It has no characteristic anatomical lesions, except those which occur in the skin and mucous membranes. The *eruption* is its distinguishing lesion; it makes its appearance on the second or third day after the commencement of the febrile symptoms. At that time it consists of very numerous and closely aggregated points about the size of a pin's head; between these the skin is of its natural color. In typical cases, these points are equally distributed over the entire body, except the face. These red spots are usually circular in shape, slightly elevated above the surrounding skin, and so close to each other that they give a confluent redness to the entire surface. In mild cases the red points remain isolated, and do not become confluent; as the eruption develops, these red points unite. In severe cases the skin becomes turgid and swollen, and presents a uniformly red and glistening appearance. In malignant cases the hyperæmia of the skin is often accompanied by more or less extensive hemorrhages, causing petechiæ and extensive ecchymosis. The redness of the eruption gradually increases up to a certain point, which is not the same in all cases, then remains unchanged for twelve or twenty-four hours, after which time the redness slowly passes away. During the course of the disease, the color often changes with the exacerbations and remissions of the fever. As a rule, the degree of redness depends upon the intensity of the fever, and may vary from a *pale red* to a *deep scarlet*. If the respiration becomes impeded, the eruption assumes a bluish-red hue. During the first forty-eight hours after the appearance of the eruption, when the respiration is unimpeded, the redness completely disappears under firm pressure, and reappears as soon as the pressure is removed. After this period, the pressed point does not entirely lose its red color. In a certain proportion of cases, the eruption only appears in spots on the surface of the body, on the trunk, or face, or about the flexor surfaces of the joints. When it only appears on the face, the diagnosis is difficult.

In addition to the cutaneous hyperæmia which gives the redness to the surface, there is more or less serous and lymphoid exudation into the "rete Malpighii," which is followed, on the decline of the redness of the surface, by an abundant epidermic exfoliation. Blood extravasations into the sweat-glands often occur. The exfoliation marks the period of desquamation, which may immediately follow the decline of the redness or may be delayed a few days. This is due to an excessive production of newly-formed epidermis, and the process may last only a few days, or if the eruption is abundant it may continue for several weeks, and may recur a second time on the same surface. After the desquamation has ceased, it does not reappear, except in cases of relapse; these are followed by renewed and sometimes by a very complete desquamation. Desquamation has not infrequently occurred on skin that has *never been* the seat of the eruption.

In connection with these cutaneous changes the scarlatina poison causes changes in the mucous membrane of the mouth and throat, the most frequent of which is catarrhal pharyngitis, which at first gives to the mucous surface of the tonsils and pharynx a red, swollen, and dry appearance. After a little time, these mucous surfaces become covered with a tenacious mucus. Upon the reddened mucous membrane small elevations arise, like the smaller follicles in an ordinary catarrh. In mild cases, all these changes disappear in a few days; in the severer cases, the mucous surface assumes a dark, livid color, the parts become more or less œdematous, and are covered by an abundant secretion. Follicular ulcers also form. The œdema may be so extensive as to render deglutition difficult; the tonsils are often so swollen that they touch each other.

Besides the redness and œdema of the mucous membrane of the mouth and throat, there is often inflammation of the parotid and sublingual glands as well as of the connective-tissue of the neck. This glandular inflammation may end in resolution, but often it terminates in suppurative or diffused necrosis. It may give rise to extensive gangrene of the tonsils and adjacent soft parts; sometimes it is followed by extensive abscesses and destruction of the cellular tissue about the neck; the skin in the region may slough, and not infrequently fatal hemorrhage results from the destruction of small vessels; or the whole region may lie open as if dissected out.

Diphtheria is so often a complication of scarlatina anginosa, that it has been assumed that there is some necessary relation between the two diseases.[1] Yet diphtheria is as frequently met with in the mild as in the severe types of scarlatina, and occurs in every stage of the disease; it is often present during the period of incubation, so that the symptoms of the two diseases appear simultaneously. Again, it is met with during the period of convalescence. In some instances, scarlatina seems to complicate diphtheria.

In a mild form of scarlet fever, when the disease runs a regular course, the nasal mucous membrane is usually pale, and its secretion is not increased. When the disease is severe, the nasal mucous membrane becomes secondarily, never primarily, involved. This is the result of a catarrhal affection of the throat. It is a purulent catarrh of the posterior nares, which gradually extends to the anterior nares, and gives rise to a very troublesome form of coryza. During the eruptive period of scarlatina, affections of the ear frequently occur in connection with those of the throat. Usually these have their seat in the middle ear, pus being the product. They are always tedious and may become chronic. The eye may be involved; keratitis and ulcers are not uncommon.

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[1] Hubner states that the pseudo membranes are much thinner in scarlatinal than in ordinary diphtheria, and that in the former, fibrin is found between the epithelia and in the mucous and submucous connective tissue.

Next to the skin and mucous surfaces, the kidneys are the organs most frequently affected in this disease. There is no question but that, in a certain proportion of cases, recovery takes place without any kidney lesions; but these are the exceptions and not the rule. In some epidemics the scarlatina poison induces a so-called "croupous" inflammation of the uriniferous tubules. The tubules of the cortical substance of the kidneys are most extensively affected; the morbid processes commencing at the Malpighian tufts[1] follow the course of the convoluted tubules. If the tubules are only slightly affected there will be no symptoms except a slight albuminuria. The kidney changes are rarely well marked before the second or third week of the disease, and usually terminate in complete recovery. The character and extent of these kidney changes vary in different epidemics. During some epidemics, the kidney changes are slight; during other epidemics almost every case, whether mild or severe, will be attended by extensive kidney lesions.

At the post-mortem examination of one who has died of scarlet fever, there will be found more or less extensive congestion of the internal organs, the brain, liver, spleen, etc., but these congestions do not differ from those met with in other acute infectious diseases. The changes in the constituents of the blood are such as to diminish its coagulating power. The Peyerian patches will often be found presenting the "shaven-beard appearance." There may be parenchymatous degeneration of the gastric tubules.

Etiology.—The *cause* of scarlet fever is a contagion, which is transferable from the sick to the healthy. No specific microbe of the disease has as yet been discovered. It has been claimed that sporadic cases do occasionally occur; but there is little doubt that if the history of every case of supposed spontaneous scarlet fever could be carefully taken, it would be found that at no place and at no time had the disease ever been of spontaneous origin. It may be conveyed directly from the affected to the healthy by contact, through the atmosphere and by clothing which has been thoroughly saturated with the scarlet fever poison; therefore it may be considered a portable disease. Animals that have been around those sick with scarlet fever may convey it. I recall an instance in which the scarlet fever poison was conveyed in this way:—For a number of days a little dog had been around children sick with scarlet fever, and by a single visit of the dog to the children of another family the disease was conveyed. There has been considerable discussion as to whether the disease can or cannot be conveyed in milk. This is possible.[1]

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[1] There is proliferation of epithelial nuclei in the glomeruli, distending them to twice their size, and thus compressing the vascular tuft. There is hyaline degeneration of the capillaries (Klein).

[2] Micrococci are found in the blood.

The infection of scarlatina is not so certain as that of measles or small-pox. When one member of a family is sick with measles, usually every other member of that family who has not had measles will contract the disease; whereas one member of a family may be sick with scarlet fever and every other member may escape. Some seem to have a certain idiosyncrasy, so that when they are brought in contact with the poison of scarlet fever they do not contract the disease. The poison which they receive into the system has power to produce some of the *symptoms* but has not power to fully develop the disease.

Scarlet fever can be communicated from one individual to another by inoculation. If some of the watery material or serum that can be obtained from the minute vesicles occasionally seen upon the surface of the body in connection with the scarlet fever eruption, be taken and introduced into the body of an individual who has not had scarlet fever, it will develop the disease. It has been proposed to inoculate those who have not had scarlet fever in the same manner as one would inoculate those who have not had small-pox, and, by so doing, produce a modification of the disease. But it has been found by experiment that those who have been inoculated for scarlet fever have suffered *more severely* than those who contracted the disease by any of the common methods of contagion. There is no question but that the scarlet fever poison can also be introduced into the system through the respired air, but whether it can be taken into the system through the medium of food or fluids is still an unsettled question.

A question of great practical importance is: if the disease can be conveyed by clothing, is it safe for a physician to visit patients sick with scarlet fever, and go from them directly to those who have not had the disease? Unquestionably it is possible to so convey the disease, but in my own experience I know of no case where it has been so conveyed. The clothing in order to be sufficiently impregnated with the poison to render it a means of contagion must be longer exposed than is the case when a physician makes a visit of ordinary length. Unquestionably, nurses who have been with a scarlet fever patient for a number of days, and whose clothing has become filled with the poison, may carry the disease. These should change their clothing before they go from the sick to the healthy. The real nature of the scarlatina poison is undetermined. The period at which this disease is most infectious is probably the desquamative period, although some maintain that it is most infectious during the eruptive period.

An individual is almost certain never to have a second attack.

The period of incubation varies from two to ten days, the average duration being from three to five. It may be only three hours. Age has a great influence on individual predisposition. The greatest susceptibility to the influence of the poison exists between the second and seventh years;

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[1] Quain says: "Milk is a great medium for carrying scarlet fever, and *cream*, even more than milk, often carries it from sick to well."

it rapidly diminishes after the ninth year, so that adults, and especially the aged, have only a slight predisposition to the infection. Those who have just undergone surgical operations seem to be especially prone to contract the disease. Scarlet fever may be endemic or epidemic. No reason can be assigned for its variations in type or severity. For years the type of fever which appears in a given locality will be exceedingly mild in character, when suddenly, without any assignable cause, a most malignant epidemic will prevail. Usually epidemics of scarlatina prevail in the autumn and spring.[1]

Symptoms.—The symptoms of scarlet fever vary with the type and with the severity of the fever. In moderately severe cases, before the appearance of the eruption, the patient will have a more or less severe headache, pain in the back and limbs, and at first coldness of the surface. Epistaxis is not rare. In some cases rigors will occur, and perhaps distinct chills. In children convulsions and coma often occur. These ushering-in symptoms are immediately followed by a sensation of intense heat, with great acceleration of the pulse, which at this time often beats 120 or 130 per minute. There will also be nausea and vomiting, frequently most persistent and distressing. Besides, there will be a rapid rise in temperature. It may reach 103° F. or 104° F., within a few hours.

Within a period lasting from twelve to forty-eight hours, the average being thirty-six hours, the eruption makes its appearance, and the fever increases. The elevation in temperature is accompanied by restlessness, a burning sensation, perhaps delirium; the nausea and vomiting become more urgent, and now the papillæ of the tongue become swollen, and the organ presents the appearance of a strawberry :—(the "strawberry tongue" of scarlet fever). This appearance is not commonly seen in the milder cases, but, as a rule, is present in all the severer cases. With the appearance of the eruption, all the symptoms, perhaps excepting the pain in the head, increase in severity. The urine, if it has been scanty, will now become more so, and may be nearly suppressed; if it has been sufficiently abundant, not infrequently, as the eruption makes its appearance, it becomes scanty and high-colored. In some cases the disease is so mild that there is but little disturbance, except that caused by the eruption, the temperature being not over 102° F. In other cases the disease is ushered in by violent nervous symptoms, such as delirium and coma, accompanied by extreme exhaustion, and the patient dies before the eruption makes its appearance. In other words, the patient dies during the period of invasion, from the overwhelming of the nervous system with the scarlet fever poison.

During the earlier stages of the disease the throat symptoms are quite characteristic. Adults and older children complain of a pricking sensation in the throat, and difficulty in deglutition ; the tonsils, uvula, and posterior wall of the pharynx are red and œdematous, and from their appearance, with the attendant symptoms, in most instances, one is able to very early decide that the case is one of commencing scarlatina. There are cases in which the throat symptoms are altogether absent at first, and do not come on until later in the disease. The symptoms which mark the development of this disease remain to be studied in detail.

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[1] Trojanowsky and Thomas describe a variety called "recurrent," where two series of eruptions overlap, as it were, and finally merge into one attack. But the latter's cases all occurring in marshy districts, he inclines to the view that the poisons of malaria and scarlatina were combined and perhaps modified by such an union.

As already stated, the whole course of scarlet fever may conveniently be divided into three stages. *First*, the stage of invasion, or the febrile stage. *Second*, the stage of eruption. *Third*, the stage of desquamation.

The duration of the stage of invasion varies with the type of the disease. In most cases, it is from twelve to twenty-four hours; it may be four or five days. Usually the onset is marked by chilliness and slight rigors, followed by a rapid rise in temperature. The skin becomes dry, the face flushed, and the pulse accelerated. At the same time there is slight soreness of the throat, the face appears red and dry, the neck is stiff, the eyes suffused, and there is some tenderness about the joints. Vomiting and thirst are prominent symptoms. The tongue is red at its tip and edges, the papillæ are enlarged, and it presents the so-called strawberry appearance. Lassitude, pain in the head, aching of the limbs, and restlessness are generally present. There may be some delirium at night. Twenty-four hours after commencement of the fever of invasion, the eruption may make its appearance. The period which elapses between the exposure and the appearance of the eruption varies. In some cases the eruption is said to have appeared as early as twenty-four hours after exposure, while in others one or two weeks have elapsed after the exposure before the disease was developed. No definite statement in regard to the duration of the period between the exposure and the appearance of the eruption can be made. The eruption first makes its appearance upon the neck and upper portion of the chest, and is first seen as little red dots, varying in size from a line to a line and a half in diameter. These gradually coalesce and the eruption extends over the entire surface of the body, perhaps on the face, and lastly it appears on the lower extremities. It presents its brightest appearance upon the evening of the fourth day. After the second day of the eruption, if not before, the entire surface will present a uniform redness, the color varying with the severity of the disease.

In the milder cases one will have a bright rose-red eruption or rash, while in the severer types the eruption will assume an appearance resembling the deep-red color of the boiled lobster. The darker the eruption, the more severe the form of the disease and the greater the danger. When the eruption is fully developed, it will be noticed that the surface is somewhat elevated, the parts present a swollen appearance, the vessels of the skin seem to be congested, and there will be soreness of the throat more marked than in the febrile stage. Miliaria appear when the rash is most intense, and sudamina are common.

Usually, vomiting is present at the commencement of the disease, but becomes more severe and a more marked symptom as the stage of eruption is ushered in; if not present at the commencement, it is certain to make its appearance with the appearance of the eruption. The vomiting is peculiar, not on account of the matters ejected, but the *act* of vomiting is projectile in character.

In scarlatina the condition of the throat depends upon the severity of the disease. In some cases there is simply a blush of redness over the posterior portion of the pharynx and uvula and anterior pillars of the soft palate. In other cases a general tumefaction and œdema of all the soft parts of the throat will be seen, and the tonsils will be the seat of a more or less intense parenchymatous inflammation, which gives rise to a swelling that encroaches more or less upon the pharynx. Again, ulcerative pharyngitis will occur, or upon the surface of the enlarged tonsils and swollen mucous membrane of the pharynx there may be an exudation, which will be more fully described hereafter.

In this, the ordinary form of scarlatina, when it runs its ordinary course, there will not be much swelling of the glands about the neck, nor very much tumefaction of the soft tissues in the pharynx. On the morning of the fourth day, if the finger-end is drawn across the surface, a clear, well-defined line will be made, which will remain for some time. This distinct white line is a point of some importance in distinguishing scarlatina from roseola. Usually the eruption begins to fade upon the fourth day, and by the sixth day it has entirely disappeared, and desquamation has commenced. During the time the eruption is developing, the temperature continues to rise until perhaps it has reached 106° F. or 107° F. In the meantime the pulse may increase to 120 or even 140, or perhaps 150 beats per minute, and not infrequently there is some delirium during this stage; there may be also more or less stupor. There is an intense itching and burning upon the surface, and a great restlessness.

Between the fifth and eighth days of the eruption, the temperature begins to decline, and at the same time the eruption fades. This fading of the eruption goes on rapidly, so that by the end of the eighth, certainly early on the ninth day, sometimes as early as the sixth day, it is no longer visible. With the disappearance of the rash, desquamation commences, and with this there will be a still more marked fall in temperature, and diminished frequency of the pulse. All the febrile symptoms disappear, all the throat symptoms subside, there is no longer any difficulty in deglutition, there is no more pain in the throat, no more swelling of the external glands, if previously it had existed.

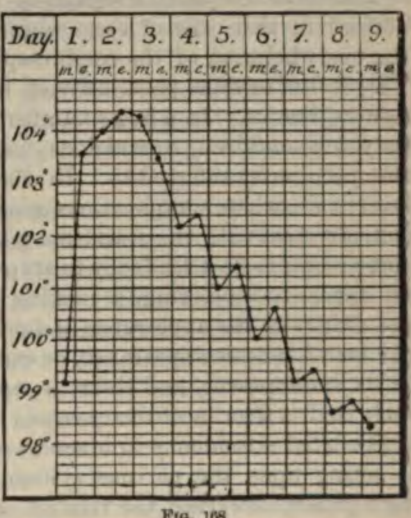

FIG. 168.
Temperature Record in a case of Scarlatina.

The period of desquamation lasts about two weeks, during which time there is the greatest danger of communicating the disease. At the end of that period, if no complication occur, the patient is well. The fine scales which are so abundantly thrown off contain the specific poison, and they are so delicate that they are blown about with every breath, and carried in every current of air, and are in the most favorable condition to be taken into the system in the respired air. Some have maintained that the contagious period in this disease does not occur until the period of desquamation. This statement is not sustained by clinical facts. The amount of the desquamation depends upon the intensity of the eruption. The skin has a dry feel before desquamation commences. Where the skin is thin the epidermis comes off in thin scales, "branny" desquamation. Where the skin is thick, as on the palms of the hands and the soles of the feet, it peels off in extensive patches, "scaly" desquamation. With the desquamation, the fever subsides more or less rapidly. The entire period occupied by a case of scarlet fever, when it runs its regular course, is from two to three weeks.

Scarlet fever is liable to *irregularities* which it is important to consider. It is claimed by some that these irregularities depend upon the organ or set of organs primarily affected by the scarlet fever poison. They are rather due to some peculiarity in the type of the disease, to the degree of poisoning, and in some instances to the particular set of organs that are involved in the different epidemics. In some epidemics even milder forms of the disease than have been described are seen. The attack may be so mild, and there may be so little fever that if the eruption was not present one would not be able to recognize the scarlet fever; and even that may be so light that the stage of eruption and the stage of desquamation may pass unnoticed, and one may be scarcely able to decide whether the patient has or has not had an attack of scarlet fever.

The most frequent irregularity in the manifestation of the disease is noticed in that class of cases where we have complications resulting from the overwhelming of the cerebro-spinal system with the scarlatina poison. This is due to some peculiarity of the poison, and is characteristic of certain epidemics. In a large number of cases in the febrile stage, especially in young children, convulsions may occur, but they do not depend upon the peculiarity referred to. In the class of cases to which reference has been made, where complications arise from the overwhelming of the cerebro-spinal system with the scarlatina poison, from the very onset of the disease there seems to be a tendency to stupor and delirium, a peculiar restlessness, an apparent wandering, a picking at the bed-clothes, accompanied by a peculiarity in the appearance of the eruption, which may cause it to assume the boiled-lobster appearance, or even a darker hue. The eruption is slow in its development, and there is not that uniform redness over the entire body that is seen in ordinary cases; it appears in patches, and with it there is exhibited a tendency to blueness of the finger-ends, indicating that there is acting upon the nervous system a poison which possesses the power of very greatly lowering the vitality of the patient. These symptoms predominate in some epidemics.

There is a class of cases in which there is not much swelling of the throat, nor is the pulse more frequent than 130 or 140 per minute, but during the second day of the eruption the temperature ranges very high, reaching 107° or 108° F., and then the pulse becomes intermittent. Under such circumstances the disturbance of the nervous system is due to the high temperature which may have been present for two or three days; these disturbances may be prevented if the temperature is not allowed to rise above 103° or 104° F.

Again, in cases where there is marked swelling of the throat, and a general infiltration of the tissues and glands of the neck, the development of the nervous phenomena is due to an interference with the return circulation. The condition which gives rise to the cerebral symptoms is one of mechanical cerebral congestion.

There is still another class of cases in which the marked nervous phenomena appear still later in the course of the disease. Under such circumstances they often indicate a typhoid condition. This typhoid condition is not induced, nor are the nervous phenomena developed, on account of the peculiar effect produced upon the nerve centres by the scarlet fever poison, nor are they due to the effects produced by a high temperature, nor by an interference with the return circulation, but they are due to septic poisoning, a poisoning entirely different from scarlet fever poisoning. The nervous phenomena develop after the eruption. During the developing period, there may be noticed a peculiar ichorous discharge from the nostrils, and frequently it is said that the patient has become repoisoned by scarlet fever poison—but this is not the case; he has become repoisoned by the septic element of these discharges. During the period of desquamation the nervous system may be involved in consequence of the presence of uræmic poisoning.

The mere terms, scarlatina simplex, scarlatina anginosa, and scarlatina maligna, do not indicate all that may be embraced under each of the divisions.

Scarlatina maligna is that form of the disease in which the cerebrospinal system becomes early involved. What the changes are that produce these nervous phenomena, when high temperature is present, is still an unsettled question.

Again, scarlet fever may run an irregular course in those cases in which there is present an extensive infiltration of the tissue of the neck, with inflammatory products, swelling of the glands, and extensive suppuration. Not infrequently these cases terminate fatally; doubtless in some cases the extensive suppuration in the areolar tissue about the neck produces this result, and in other cases it is produced by the interference with respiration caused by enlargement of the glands and swelling of the tissues of the neck. Exhaustion from sloughing is a cause of death. In these cases there is danger from œdema glottidis, the consequence of extension of the inflammation from the adjacent tissues.

There are cases in which the eruption is not very well marked; the patient passes safely through the stage of eruption, and the stage of desquamation is fully established; but, instead of making a good recovery from this point, immense abscesses are rapidly developed in the cervical region, blood-changes begin to manifest themselves—changes that favor hemorrhages. Hemorrhages are then petechial in character and occur on the mucous surfaces, and the patient passes into a typhoid condition, with hemorrhages occurring from the nose, mouth, intestines, etc., and death ensues. Such a result is produced by the peculiar action of the septic poison developed during the suppurative process, and perhaps from outside influences (as bad hygiene).

I regard scarlatinal coryza, in which the discharge contains elements capable of producing septic poisoning, as an unfavorable symptom. The clear serum which runs over the lip never causes death ; but the fact that it sometimes produces excoriation and ulceration of the tissues with which it comes in contact, indicates that there are nasal and pharyngeal changes which may destroy life ; especially is this the case in young children. Sloughing ulcers sometimes develop in the mouth and throat ; and when they do occur, the patient is said to have ulcerative stomatitis ; but these ulcerations are really due to a peculiarity of the scarlatina poison. Under such circumstances the patient may go on through the period of eruption, enter the stage of desquamation, and then rapidly sink and die, with symptoms similar to those which attend diphtheria. Although the odor of the breath may very closely resemble that noticed in some cases of diphtheria, there is no diphtheritic exudation present.

Scarlatina may also run an irregular course by the development of inflammation of the internal ear. This inflammation extends from the throat up the Eustachian tube, involves the middle ear, and gives rise to a train of symptoms, such as intense pain, delirium, and rolling of the head, all of which suggest the presence of acute meningitis. I recall several instances in which the diagnosis of acute meningitis was made, where from the after-history of the case there was no question but that the symptoms were due to such an inflammation of the middle and internal ear.

Complications and Sequelæ.—The most common sequela is anasarca. The anasarca of scarlatina usually appears at the time the patient is convalescing, during the period of desquamation, or just as desquamation is being completed. It has been thought that anasarca is due to some exposure to the influence of cold during this period. It is possible that the changes in the kidney which give rise to the anasarca may sometimes be produced by the influence of cold, and undoubtedly anasarca is occasionally developed in this manner; but in the majority of cases it is due to some peculiarity in the scarlet fever poison, or to some peculiar atmospherical condition. During some years anasarca is a very common sequela of scarlet fever ; while during other years in equally severe cases, scarcely a case of anasarca occurs. While we recognize the fact that it is possible for kidney lesions to be developed which shall give rise to anasarca in consequence of exposure to cold, it is also of importance that we recognize the fact that the lesions and the anasarca may be developed independent of such exposure. The anasarca first shows itself on the face, and from the face it extends over the entire body, and if it becomes general more or less ascites is developed.

In most cases, at the time of, or previous to, the occurrence of the anasarca, certain premonitory symptoms occur, and it is of great importance to be familiar with these symptoms, and be on the watch for their appearance. For two or three days previous to their development a certain restlessness will be noticed, with nausea and vomiting. These symptoms are almost universally present. The nausea and vomiting so commonly present during the early periods of the disease have subsided, and now, during the period of desquamation, or perhaps after it has been completed, the vomiting returns. The patient has some pain in the head, has loss of appetite, is annoyed by the light, does not sleep well, and the temperature is raised perhaps two or three degrees. But the pulse now grows remarkably slow :—50 to 60 in children. When a patient complains in this manner during the desquamative stage of scarlet fever, our suspicions should be aroused, and if the urine has not yet been examined, an examination should be made at once.

The *urine* may be entirely suppressed for a day, and then it will usually be found scanty and high-colored, will contain albumen and casts. Hæmaturia and hæmoglobinuria both occur quite frequently. If previous examinations of the urine have been made before the development of these symptoms, a cloudiness will have been noticed (non-albuminous) due to epithelia and hyaline material, but now there are present casts which indicate the existence of scarlatinal nephritis. After the anasarca has been present two or three days, and the abdomen has become tense, swollen and painful, if the case is to have a favorable termination it will begin to decline, will be less and less marked about the face and feet, the tendency to stupor which has accompanied it will begin to disappear ; and as the dropsy subsides, and the patient is not so lethargic, the appetite begins to return, the urine increases in quantity, the albumen diminishes, the casts disappear, and convalescence is fully established. Anasarca may have been developed, all the symptoms have disappeared, and the patient have recovered within two weeks from the commencement of the attack. If, however, after the anasarca is developed, the case is to go on to an unfavorable termination, the anasarca instead of diminishing will increase ; the face will become more and more puffy, the legs more and more œdematous, the abdomen more and more distended, the pulse more and more frequent and feeble, the temperature more and more elevated, until a condition of coma is finally reached, which condition is sometimes preceded by convulsions, and followed by death.

Another sequela of scarlatina is *inflammation of the serous membranes.* The serous membrane most liable to be involved is the endocardium, and this inflammation may pass unrecognized unless its occurrence is closely watched, and there may be no rational symptoms present. Endocarditis, when it does occur, is liable to be *ulcerative* in character. Inflammation of the pericardium may occur as a complication of scarlet fever, but it does so much less frequently than inflammation of the endocardium. Inflammation of the pleura, and occasionally inflammation of the peritoneum, is met with as a sequela of this disease. If peritonitis does occur it is usually subacute in character. It is possible to have peritonitis developed as a sequela to scarlet fever and to be entirely recovered from. Rheumatism may be developed during the desquamative period of scarlet fever. Under such circumstances it assumes the ordinary appearances of inflammatory rheumatism. It is not a serious sequela, and a complete recovery usually occurs within ten or fourteen days from the commencement of the attack. Suppurative inflammation of the joints is sometimes a sequela of scarlet fever.

Another serious complication of scarlet fever is diphtheria. It may occur at any period of the fever; usually it occurs during the period of desquamation. There is developed the characteristic exudation of the disease, with the attendant depression noticed in a case of diphtheria developed independently of scarlet fever. It differs in no respect from primary diphtheria, except in the rapidity of its development and in its fatality. In scarlet fever there is no more serious complication. Usually it appears quite suddenly, and perhaps does not occur more frequently in those who have a severe form of the disease than in those who have a mild scarlet fever. The *lymphatic glands* may be enlarged and swollen, *i. e.*, a lymphadenitis may be a sequel of scarlatina. Keratitis, retinitis, and total blindness are rare sequelæ of scarlet fever. Anæmia, paralysis of single nerves, spinal disease, chronic Bright's, deafness, chorea, epilepsy (melancholia and mania in adults), valvular diseases, etc., are also named as sequelæ.

Differential Diagnosis.—The diagnosis of scarlet fever is usually not difficult after the eruption has made its appearance, for, in well-marked cases, that alone will readily distinguish it from other eruptive fevers. At the very onset of the eruption, and sometimes in *irregular* cases, the differential diagnosis is difficult. The eruptive diseases which are most liable to be mistaken for scarlet fever are *measles*, *small-pox*, *roseola*, and an *erythema* which sometimes appears in surgical cases. In all doubtful cases a careful study of the history of the patient is necessary before making a diagnosis.

In *measles* the appearance of the eruption is preceded by a cough and coryza. These symptoms are never present in the ushering-in stage of scarlatina, but may *follow* the eruption. Besides, the eruption of measles first appears on the face, whereas the eruption of scarlet fever first makes its appearance upon the neck and chest. The incubation period is shorter in scarlet fever and the early pyrexia is higher than in measles. After these diseases are once fully developed, the course of the one so differs from that of the other that there will rarely be any chance for doubt after the first week of the disease. The minute punctate appearance of the scarlatina eruption before it becomes confluent is an important element in its diagnosis.

Although the eruption of *confluent variola*, for the first twenty-four hours, may sometimes resemble that of scarlatina, yet the development of the first vesicle settles the question.

The appearance of *erythema* bears a close resemblance to a perfectly developed scarlatina eruption; it is not, however, present on the extremities, neck, and portions of the trunk, and it spreads in a very irregular manner, whereas in scarlatina such is not the case. But if, on account of the scantiness of the scarlatina eruption, any doubt arises as to the nature of the eruption, the fact that in scarlatina the throat symptoms are rarely absent, that the tongue presents the strawberry appearance, and that at an early period there is usually some swelling of the cervical glands, will decide the case. In those cases in which, during the early part of the disease, it is impossible to make a differential diagnosis, the diagnosis will be readily made when the period of desquamation is reached.

The differential diagnosis between *roseola* and a very mild form of scarlatina is sometimes attended with great difficulty. If scarlatina is prevailing, and a child has an eruption which lasts for two or three days, then disappears, and is not followed by desquamation, it may be thought that the case is one of scarlatina; and yet the sequel proves that the case was one of roseola. Such a form of roseola sometimes prevails epidemically, and attacks children in a certain locality, whether they have or have not had scarlatina. Under such circumstances, adults and children are said to have had a second attack of scarlet fever. In making a differential diagnosis between this form of roseola and scarlatina, the duration of the eruption and the character of the throat symptoms must decide. In scarlatina the posterior part of the pharynx is affected, while in roseola the redness is confined to the anterior portion; besides, the throat affection in roseola is much milder than in scarlatina. In roseola the white line that the finger leaves disappears immediately; while in scarlatina it remains—indeed, a letter may be traced on the skin in well-marked cases.

One can hardly mistake *erysipelas* for scarlatina, for erysipelas commences at one point and gradually extends from it; there is also marked œdema of the connective-tissue, and there is a very marked difference in the constitutional symptoms of the two diseases.

Malignant cases of scarlet fever, in which no eruption appears, prove rapidly fatal. In such cases, the fact that an epidemic of scarlet fever is prevailing (which is usually the case), the rapid development of the disease, the very high range of temperature, and the very grave nervous phenomena, will aid in the diagnosis, and these can only be accounted for on the ground that the patient is overwhelmed by some very active blood-poison. In this class of cases the entire surface of the body should frequently be examined, for the eruption is sometimes very transient, perhaps appearing only for a few hours on the neck or extremities.

It is sometimes difficult to draw the line of distinction between scarlatina without an eruption, but with swelling of the cervical glands and ulceration of the throat, and *diphtheria*. If a patient has swelling of the cervical glands and well-marked febrile symptoms, which have come on gradually—that is, have been two or three days developing—and yet no scarlatina eruption has appeared, but a gangrenous ulceration has developed, involving the tonsils, the posterior wall of the pharynx, and the anterior pillar of the soft palate, and if scarlet fever is prevailing in the locality, it is very difficult to decide between it and diphtheria. There can be no doubt but that scarlatina poison may excite a tubular nephritis without an eruption appearing on the surface of the body, or without any of the other ordinary symptoms of scarlatina.

Prognosis.—The prognosis in scarlet fever is always uncertain. It will be influenced more by the character of the prevailing epidemic than by any other circumstance. According to statistics, the rate of mortality ranges from one death in five to one in twenty. Some epidemics are very mild. During one epidemic, in one month, I treated fifty cases of scarlet fever, with only two deaths. During the same month of the following year, I treated twenty cases, with seven deaths. In giving a prognosis one must always take into account the type of the prevailing disease. Even when the disease is mild in character, and is running a perfectly regular course, dangerous symptoms may suddenly arise without any assignable cause.

The conditions of a favorable prognosis are as follows: when the eruption appears within forty-eight hours from the commencement of the attack, and rapidly completes its course, reaching its maximum on the second day; when the throat symptoms are mild, little difficulty being experienced in swallowing; when the cervical glands are but slightly enlarged; when the temperature does not rise higher than 104° F., and the pulse beats only 120 per minute; when the cerebral symptoms are not severe, and are of short duration; and when the disappearance of the eruption is attended by a steady decline in temperature. Even if there is a slight affection of the joints and a moderately severe nephritis during the period of desquamation, a favorable termination may be predicted. The nephritic symptoms will almost always entirely disappear during the third or fourth week.

The conditions for an unfavorable prognosis are an irregular course; a temperature rising above 105° F.,[1] with dyspnœa and extreme frequency of the pulse; symptoms of collapse attended by a cold surface and a small pulse, an eruption of a livid hue, and abundant hemorrhages in the skin; ulcerative pharyngitis, especially when it extends to the nasal passages, accompanied by copious coryza and infiltration of the glands and tissues of the neck; severe nervous symptoms, with typhoid symptoms and long continued vomiting with diarrhœa coming on at the commencement of the attack; early nephritic symptoms and general dropsy, excessive hæmaturia, or almost complete suppression of urine, with high temperature. The occurrence of any of the more serious complications, such as pneumonia, diphtheria, pericarditis, œdema glottidis, etc., always renders the prognosis bad.

Before making a prognosis, decide whether the scarlet fever is regular or irregular in its course, and if irregular, what are the causes of the irregularity. It is also important to determine the patient's power of resisting disease. Autumn is the most unfavorable season. Favorable hygienic surroundings, good nursing, and well-directed medical treatment will greatly lessen the death rate in scarlet fever epidemics, and these should be considered elements of the prognosis. Patients with scarlet fever do better when left to themselves than when badly nursed, even if under the care of skilful medical attendants.

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[1] A temperature of 110° has been reached and yet followed by recovery; it rose to 115° F. in a fatal case. Scarlet fever is an intensely febrile disease; hence the temperature is not such a very important element in the prognosis.

Age is an important element of prognosis. The period of greatest mortality is from infancy to five years of age. Beyond this period until adult life, the prognosis is decidedly better. Five per cent. of the whole mortality falls in the first year, fifteen per cent. in the second, twenty per cent. in each of the next two years, and then decreases progressively. In adults, the mortality is greatest in pregnant women, and those who are suffering from some organic disease, especially some disease of the heart or kidneys.

Treatment.—In connection with the treatment of this affection, the first question that presents itself relates to *prophylaxis* or *prevention.*

The *prophylaxis* of scarlet fever is a system of the strictest quarantine. The sick must be removed from the healthy. All useless articles of furniture must be removed from the sick-room. Fresh air renders the contagion of scarlet fever less powerful; therefore, free ventilation is of the utmost importance. All the clothes and excretions of the patient should be disinfected in the same manner as in typhoid fever. To prevent the dissemination of the dusty particles of the desquamating epidermis, during the period of desquamation the surface of the body should be frequently sponged, and after each sponging the surface should be rubbed with olive oil. Those convalescing from this disease should not be allowed to leave their apartment until desquamation is completed, which usually requires at least three weeks after the commencement of the period of desquamation. The sick-room and everything which has been used about the patient should be thoroughly disinfected, and the windows and doors of the apartment should be allowed to remain open for a long time before it is again occupied.

To prevent the spread of the disease, nurses and attendants upon the sick should not be allowed to have any intercourse with the healthy until the period of desquamation is passed, and after that time not until there has been thorough cleaning and disinfecting. The funeral of those dying of scarlet fever should not be public. There is no known prophylactic treatment, except isolation, and a thorough disinfection of everything contaminated by the contagion.

A theory has been advanced that belladonna has power to prevent the development of this disease in those who have been exposed to its contagious influence. This drug has been very extensively administered in order to test its effects as a preventative in scarlet fever. After having carefully examined the subject, both in its literature and clinically, I am convinced that belladonna has no power to prevent the development, or mitigate the severity, of the fever in those who have been exposed to its infection. Fresh air is the only agent which can render the contagious influence of this fever less powerful.

Medicinal Treatment.—The medicinal treatment of scarlet fever is almost entirely expectant. It is a disease which cannot be aborted, and if left to its natural course tends to recovery if the fever and the local symptoms remain within certain bounds. It has certain stages to pass through, and one cannot safely interfere with its regular course. To stand by and watch, and, as far as possible, to guard against complications are the physician's chief duties. There are certain details which it is important to attend to. The bed and body linen should be frequently changed. As soon as the period of desquamation has been reached the patient should have a warm bath once or twice during the day, the surface of the body being well washed with carbolized soap. The baths hasten the process of desquamation and aid in bringing the skin into a healthy condition as rapidly as possible; the kidneys will also be relieved, and serious lesions of these organs may thus be prevented. Such general means as are applicable in the treatment of all fevers may be employed.

If the temperature of the patient rises above 104°, certainly if it rises above 105° F., it is important that some measures be resorted to for its reduction. The temperature should never be allowed to remain at 105° F. longer than twenty-four hours. The means which are to be employed to accomplish this reduction are the antipyretic measures already referred to. There is a strong prejudice against the application of cold to the surface of the body in scarlet fever. I am by no means certain that cold baths are always safe, or that in all cases the application of cold to the surface is judicious treatment. It is said that the kidneys will be most readily relieved of the scarlet-fever poison when cold is used for the purpose of reducing the temperature. It is claimed that when the temperature of a patient is kept below 103° F., scarlatinal nephritis rarely occurs. This statement is not sustained by facts; it has been found that kidney complications are as extensive in the cases where cold is employed as in those cases where the temperature ranges higher and cold to the surface is not employed. We should be governed by the same rules in the application of cold to the surface in scarlet fever as govern us in the treatment of typhoid fever.

With regard to the use of other antipyretics I need add nothing to what has already been said in connection with the treatment of other fevers. Unless the temperature in a case of scarlet fever ranges above 105° F., do not apply cold to the surface or give antipyretics. With such a temperature there probably will be delirium, but it must be regarded as one of the phenomena of the disease, requiring no special treatment. If the temperature rises above 105° F., perhaps reaching 106° or 107° F., and the patient manifests the nervous phenomena which have been referred to, such as restlessness, tossing, blueness of the surface, tendency to coma, etc., the temperature is to be reduced either by the application of cold to the surface or by the administration of antipyretics.

In all cases the patient is to be sponged frequently with tepid water, and if there is intense burning of the surface, a saline is to be added to the water. Sponging in this manner will give the patient very great comfort.

Some advise that the surface be anointed with oil for the relief of the burning. My own experience has led me to rely upon simple tepid saline water. I have found that it gives patients greater relief, is more easily applied, and is every way more agreeable than any of the substances which have been used for this purpose. I have not found that the application of oil to the surface has any effect in controlling the temperature, nor does it seem to have any effect on the process of desquamation. As soon as desquamation commences the process should be assisted by frequent washings with soap and water.

For the throat complications, which will give more or less trouble in all severe cases, especially when there is much enlargement of the glands at the angle of the jaw, causing difficulty in swallowing, leeches were formerly employed, but their use has now been almost entirely abandoned. Of all the remedies which I have employed for the relief of throat complications, cold carbonic acid water proved best. Whether it does more than afford relief, I am not able to say, but I am certain that cold carbonic acid water or pieces of ice held in the mouth, and brought as much as possible in contact with the swollen mucous membrane of the throat, if used early, afford most marked relief.

In the advanced stages of the disease, where there is great infiltration of the glands and tissues of the neck, cold applications do not afford the same relief as when they are used in the early stage; then cloths wrung out in tepid water and applied to the surface seem to be of service. During this stage, hot applications are generally much more agreeable to the patient, and the hot cloths may be covered with oil-silk. These applications will not hasten the suppurative process, unless suppuration is already established. While using hot applications externally, warm water gargles and steam inhalations may be used internally. Of these methods of treating throat affections, the one which seems to be the most rational plan of treatment should be chosen. In scarlet fever I favor the use of hot water rather than cold applications. The superficial and deep ulcers which are sometimes seen in the throat of scarlet fever patients can best be treated by spraying them with carbolic acid, muriated tincture of iron, chlorate of potash, tannic acid, or any of that class of remedies. Whatever remedy may be chosen, it can be much more successfully applied by means of spray than by a camel's-hair brush or a probang. Such local remedies thus applied afford great relief. The pain from these ulcerations is sometimes very severe, and cocaine, ether, or other anodyne applications in a form of spray may be used with satisfactory results.

In a certain class of cases, where there is marked disturbance of the nervous system accompanied by great depression of the vital forces and feeble heart action, stimulants will be demanded early. It is not necessary to wait until a certain stage of the eruption or of the disease is reached before commencing their administration. It may be necessary to resort to their use within twelve hours, or even within a less time, from the commencement of the attack. In some cases the beneficial effect that may be produced by the free and early administration of stimulants will be the physician's sole reliance.

The approach of kidney implication in scarlet fever will be indicated by the development of those premonitory symptoms which precede the anasarca; and whenever such symptoms are developed, dry or wet cups, according to the condition of the patient, should be applied over the region of the kidneys, upon either side of the spine; three or four cups are to be applied on each side, and their application followed with hot fomentations over the kidneys. At the same time the temperature of the sick-room is to be raised to 73° or 74° F., the body of the patient covered with flannel, hot-air or warm baths are to be administered, and the administration of diuretics is to be commenced early. Of these, digitalis will act most favorably. If the anasarca does not disappear under the influence of the digitalis and the other means employed, calomel may be combined with the digitalis and its use continued for a few days. Pilocarpin is recommended by some; my experience with it has not been satisfactory. The action of diuretics is increased by having a mercurial combined with them. In certain cases, when the patient is going from bad to worse, when the anasarca is increasing, the tendency to coma is becoming more and more marked, indicating an unfavorable termination to the case, cups have been applied, and hot baths and diuretics employed with no satisfactory result—if then small doses of calomel are combined with the diuretics, and its use continued for two or three days, the entire phase of the case may be changed. When toxic symptoms are marked, some advise carbolic acid, the sulpho-carbolates, the hypophosphites, inhalation of ozone, etc. In conjunction with the measures recommended, the patient may drink as freely as possible of water. If convulsions occur, or threatening symptoms indicating the approach of convulsions are developed, opium, either hypodermically or by the mouth, may be given. Under such circumstances, the effect of opium is often most satisfactory. It not only arrests the convulsive tendencies, but produces the most profuse diaphoresis, and aids in restoring the renal functions.

MEASLES.

Measles, or *rubeola*, is a disease from which few persons escape. It is essentially a disease of childhood, but it may occur at any age; it is, however, less liable to occur in young infants than in children after the period of dentition. A second attack is of rare occurrence. It is characterized by an eruption of red spots, accompanied by a catarrh of the mucous membrane of the air-passages, and a more or less severe fever. It is contagious. It may prevail as an epidemic, but occurs more frequently as a sporadic disease.

Morbid Anatomy.—Its anatomical lesions, with the exception of the eruption, are similar to those of small-pox and scarlatina. There are similar changes in the blood, and the same tendency to congestion of the internal organs. The spleen and liver are moderately enlarged. The mucous membranes of the nose, pharynx, larynx, and larger bronchi, and the conjunctivæ, are more or less intensely congested and present all the lesions of acute catarrh. In the majority of instances this catarrh is most severe just before and during the early period of the eruption; generally, it begins to disappear when the eruption has reached its height, and within two or three days entirely disappears. Where death has resulted from measles, in the majority of autopsies, evidence of capillary bronchitis is found, and not infrequently of catarrhal pneumonia also.

Strictly these are not anatomical lesions of measles, but complications; they are, however, such frequent attendants of this disease, that they are almost a part of its history.

The eruption is papular; the papules first show themselves upon the face, especially upon the chin; gradually they extend to all parts of the body, and lastly appear upon the back of the hands. When the eruption is well developed the spots are slightly elevated, and have a diameter varying from two-fifths to one-twentieth of an inch; in form they are crescent-shaped, their margins are sharply defined, usually their color is bright-red, sometimes shading off into blue. In most cases the spots are distinct and separated from each other by pale tracts of skin; they may become confluent, and thus give to the surface a uniform redness. When this occurs the surface presents an appearance similar to that seen in scarlatina. The earlier papule in each spot usually occupies the place of a hair-follicle; hence some regard inflammation of a sebaceous follicle of the skin as the first event. The spots disappear on pressure, but immediately return when the pressure is removed. Sometimes each spot contains several papules. The diversity in form and appearance of measle-spots in different cases depends upon variations in size, elevation, and grouping of the papules. When the spots assume a dark-red color, and do not disappear on pressure, capillary hemorrhages have taken place into the papules, and the eruption is called hemorrhagic. When the eruption is very abundant, little vesicles sometimes appear upon the papules, especially upon the trunk when there has been profuse perspiration, called by some *vesicular* or *miliary* measles. As soon as the spots have reached their maximum of development, their color begins to fade; the fading is progressive, the centres of the spots retain their redness longest; the elevations subside with loss of color. In a varying time, from one to five days, the spots entirely disappear, leaving a yellowish or brownish stain. This staining is due to pigmentation of the skin, and is sometimes visible for two weeks.

Exfoliation of the epidermis or desquamation takes place only upon the sides of the measle-spots; it is never so extensive as in scarlet fever. The skin does not desquamate in layers, but in fine brown scales, *i. e.*, is furfuraceous, not squamous, hence it is called the *bran-like desquamation*. It may commence before the redness of the eruption disappears, but it does not usually occur until the eruption has entirely faded. In most cases the period of desquamation is short, rarely lasting a week.

Etiology.—It is essentially a contagious disease. So far as has yet been determined, it is only propagated by contagion. There are places, extensive districts, and countries thickly inhabited, where this disease has never prevailed. The poison of measles is located either in the mucous secretion, or in the exhalations from the body of the infected, and the air becomes so contaminated about the sick that when persons who have not had the disease are brought within its influence, measles will be developed. It has been proved that the blood, the mucous secretions, especially the nasal, and even the tears, have the power of conveying the disease by inoculation.[1] There is little question but that the disease can be conveyed in clothing, or, in other words, that it is a portable disease. One not protected when exposed to measles is much more certain to contract the disease than is an unprotected person to contract small-pox or scarlet fever. It is possible for the infection to be conveyed from one place to another in clothing and in fluids. Its microbe has not yet been definitely determined.

The average period of incubation is eight days. During this period the poison remains latent, giving its possessor no knowledge of its presence. In most cases a slight exposure is sufficient to induce the disease; in some cases it is contracted only after prolonged exposure. Susceptibility to this contagion is almost universal. All classes are equally subject to the infection. Second attacks are exceedingly rare. The exact time in the course of the disease when measles is most infectious is not definitely determined. Statistics furnish almost absolute proof that it may infect throughout its entire course.

Symptoms.—Measles, like the other exanthematous fevers, if uncomplicated, runs a definite course.

Premonitory or *precursory Stage.*—At the end of the stage of incubation or latent period of the disease, which is without fever, and free from local symptoms, or from eight to ten days after exposure, the patient begins to suffer from coryza, is languid, chilly and exceedingly irritable. Sometimes a subnormal temperature precedes the first symptoms. Occasionally, in young children, convulsions occur. The coryza and other catarrhal symptoms, at first, may or may not be accompanied by fever, or the sudden initial fever may be very intense. Very soon, in either case, occurs a marked febrile movement. The eyes will be injected and watery, there will be a burning sensation and an aversion to light, and the eyelids will be red and tumefied. There is a constant, irritating, watery discharge from the nose, with frequent sneezing and pain over the frontal sinuses. Sore throat is complained of, and the voice is a little husky. Bronchial catarrh is indicated by uneasiness and constriction over the chest, with a frequent dry, hoarse cough, hurried respiration, etc. The suffused, red appearance of the eyes is peculiar, and distinguishes measles from scarlet fever and other forms of eruptive fever.

After the early symptoms have continued perhaps for twenty-four hours an initial fever will be developed which, with the catarrhal symptoms, continues from forty-eight hours to four days; then the eruption makes its appearance.

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[1] An organized ferment, *bacterium* or *tarnea* (which develops to a certain point in a proper medium and then suddenly ceases its career), has been found in blood and breath and in glycerine on which children with measles have breathed. They have been found in the true skin, lymph-spaces and sweat-glands; in shape they are rod, spindle and canoe shaped, also spherical and ovoid. They are also found in the lungs.

Eruptive Stage.—The eruption is first seen upon the face (about the chin, forehead, mouth, and side of the nose), then upon the neck, then upon the chest and over the body, afterwards upon the legs and arms, and lastly upon the back of the hand. The eruption on the face feels like small shot early in the disease. The eruption *may* appear first on a part of the skin that has been the seat of injury. Usually it is about four days from the time of the appearance of the eruption upon the face before it has passed over the entire body, and it begins to fade from one part about thirty-six hours after its appearance upon that part; first, it begins to fade from the face, then the neck and chest, and finally from the back of the hands. If closely examined, the eruption will be found composed of little, fine, red, crescentic dots, which, after a little time, will be seen crowded together in patches of irregular shape. Between these patches the skin usually has its natural appearance. The odor is peculiar during this period. The eruption of measles presents more of a papillary appearance upon the face than upon any other part of the body. With the appearance of the eruption there is more or less swelling of the surface, with itching and burning, and the color of the eruption will vary from a bright rose-red to a dark mahogany hue. The difference in color depends upon the condition of the individual and the peculiarity of the type of the disease, rather than upon any change in the skin itself. The respirations are hurried, and convulsions may, in children, prove fatal. Epistaxis is common, and the lymphatic glands are enlarged. As the eruption disappears it loses its bright red color and becomes a yellowish red, until finally nothing but a staining of the surface is left; then desquamation commences. Increase in fever and rise in pulse and nocturnal delirium often follow the first outburst of the eruption.

Desquamative Stage.—The desquamation which follows the eruption is not like the desquamation of scarlet fever—scaly or "peely,"—but it occurs in very fine dust-like flakes, which may pass unobserved. The eruption reaches its height by the third day from the time of its appearance, and generally has disappeared by the end of the sixth day. As a rule, during the development of the eruption the catarrhal symptoms and fever are increased in intensity; the patient will sneeze and cough, and frequently with such severity and with such a coarse, grating tone, that it has received the name of "*iron cough.*" It is not the cough of croup (though a true croupy cough is sometimes present); there is no stridulous breathing accompanying it, but it is the result of an ordinary catarrhal laryngitis, which causes the patient to cough perhaps for two or three days without expectoration, or any attempt at expectoration. During this period the pulse will range from 100 to 120 beats per minute, and in young children it may reach 160. In the majority of cases the temperature does not rise above 103° F., but it may rise as high as 106° or 107° F. As soon as, or *even before* the eruption begins to decline the temperature often falls two or three degrees. As the decline in the eruption goes on, the temperature gradually falls, until by the time the eruption has entirely disappeared the patient will be fully convalescent.

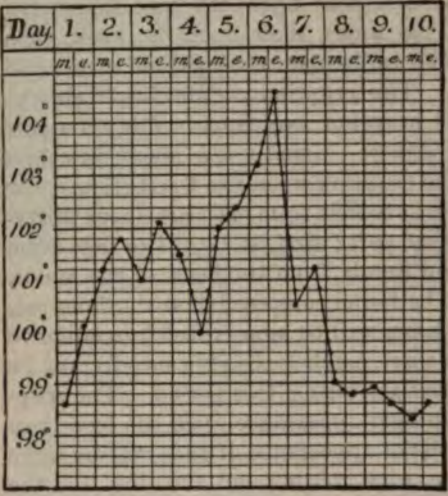

Fig. 169.
Temperature Record in a Case of Measles.

Measles, like scarlet fever, is liable to irregularities in its development. When it is prevailing in a locality, cases occur in which all the catarrhal symptoms of the disease are present without an eruption; again, there is an eruption closely resembling that of measles, with no catarrhal symptoms; from the appearance of the eruption one will not be able to say whether the patient has or has not measles; if the subject has been exposed to the contagion of the disease, the case will probably be regarded as one of measles, and yet if there are no catarrhal symptoms, but simply an eruption, such a diagnosis would be made with a question.

There is a form of roseola which very closely resembles measles in every aspect of the disease, except the catarrhal symptoms. There is an irregular form of measles which prevails epidemically, which is characterized by a tendency to ulceration of mucous surfaces. This form shows its peculiar tendency by the development of ulcers at the angle of the mouth, within the nose, around the vulva, anus, etc. Sometimes these ulcers spread and so interfere with deglutition and respiration as to endanger life. The ulcerations are accompanied by great prostration of the vital powers and a tendency to gangrene of the above-named parts and also of the lungs. This irregular variety only occurs in those who are poorly nourished, live in badly ventilated houses, and are surrounded by unfavorable hygienic influences.

Again, there is a form of measles in which, at the very onset of the disease, there is a very high range of temperature. There will be no more severe catarrhal symptoms than in the ordinary forms—no more bronchitis; but there will be a higher range of temperature, perhaps ranging as high as 106° or 107° F. Associated with this pyrexia there will be restlessness, a dry tongue, and, very soon after the appearance of the dry tongue, a change in the color of the eruption, which will assume a dusky purplish hue. The eruption may present this peculiar appearance at the very commencement of its development. This type of measles is called *"black measles."* The color of the eruption simply shows that there have been extensive blood-changes. In most cases, these changes have taken place prior to the development of the eruption. By some it has been claimed that there is at work a peculiar epidemic or endemic influence that gives rise to the peculiar type of the disease; but as I have been brought in contact with it, it has seemed to me that it differed from the ordinary type only in the intensity of the fever. It is the high range of temperature which stamps it as a peculiar type of the disease ; but as soon as the eruption has made its appearance, although at first it may be of a bright red color, within a day or two it assumes the peculiar dusky black appearance which has given rise to its name.

There is another irregular form of measles in which the eruption is largely made up of petechial spots scattered over the surface of the body, due to a hemorrhagic diathesis. It is really a hemorrhagic form of measles, and is a very unfavorable type of the disease. At first the eruption presents the same appearance as the ordinary eruption of measles; but, after the fever has continued a few days, it assumes a dark color, the patient becomes restless, the tongue dry, there may be vomiting and diarrhœa, and, if death occur, at the post-mortem examination lesions very closely resembling those of typhoid fever, such as changes in the spleen and elevation of Peyer's patches, will be found. These cases are also known by the term "black measles." Hemorrhages also occur from nose, mouth, urethra, intestinal and other mucous tracts.

There are thus two forms of black measles—one in which the eruption consists of petechial spots scattered over the surface, and dependent upon a hemorrhagic tendency; in the other form the eruption assumes a dark appearance on account of changes which have occurred in the blood, the result of a very high temperature at an early period of the attack. There is always more or less danger connected with any of the more severe forms of irregular development. Although measles is usually not a disease of much severity, yet, however mild the type may be, it is liable to complication, and the most frequent complications are to be found in the respiratory organs.

Complications.—Of these the most important is capillary bronchitis. Rarely is there a case of measles without more or less bronchial catarrh ; but the bronchial catarrh which ordinarily attends it is not of much consequence. When, however, the bronchitis becomes capillary, the patient is in great danger. Upon auscultation, if instead of loud, sonorous râles, which indicate that the catarrh is confined to the larger bronchial tubes, there are fine crackling sounds, accompanied by an entire loss of, or an extremely feeble, vesicular murmur, the catarrhal inflammation has extended into the finer bronchial tubes, and there is always danger of lobular pneumonia (*q. v.*). A lobular pneumonia which complicates measles is always attended with danger, and when depression of temperature follows decline of the eruption, all the pulmonary signs may grow very intense. With serious lung complications, the eruption may recede. As a rule, it attacks both lower lobes at the same time, especially their dorsal aspect, while in the upper lobes only a few tubes are involved. This complication may occur at any time during the course of measles, but it is more liable to occur just after the eruptive stage. Its development is attended by a rise in temperature, in proportion to the extent of lung involved. The urine is always scanty and may be suppressed.

Secondary meningitis not infrequently occurs as a complication of measles. When it does occur, it is developed during the period in which the eruption is disappearing. It is more likely to occur in this disease than in scarlet fever.

A sequela of measles is a mild form of ophthalmia. This ophthalmia may considerably inconvenience the patient, and lead to permanent injury of the eyes. It is especially important to remember that it appears during the convalescing period, that it is a conjunctivitis, and usually entirely disappears if the eyes are frequently bathed with warm water and properly protected from the light.

Otorrhœa, or inflammation of the external ear, is another sequela of measles. It most commonly appears in those patients who have what is called a strumous diathesis, have phthisical parents, are themselves badly nourished, or who have suffered from a severe form of measles. This otorrhœa is sometimes very obstinate, and if it yields to treatment does so very tardily; it may be followed by permanent deafness.

In adults acute miliary tuberculosis not infrequently occurs as a sequela of measles. The mucous membrane of the intestinal canal may also become the seat of important complications in measles. A mild form of gastric catarrh is of quite frequent occurrence, but is rarely serious in character. Severe intestinal catarrhs, giving rise to troublesome diarrhœa and dysentery, are sometimes very serious complications, especially in very young and feeble children. Occasionally malignant epidemics of measles prevail, during which the fatal results are chiefly due to intestinal catarrhs.

Diphtheria does not so frequently complicate measles as it does scarlet fever. It generally makes its appearance when the eruption is at its height, and when severe its occurrence is marked by a rapid rise in temperature. The symptoms of the diphtheria are the same as when it occurs as a primary disease. It must always be regarded as a serious complication. Not infrequently measles leaves the patient in a state of general ill-health. Especially is this the case in scrofulous and rachitic children.

Differential Diagnosis.—Ordinarily, when the eruption is well defined, the diagnosis of measles is not difficult. In some cases, however, the eruption presents an appearance which closely resembles that of scarlet fever and roseola. In nearly every case of measles the catarrhal symptoms accompany the precursory stage, and increase in severity during the period of eruption. The presence or absence of these catarrhal symptoms will enable one in the majority of cases to make a differential diagnosis.

In children, the eruption of *typhus fever* very frequently closely resembles that of measles, but it does not appear upon the face, and is not accompanied by catarrhal symptoms. In typhus fever, nervous symptoms are quite frequently present, such as delirium, prostration, and tendency to coma. Such symptoms are only met with in the hemorrhagic or typhoid variety of measles. Before the appearance of the eruption a careful examination of the mucous membrane of the pharynx will settle the question of diagnosis. In measles the mucous surface will be more or less intensely injected; in typhus fever it will not be so injected.

The differential diagnosis between measles and *small-pox* has been considered.

The eruption of measles differs from that of *roseola*. In measles it is partially confluent, in roseola it is non-confluent. In roseola the mucous membrane of the fauces is not intensely injected, and the fever does not run a characteristic course, the reverse of which occurs in measles. If the temperature is normal, if the eruption on the trunk is of a bright color, if the surface is smooth, and if catarrhal symptoms are absent, measles may be excluded.

It is hardly possible to mistake *syphilitic exanthemata* for measles, for there are certain glandular changes which attend the development of syphilitic eruptions which establish the diagnosis. In the early period of the disease, when coryza is a prominent symptom, before the appearance of the eruption, measles may be mistaken for an ordinary influenza.

Prognosis.—The prognosis in uncomplicated measles is always good. Any irregularity in its development, and dentition in children, may render the prognosis unfavorable. In the hemorrhagic, ulcerative, and in the typhoid varieties the prognosis is grave. Measles occurring in pregnancy does not prove fatal to the extent that scarlet fever does; but abortion is very common. Intra-uterine measles may be recovered from, and the child is then proof against a second attack. In severe cases, the deviations from the typical course of the disease which render the prognosis unfavorable are a temperature of 105° or 106° F. during the period of initiatory fever, a retardation or an irregularity in the appearance of the eruption at the beginning of the eruptive stage, and the occurrence of complications, especially broncho-pneumonia, croupous laryngitis and diphtheria. Profuse hemorrhages from the mucous surfaces, during any period of the fever, render the prognosis unfavorable. Recession of the rash is very unfavorable when there are any pulmonary symptoms. The hygienic surroundings of the patient greatly influence the prognosis.[1]

The prognosis also depends upon the age of the patient; the rate of mortality is much greater among adults than children, and in *very* young children than in older children. The character of the prevailing epidemic determines to a very great degree the prognosis. When measles is developed in one who is suffering from a severe chronic disease, especially some organic disease of the lungs, the prognosis is unfavorable. The patient will not probably die during the active period of the measles, but the chronic pulmonary disease may terminate fatally from the effects of the measles. For instance, a patient has evidences of consolidation about the apex of the lung, a condition which justifies a favorable prognosis; let measles be developed in such a case and capillary bronchitis, terminating in more or less extensive pneumonia, will probably occur, from which acute phthisis may be developed.

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[1] The presence of sewer-gas renders nearly every case fatal.—Quain.

In measles, death rarely occurs during the first week of the disease; it usually takes place during the second week; if serious complications occur, it may take place later in the disease. The rate of mortality is estimated at from one to four per cent.

Treatment.—The prophylactic treatment of measles consists in isolating the affected person. When measles run a regular course, the principal duty of the physician is to watch for, and guard against the occurrence of pulmonary and other complications. All that is necessary is to place the patient in a large, well-ventilated, darkened room, with the temperature of 63° or 65° F., so that the congested conjunctivæ may not be exposed to light.

The chief article of *diet* should be milk. If the patient complains of itching and burning of the surface, he may be frequently sponged with tepid water; this causes an alleviation of the itching and burning, and reduces the temperature. In an ordinary case this is all that will be required. Hot drinks or stimulants have no power to hasten the appearance of the eruption; the administration of the latter may be followed by very injurious results; convulsions and death may occur. In an ordinary case, stimulants should never be administered during the initiatory period of the fever, unless there is some special indication for their use, such as great prostration or bronchial complication; then they may sometimes be used with benefit. Covering the patient with heavy clothing does not hasten the appearance of the eruption. The greatest cleanliness should be observed; besides, there should be free ventilation, avoiding all draughts, in the sick-room. If there is thirst, cold water may be freely taken in small quantities at a time.

If the case is severe, and the temperature rises to 103 or 104 degrees F., it may be reduced by frequently sponging the surface with tepid water.[1]

Post-pharyngeal catarrh is liable to extend into the larynx and bronchial tubes and give rise to bronchitis. One of the most important duties of the physician is to watch for the occurrence of this complication; he should frequently examine the chest, and when the bronchitis is found to have reached the capillary tubes, should immediately commence treatment for its relief. I have found the inhalation of steam to afford the greatest relief, and to best control the bronchial inflammation. As soon as the larynx has become so involved as to interfere with the respiration of the patient, and there is danger of croupous laryngitis, immediately order vapor inhalations, and insist upon their continuance until the laryngeal symptoms shall have subsided. Sometimes this subsidence will take place within two or three hours, and, again, not until after two or three days. The value of vapor inhalations in the treatment of the laryngeal and bronchial compli tions of measles, I regard as very great. When catarrhal pneumonia is veloped, it is to be treated in the same manner as catarrhal pneumonia developed under any other circumstances; the patient should be sustained by the free use of stimulants.

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[1] German writers recommend the cold bath in the treatment of measles. I should hesitate to pl patient with measles in a cold bath, on account of the great tendency in this disease to pulmonary plications.

Pulmonary complications in measles are often the result of exposure to sudden changes in temperature; the severity of catarrhal symptoms will always be increased by such exposure, therefore it is of great importance in the management of every case of measles that the patient should be protected against such changes. When there is great restlessness during the fever of invasion, or during the early period of the eruptive stage, small doses of opium, in the form of Dover's powder, may be administered with marked benefit.

The management of the different varieties of measles will be indicated by the general condition of the patient. In the ulcerative, hemorrhagic, and typhoid varieties, the free administration of stimulants should be commenced early. Usually in these varieties there is great prostration, and the main indication is the support of the patient. Diarrhœa at the close of measles may take the place of lung complications, and should not be too suddenly checked.

GERMAN MEASLES.

(*Rotheln.*)

German measles, or *epidemic roseola*, has been regarded by some as a modified form of measles; by others as a modified form of scarlet fever; again, it has been thought to be a combination of the two diseases—a hybrid disease. Some maintain that it is not an independent and specific disease, but that it may embrace any blotchy exanthem.[1] I am disposed to regard it as a different type of measles from that which ordinarily prevails, and by way of distinction would call it *German measles*, or *epidemic roseola.*

Morbid Anatomy.—It is one of the mildest of the eruptive fevers. It prevails epidemically and endemically. The study of its morbid anatomy has been almost exclusively restricted to the eruption. This consists of irregular spots, or hyperæmic blotches, varying in size from a pin's head to a large pea, usually slightly elevated, so that when the hand passes over the surface of the skin it feels somewhat rough. Sometimes these spots occasion intense itching; they are quite distinctly separated by healthy skin, and disappear under pressure. As a rule, even at the acme of the development of the eruption, their color is a pale rose-red, paler than the intense red of the eruption of scarlet fever, or the peculiar bluish hue of the eruption in severe cases of measles. It appears upon all parts of the body, but is most abundant upon the face and trunk. The spots are usually discrete, are round (not crescentic), they often lie crowded closely together, but they are not confluent.

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[1] Later German writers regard it as an independent affection, a specific, acute, and contagious eruptive fever, and have given to it the name of *rubeola*.

Mild roseola is a punctate rash. The throat is red and the glands in the neck may be enlarged. The rash rarely lasts more than two days, and it is attended by itching. In some cases there is slight desquamation; it disappears and leaves no trace, except in occasional instances, when there is a transient, yellowish discoloration of the skin.

Some affirm that the rash may disappear and reappear alternately for several days, and when it has finally disappeared the disease has terminated, and there is nothing to fear from complications or sequelæ. In certain rare cases vesicles resembling miliaria may be developed upon the hyperæmic spots, especially upon the back; these are chiefly due to external conditions.

Etiology.—Doubtless this disease is contagious. Nothing is known concerning the nature of its contagion. It is essentially a disease of childhood. In those over forty years of age its development is of very rare occurrence. It is conveyed from one person to another in the same manner as measles. It has been stated that women are more susceptible to it than men.

Symptoms.—Epidemic roseola is so mild an affection, that it is questionable whether it has an invasive stage. The duration of the stage of incubation has not been determined. Generally, the symptoms which manifest themselves two or three days before the appearance of the eruption are much less marked than they are in any other eruptive fever. Perhaps in many cases they escape notice. The period of invasion is seldom more than twenty-four hours. Quite frequently the eruption is the first symptom of the disease.

In most cases there may be nothing more than a feeling of discomfort. In other cases the disease may be ushered in by vomiting, diarrhœa, and convulsions. In many cases, immediately preceding the eruption, and accompanying its appearance, there is well-marked fever, headache, loss of appetite, and sometimes noticeable prostration. When the eruption is regular in its appearance it affects first the neck and chest, then the face and scalp, and then gradually extends downward over the trunk and extremities. Usually, the development and spread of the eruption are rapid, perhaps no more than two or three days being occupied in its passage over the entire body. Its duration upon any one part of the body before it begins to disappear is not more than twelve or twenty-four hours. In the majority of cases the temperature does not rise more than 1° or 2° F. It m rise to 102° F. or 104° F. During the second day, the temperature begi to fall. Sometimes it reaches the normal within twelve hours, occasio ally not until the third day. Sometimes it reaches it by crisis, at othe by lysis. The pulse increases and diminishes in frequency according the rise and fall of temperature. The tongue is usually covered with whitish coating, and is dotted here and there with red and swollen papill The mucous membrane of the fauces is generally congested and the tons moderately swollen; there may be some soreness of the throat. The mucous membrane of the air-passages is usually in a condition of mild catarrh, consequently, at the onset of the disease, sneezing and coughing are frequently present, but they are less marked and are of shorter duration than in measles. Suffusion of the eyes with congestion of the conjunctival vessels is rarely present; there may be a slight degree of photophobia. The face and eyelids are usually slightly swollen at the time the eruption makes its appearance, but this swelling rapidly disappears.

In most cases, there is moderate swelling of the lymphatic glands of the neck, and enlargement of the glands at the nape of the neck. Moderate enlargement of the occipital glands may continue for a number of days. The disease is so mild in character that children are with difficulty kept in bed.

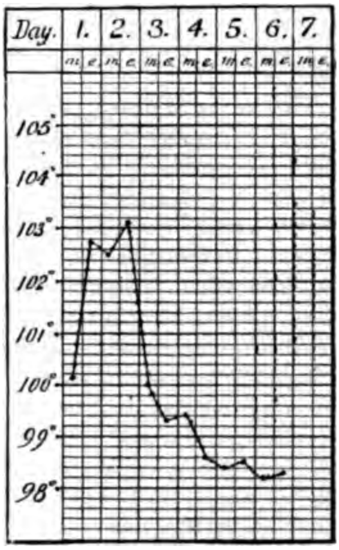

Fig. 170.
Temperature Record in a case of German Measles.

Differential Diagnosis.—One of the prominent features of this disease is the close resemblance which its eruption bears to that of *measles*. In certain cases it may be impossible by the eruption alone to make a differential diagnosis. When the eruption of measles is not typically developed, a complete history of the case must be taken into consideration. When this has been done, there is usually no great difficulty in arriving at a correct diagnosis. Perhaps that which will best aid in making a differential diagnosis between roseola and measles is the fact that an attack of one does not protect against the other, any more than does an attack of varicella protect an individual from an attack of variola. This fact certainly establishes the non-identity of the two diseases. The short period of invasion, the eruption appearing first on the chest and neck, the *very* mild nose and throat symptoms, and the *low* temperature are in contrast with the symptoms of measles.

It has been questioned whether a person may not have a second attack of epidemic roseola. The latest observations go to prove that a second attack is of as rare occurrence as a second attack of measles or scarlet fever. Again, one attack does not protect an individual against the contagion of scarlet fever; nor does an attack of scarlet fever protect one against the contagion of roseola. An individual may have an attack of epidemic roseola very soon after he has been ill with measles or scarlet fever.

Prognosis.—The prognosis is always good. Complications rarely occur; when they do, they are usually pulmonary.

Treatment.—The treatment of this affection consists in protection against exposure. Tepid sponging will relieve troublesome itching, and reduce fever. Regulate the diet, and carefully watch the catarrh of the air-passages. As a rule, convalescence is rapid.

MILIARY FEVER.

This form of fever cannot strictly be regarded as a contagious disease, but it so frequently prevails in connection with measles and scarlet fever, and has apparently so many elements of contagion, that it is included in the list of contagious fevers. Some deny its existence as a distinct fever. Writers have described it under the names of *sudamina, sudoral exanthema, miliaria alba,* etc. Several diseases which are accompanied by sweating, and which exhibit a tendency to the formation of miliary vesicles, have been called miliary fever.

Until the occurrence of the severe epidemic of the disease known as the "English Sweating Sickness," its specific type was not recognized. It has prevailed epidemically over limited areas, in Belgium, France, England, Germany, Italy, and Austria. In some of these epidemics eleven to twenty per cent. of the whole population of the invaded district has been attacked. The average duration of the epidemics has been from three to four weeks; occasionally they have lasted from three to four months.

Morbid Anatomy.—Few post-mortem examinations have been made, and those few have failed to reveal *any characteristic* lesion. The miliary vesicles which are seen upon the surface of the body, and the cutaneous eruption, are developed because the secretion of the sudoriferous glands cannot escape. The escape of the contents of these glands may be prevented by two causes: (1) the gland-ducts may become obstructed; or, (2) the secretion may be so abundant that it cannot be transmitted by the gland-duct. In either case, the secretion emerges under the epidermis around the sweat-duct, and as the scales are lifted up, a small clear vesicle is formed. The liquid contained in the vesicle at first is transparent, has an acid reaction, and is said to contain free nuclei and cells which have three or more nuclei; these nuclei remain visible after the cell membrane has been destroyed by the addition of acetic acid. The contents of the vesicle becomes milky and yellowish from pus (m. alba). It has been claimed that the virus of the disease is contained in these polynucleated cells. After death, the skin becomes œdematous, and very soon the odor of decomposition is perceptible.

Etiology.—It has been supposed that miliary fever was indirectly induced by scarlatina, the puerperal condition, variola, vaccinia, typhus fever and like diseases, and that it was not a distinct disease arising from some constitutional cause. The prevalence of this fever in connection with these diseases gave rise to this supposition. Epidemics of this disease have generally prevailed during the *spring* and *summer* months; from this fact one would be led to think that there is some atmospheric condition peculiar to these months. Again, the disease has most frequently appeared in *warm, moist weather,* and from this fact it has been supposed that some peculiar condition of the soil is necessary to its development. Certain epidemics have shown a close connection with contaminations of the soil, such as arise from neglect of drainage, collections of refuse, etc. Doubtless, such conditions of the soil may increase its severity, and cause it to prevail more extensively, but facts do not prove that, directly or indirectly, they cause its development.

The disease usually attacks healthy adults, and occurs more frequently among females than males. It attacks all classes, and its spread does not seem to be affected by crowding.[1]

Symptoms.—The average duration of the disease is from five to eight days. It has three stages: (1) *the stage of invasion;* (2) *the stage of sweating;* and, (3) *the stage of eruption and desquamation.*

The Stage of Invasion.—The average duration of this stage is from forty-eight to seventy-two hours. It is characterized by an excessive irritation of the skin, thirst, general lassitude and headache. There is also more or less febrile movement. Some writers mention a feeling of suffocation, which is usually preceded by a sense of oppression at the epigastrium. These are the characteristic symptoms of the stage of invasion.

The Stage of Sweating.—This stage is usually ushered in by rigors; rarely, by a well-marked chill. The characteristic symptom of this stage is profuse and persistent sweating. The sweating is accompanied by a prickling sensation of the skin, distress, and a sense of compression at the epigastrium, and by more or less violent palpitation of the heart, with precordial pain. Usually the sweat appears on all parts of the body at the same time. Sometimes it appears first upon the head and breast, then gradually descends, and soon becomes so abundant that every article of clothing, bed-clothes and bedding, becomes saturated. The pulse sometimes reaches 140 beats per minute, the temperature rises to 103° F., 104° F., or even 105° F., and the skin, notwithstanding the profuse perspiration, feels extremely hot. During this stage the headache and the sense of suffocation increase; the epigastric and precordial pain and the palpitation increase in severity, and sometimes become alarming, although the most careful physical examination fails to discover any lesion in the heart or lungs to account for them. The respiration becomes rapid, often irregular and intermittent.

Irregular exacerbations, or even intermissions, in these symptoms may occur, but, as a rule, they continue without abatement until the vesicle appears, on the third or fourth day of the disease.

The Stage of Eruption.—This stage is characterized by the appearance of a rash. It is first seen upon the neck and breast, then upon the back and extremities, sometimes upon the mucous membrane of the mouth, nose, and conjunctiva, sometimes upon the abdomen and scalp. This eruption consists of irregularly shaped spots, 1-8 to 1-16 in. in diameter. In some cases they stud the skin so thickly that it appears like an uniform sheet of vivid redness. After the lapse of a few hours, vesicles can be seen in the centre of these spots; perhaps at first they are so small as to necessitate the aid of a lens to discover them. These vesicles rapidly increase in size, and may reach the size of a millet-seed or a small pea. The contents of these vesicles have already been described. Occasionally, as the eruption appears, all the constitutional symptoms are increased in severity, but usually they are modified and disappear either suddenly or gradually after its development. In the milder cases the vesicles only, without the efflorescence, are seen. Vomiting is rarely present, although nausea is a common symptom, as is also constipation.

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[1] It can hardly be regarded as a contagious disease, in the sense that it can be communicated directly from the sick to the well. It does not seem to be well established that the disease can be developed by inoculation with the contents of the vesicle, notwithstanding it has been supposed that certain cells in the fluid hold the contagion of the disease. The infrequency of the simultaneous occurrence of miliary fever with epidemics of measles or scarlet fever, is unfavorable to the theory that there is a specific relationship between the poisons of these diseases. The view that there is an intimate relationship between cholera and miliary fever has been accepted by some writers, and the accession of the latter during the course of the former has been supposed to exert a favorable influence over the course of the disease; the opposite, however, does not appear to hold good, but on the contrary, favors a fatal termination. Much remains to be learned in regard to the relationship existing between miliary fever and the other diseases which we have mentioned.

The urine is usually scanty and high colored; in some cases there is suppression of urine. Occasionally, during the stage of eruption, profuse secretion of urine takes place. This has been regarded as a favorable symptom. The vesicles, clear at first, soon become opaque and yellowish, remain for two or three days, then burst and begin to fall off in scales. Desquamation is usually completed within forty-eight hours, but convalescence is often quite protracted on account of the debility and emaciation.

Such is a brief description of miliary fever, when it runs a regular course, but there are certain variations in the development of the symptoms which should be noticed. In the severest form of the disease, the temperature may rise to 107° or 108° F., and there may be delirium and a sense of suffocation. Again, even in fatal cases, the eruption, sweating and convulsions may be absent. Occasionally sudden and fatal collapse follows the sweating stage. The typhoid condition may be developed in the sweating stage, and may be attended by black sordes upon the teeth and tongue, epistaxis and uterine hemorrhage, and may terminate fatally, without any considerable anatomical changes recognizable after death.

Complications are not of frequent occurrence. Occasionally there is bronchitis, pneumonia, and diarrhœa.

Relapses are of common occurrence, but recovery generally takes place after a short relapse.

Differential Diagnosis.—Miliary fever may be confounded with *measles*, with *malarial fever*, and with *typhoid fever*.

The profuse sweating, the prickling of the skin, the intense oppression at the epigastrium, the sense of suffocation, with precordial pain, and the peculiarity of the eruption, are sufficient to distinguish it from measles, from intermittent fever (although a decidedly intermittent type of the disease sometimes prevails), and from typhoid fever. When the disease prevails epidemically, the diagnosis cannot be difficult.

Prognosis.—When the disease runs a regular course, with only a moderate degree of severity, the prognosis *is good;* whereas great severity of the febrile symptoms, exceptionally profuse sweating, and increasing sense of constriction of the chest, with suffocation, render the prognosis unfavorable. The accession of profuse hemorrhages, coma, convulsions, active delirium, or symptoms of collapse, render the prognosis unfavorable. The severity of the symptoms is usually mitigated when the eruption makes its appearance, and death rarely occurs after that stage is reached. If a fatal termination is reached, it usually takes place during an exacerbation, prior to the appearance of the eruption. In some epidemics, the mortality has been very great; in other epidemics the disease has been mild in character; eight or nine per cent. is the average death-rate. The character of the epidemic affects the prognosis.

Treatment.—At one time diaphoretics were employed in the treatment of this disease, on the supposition that the sweating and eruption were critical manifestations, and must be aided by all possible means. The sense of suffocation, with that of constriction of the chest, was thought to indicate blood-letting; but it was soon decided that loss of blood aggravated rather than improved the patient's condition. Antispasmodics, nervines, quinine, emetics and counter-irritants at different times have formed the basis of various plans of treatment. Of late, subcutaneous injections of morphine have been used with advantage. Sinapisms and blisters have been employed for the relief of the sense of constriction in the chest, and for the epigastric and precordial distress, with benefit to the patient. It is now acknowledged that the administration of purgatives in large doses should be carefully avoided, as well as blood-letting, general or local.

At present the expectant plan of treatment is regarded with most favor. It chiefly consists in the use of cooling drinks, aromatic teas, acidulated water, sponging with warm water, or the employment of warm baths. It has been thought that the addition of alum or vinegar to the water used for sponging or bathing is beneficial. In the treatment of this affection, quinine seems to be regarded with almost universal favor. If restlessness is persistent, opium, ether, valerian, and antispasmodics may be employed in moderate doses, carefully watching the effect produced. The patient should be surrounded by proper hygienic influences, the diet should be moderately nutritive, and in those cases in which convalescence is tedious a steady and continued tonic treatment is indicated. In the severest form of the disease stimulants may be employed with benefit.

INFLUENZA.

(*Epidemic Catarrh.*)

Influenza is a specific continued fever, generally widely epidemic, and attended by catarrh of the respiratory and digestive tracts. It has received a great variety of names. In 1830 and '31 a severe influenza epidemic swept over the whole civilized world.

Morbid Anatomy.—There are no special pathological lesions of influenza. There is generally more or less extensive inflammation of the respiratory organs; the lungs are usually inflated so that when the chest is opened they protrude from the cavity instead of collapsing. Sometimes they are very dry, at others œdematous. Spots of lobular consolidation appear as depressions between the inflated portions. The mucous membrane of the trachea and larger bronchi is red and covered with frothy or viscid mucopus. The injection is usually most marked in the smaller tubes. The bronchial glands are enlarged and softened. Pale, firm clots are found in the right heart. The gastric and intestinal mucous membrane is congested; the stomach is usually more congested than the intestines. Hence the name *gastric influenza*.

Etiology.—All conditions and ages suffer alike; but children are sometimes remarkably exempt. The disease travels very rapidly; it has passed over the whole of Europe in six weeks. It passes quickly from one country to another, visiting whole continents in a short time. It rarely continues in one locality more than two months. There is no doubt that influenza is due to some powerful special morbific agent, which is given off by the mouth of the infected, and which acts specifically upon the respiratory mucous membrane and also upon the nervous system.[1]

Symptoms.—Influenza comes on suddenly. A feeling of chilliness, sometimes distinct rigors, flashes of heat, and a feeling of lassitude are followed by symptoms of a severe naso-pharyngeal catarrh, with frontal headache, pains in the limbs and back, soreness of the throat, hoarseness, and a frequent racking cough, difficult breathing and constriction across the chest. The sputa are first mucous and then scanty, later copious and muco-purulent. The respirations are accelerated; there is great prostration, lassitude, apathy, muscular weakness, and precordial oppression. The fever assumes a remittent type, attended by profuse perspiration. Sudamina appear on the surface and herpes on the lips. The pulse is rarely over 90; yet a temperature of 104° has been observed, and the pulse has been feeble, irregular, and 120 per minute. The tongue is moist and covered with a white fur; it may be dry and brown. There is anorexia. Nausea and vomiting may be early symptoms and continue throughout the whole course of the disease. The bowels at first are constipated, later there is diarrhœa. There may be hepatic tenderness and slight jaundice. As the disease advances the face gets congested and livid, the frontal headache becomes more severe, the pulse increases in frequency and becomes feeble, the tongue becomes brown and dry. There are muscular tremors and subsultus, the patient becomes dull and listless, and delirium is often present.

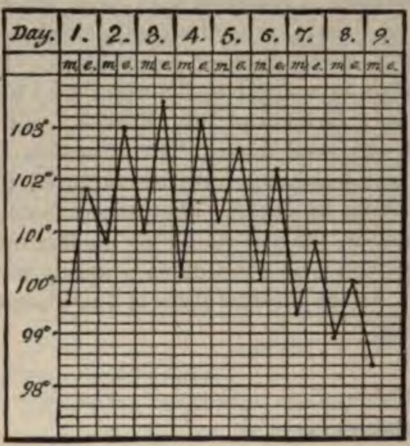

Fig. 171.
Temperature Record in a case of Influenza.

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[1] Brimer (in Virchow's Handbuch) thinks it is a miasmatic disease, caused by a living miasm capable of being transmitted by the air and having an independent existence. It is probably contagious.

On auscultation sibilant and sonorous râles are heard over some portions of the chest, while at others the inspiratory sounds are dry and harsh. The vesicular murmur is always indistinct. Measle-like spots are often seen on the palatal mucous membrane. In mild cases the disease is at its height on the third day and then gradually declines. In the severe cases where the pulmonary symptoms are prominent, convalescence does not commence until the tenth or twelfth day; convalescence is protracted and relapses are frequent. The *urine* is less in quantity than normal; sometimes there is complete suppression. It is high-colored and deposits a sediment on standing. The different "varieties" of influenza as described by writers are due to the different complications.

Differential Diagnosis.—The large number of persons attacked, the nervous debility which accompanies it, and its short uniform course are generally sufficient for its diagnosis.

The **Prognosis** is good except in the very old, very young, and in those already subjects of pulmonary or renal disease. Whenever there is a high mortality-rate, the fatality is due to complications which have been engrafted on the influenza. Its *complications* are chiefly of the respiratory tract, the more frequent of which are laryngitis, bronchitis, pulmonary congestion and œdema, pneumonia,[1] which is usually lobular, and pleurisy; these complications have given to the disease the name of epidemic catarrhal fever. Pharyngitis, parotitis, salivation, hyperæmia of the liver, and subacute gastritis are rare complications of the digestive apparatus. Herpes labialis occurs often. The duration of influenza varies from four to twelve days; and an epidemic rarely continues more than from four to six weeks in one locality.

Treatment.—When influenza is prevailing all exposure to cold must be carefully avoided, and in its treatment the general hygienic measures of the acute infectious fevers are to be employed; medicinal treatment is not very efficacious. Quinine sometimes aborts it, if given in large doses at its very onset. In the early stages liquor ammonii acetatis and pulvis ipecacuanhæ (one grain of the latter in one-half ounce of the former) every two or three hours is all that is required. The bowels should be kept freely open with salines; milk combined with alkaline waters is the only food which should be allowed for the first forty-eight hours. If patients are restless, Dover's powders may be given in small doses. Steam inhalations will relieve the laryngeal and bronchial symptoms, and may be constantly employed during the acute stage. The prostration which occurs in the old, young, or feeble must be combated early with stimulants.

All revulsives, blood-letting and depressing remedies are contraindicated. Colchicum, carbonate of potash, and opiates are of service in those cases of influenza where pain and rheumatic symptoms are predominant. When convalescence commences the patient should begin to take quinine and iron in small doses with a nourishing diet; a change of air is often of great service, especially if there have been pulmonary complications. It must be remembered that influenza is often the exciting cause of a phthisical development in subjects who are so predisposed.

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[1] In one hundred and eighty-three patients at Hôtel Dieu over twenty per cent. had lobar pneumonia. —Copland.

PERTUSSIS.

(*Whooping-Cough.*)

Whooping-cough is an acute contagious disease, attended by a peculiar spasmodic cough. It should be classed among the diseases of children, although it may occur at any age.

Morbid Anatomy.—The principal, if not the only, morbid changes in this affection are those of catarrhal bronchitis. Those who regard the disease as of nervous origin claim that there are evidences of inflammation of the vagus nerve, or congestion of the medulla oblongata. I am disposed to regard it as a specific catarrh of the respiratory mucous membrane, which differs from other forms of catarrh in being contagious and attended by peculiar laryngeal and bronchial spasms. Its complications are cerebral œdema and congestion, lobular collapse, lobular emphysema, bronchial dilatation, or capillary bronchitis and catarrhal pneumonia.

The specific catarrh is located chiefly in the bronchi, although some regard it as at first limited to the pharyngeal mucous crypts, and still others regard it as confined to the larynx. The bronchial and mediastinal glands may undergo softening, the pleuræ and pericardium may be ecchymotic. The mucous membrane of the stomach is congested and sometimes studded with petechial extravasations. Follicular enteritis is not uncommon. The liver and the spleen are often enlarged and fatty. Letzreich claims to have found a fungoid vegetation in the epithelium of the air tubes. Buhl, Oertel, and Hüter also found them.

Etiology.—Whooping-cough depends upon a specific poison which is given off in the breath of the affected, and conveyed through the air to the healthy. A second attack is rare. The period of incubation varies from five days to two weeks; micrococci are sometimes found in the sputum. Teething and measles predispose to the reception of the infection. It may be carried in clothes. It may prevail epidemically, attacking nearly all the children of a neighborhood or township.

Symptoms.—There are three recognized stages in whooping-cough, a *catarrhal, a spasmodic,* and a *stage of decline.*

The *catarrhal* stage is marked by the ordinary symptoms of a severe nasopharyngeal and bronchial catarrh. It rarely commences with a chill, but fever, restlessness, and languor are marked. The fever in the early stage is intermittent. It commences with coryza, and a severe dry paroxysmal cough, which is soon attended by an abundant, tenacious, viscid, transparent mucus. The respirations are shallow and the pulse is rapid. The duration of this stage is from two days to three weeks; nine to ten days is the average.

The *spasmodic* stage is attended by a characteristic spasmodic cough. This cough is very severe and distressing; the face grows red, and then begins a long, clear, piping sound, followed by a series of rapid, convulsive and forcible expiratory puffs, which are succeeded by a prolonged, shrill inspiratory sound or whoop. If the fit lasts any length of time, the cough becomes inaudible, and a considerable quantity of clear, viscid mucus is expectorated or vomited with the contents of the stomach. During the paroxysm the patient grows red or purplish in the face, the eyes protrude, the tongue assumes a dark appearance, and he seems on the verge of suffocation. Bleeding from the mouth, nose, ears, and lungs often occurs during a violent paroxysm. The face becomes puffy, and ulcers form on the tongue; and hemorrhages occur into the conjunctivæ. The subsidence of the paroxysm is usually followed by a sense of exhaustion, with soreness about the muscles of the chest, and expectoration of whitish, viscid mucus.

A *physical examination* of the chest during a paroxysm of whooping cough shows a feeble or absent respiratory murmur over the whole chest, with sibilant and sonorous râles; during the interval mucous râles are usually heard. The frequency and duration of the paroxysm vary greatly in different cases. There may be one hundred in twenty-four hours. They are most frequent, or occur only at night. As a rule the more violent the paroxysm the sooner it is followed by another. The disease usually attains its height by the end of the fourth or fifth week. In mild cases the patient is well in the interval between the paroxysms, but in severe cases there may be languor and debility, loss of appetite, headache, and more or less fever. Moist or dry crepitations and a weak inspiratory sound are often heard during the interval.

The *stage of decline* is marked not by any sudden transition, but by a gradual diminution in the frequency and severity of the paroxysms. The peculiar whoop ceases, the expectoration is less difficult and becomes more purulent in character, and finally, after a period of about nine weeks, the characteristic cough ceases altogether, and the patient passes into a rapid convalescence. Whenever the coughing fits lose their characteristic features and become dry and hacking, and the dyspnœa is greatly increased and continues through the intervals with a marked rise in the temperature, it indicates some pulmonary complication. Another complication which is particularly to be feared in this disease is cerebral congestion. When, during a paroxysm, the countenance becomes flushed and swollen, the jugular veins turgid, with a gush of blood from the nose, there is danger of such an occurrence. When the face is continually flushed, the head hot, the patient drowsy or restless in his sleep, moaning and grinding his teeth, there is danger of convulsions and coma, and the disease often terminates fatally.

Differential Diagnosis.—In its earlier stages, it is not possible to diagnosticate whooping-cough with certainty; but its existence may be suspected if the cough is of a violent spasmodic character, and if the disease is prevalent. When the disease is fully established, the peculiar cough and expectoration distinguish it from all other catarrhs.

Prognosis.—Whooping-cough is always a serious disease, although it is rarely directly fatal; yet indirectly it frequently causes death. It is dangerous in proportion to the number and severity of the paroxysms, the intensity of the fever, and the character and severity of the complications. Cerebral or pulmonary complications are always dangerous. Teething children are liable to convulsions during paroxysms of the coughing. Death may result from laryngeal spasm independent of complications. A condition of general debility, rickets, poverty and destitution, a residence in a city in badly ventilated apartments, and epidemic influences, tend to render the prognosis unfavorable.

Treatment.—The chief indications in the treatment of whooping-cough are, *first,* to diminish the severity of the paroxysms; *second,* to prevent and treat as far as possible the complications; *third,* to attend to the general health of the patient. There are no known means by which this affection may be averted. The paroxysms cannot be altogether prevented, but their severity may be lessened.

All of the internal and external specifics for the prevention of the paroxysms of whooping-cough, which have been proposed, and in some instances strongly advocated, are of very doubtful benefit. The most important and reliable remedies for relieving the paroxysms of coughing are the sedatives and antispasmodics, the most efficient of which are belladonna, hydrocyanic acid, hydrate of chloral, hyoscyamus, cannabis indica, the bromides, chloroform and musk; all of these remedies must be given in minute doses, and their effects closely watched. Recently antipyrine and quinine have been strongly recommended. The dilute mineral acids, arsenic, nux vomica, cochineal, bromide of potassium, and repeated emetics—emetics are no longer given—have each in turn been highly recommended as specifics for the control of the paroxysms in whooping cough. Alum is recommended by Golding Bird (gr. i–v every four hours) and Meigs[1] says it is the best remedy. No form of opium or belladonna is to be used till after the catarrhal stage is past. Infusion of chestnut leaves is regarded highly. Inhalation of coal gas is recommended by the French Academy of Medicine. Ergot, the *carbolic acid spray,* asafœtida, arsenic and quinine are highly efficacious,[2] the second and last especially. The nitrite of amyl and jaborandi are drugs that I would not give to very young children. Local applications to the larynx, such as solution of nitrate of silver, etc., according to my experience, do more harm than good; and the same is true of counter-irritants, such as liniments and plasters.

I desire to impress this fact, that whooping-cough is a self-limiting disease, and, like all other diseases of that class, must be treated expectantly. The patient, by warm clothing, should guard against undue exposure. In bad weather, he should be confined to the house in a room of uniform temperature; but there is no reason, if the weather is favorable, why he should not go out into the open air. The diet should be simple, and the state of the alimentary canal carefully looked after. Adults and older children should be taught to suppress the cough as much as possible.

Complications must be watched for, and treated as soon as they occur. Bronchitis is the most frequent complication; when it occurs it should receive prompt attention, according to the rules already given for the management of bronchitis, great care being taken that it does not become a broncho-pneumonia. If the symptoms of congestion of the brain or of pneumonia are developed, they should be met by the most prompt and efficient remedies adapted to these conditions, and their earliest appearance should be watched for. It is important to remember that in any or all of the complications of whooping-cough, the treatment should be supporting in character.

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[1] *Dis. of Children.*

[2] Binz and Squire.

During convalescence, tonics, such as iron, quinine and cod-liver oil, are indicated ; in fact, in a large proportion of cases these remedies are serviceable throughout the whole course of the disease. Astringents and restoratives are called for in the third stage and at the commencement of convalescence. Sometimes this affection assumes a chronic form, continuing after several relapses much beyond the usual period. In these cases, the great remedy is change of air. In all stages of whooping-cough, benefit is derived from a short sea-voyage and a temporary residence in a warm climate. It has been recently stated by some very judicious observers, that large doses of the sulphate of quinine have the power of aborting this disease. My experience in this direction is not sufficient to deny or sustain the statement ; but my impression is that this, like many other so-called specifics, after a more extended trial, will be found unavailing.

HYDROPHOBIA.

Hydrophobia, or *rabies*, is a specific contagious disease special to animals of the canine and feline species, which may be communicated to man and to all warm-blooded animals.

Morbid Anatomy.—There are no constant pathological changes. The mucous membranes of the alimentary and respiratory tracts, especially of the fauces and pharynx, are congested, œdematous, and possibly show points of hemorrhage. The tongue, tonsils, and the salivary glands are enlarged and softened, and the lungs and other internal organs are congested.

Recent investigations have shown[1] congestion of the nervous centres, most marked about the basal ganglia, the medulla and the gray matter of the cervical cord. This is accompanied by a diffuse cellular infiltration of the adventitia of the veins, with venous injection and thrombosis. Miliary aneurisms and minute hemorrhages have also been noted in the medulla, cervical and dorsal regions of the cord.[2]

The blood is dark, forming soft clots, and putrefactive changes appear early after death.

Etiology.—The cause of the disease is a specific virus which is most abundant and concentrated in the saliva and secretions of the mouth and pharynx. Pasteur's experiments show that the poison is present abundantly in the nervous centres, and particularly in the spinal cord.

The poison retains its vitality for some time after the death of the affected animal. Although not proven, it is probable that the disease is never of spontaneous origin, but spreads among animals by contagion. It certainly is communicated to man solely by inoculation, which can take place only through some break in the surface. Applied to the skin or swallowed the virus is inert.[1]

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[1] Fitz and Shattuck.

[2] Benedict considers the essential pathological change in the nerve centres to be "an acute exudative inflammation attended by hyaloid degeneration."

Symptoms.—As in other infectious diseases, there is a period of latency following the inoculation, during which the wound heals readily and presents no peculiarities. This period of incubation varies from a few days to several months, and in some cases even to years. It is seldom, however, that the disease appears after five months, and usually within two to six weeks the stage of invasion begins.

This may or may not have been preceded by slight reddening about the seat of the inoculation, with pain which radiates from the wound along the nerve trunks. In a few cases the inflammation causes suppuration and reopening of the wound.

The period of invasion, or *melancholic stage*, is attended by marked depression of spirits and change in the disposition. The patient is feverish and shivering alternately, is restless, uneasy and sleepless, and speaks in a sharp, quick manner. The pupils are dilated and the eyes bright, and the countenance has a look of anxious anticipation of some unknown danger. The pulse is increased in frequency, the skin dry, and there is constipation, with perhaps nausea and vomiting. In this stage the respiration is oppressed, and shows evidences of the approaching spasms. There is epigastric heaviness, and with inspiration the shoulders are elevated and the epigastrium protuberant.

There may be also slight constriction of the throat and hesitancy in swallowing, with general hyperæsthesia and sexual excitement.

These symptoms increase in severity for two or three days, when the patient passes into the *convulsive stage*.

The restlessness and undefined dread are more marked, the eyes have a wild look, are bright, staring, and constantly moving; the brows are contracted, the surface pale, and the patient not only often appears like one with acute mania, but the fear and horror may pass on to halluciations and delirium.

The mouth and fauces are dry, congested, and covered with thick, tenacious saliva, which gathers about the lips in frothy masses. Thirst is intense, but every effort the patient makes to drink, and later the sight of water or thought of drinking is followed by increase of the pharyngeal constriction at first, and later by violent spasms of the muscles of deglutition and respiration, attended by general tremors and most terrific mental distress.

At first the convulsions only follow attempts at drinking, but the general hyperæsthesia increases rapidly and becomes so intense that the weight of the clothes, loud harsh sounds, bright lights, or a draught of cold air will excite general convulsions that leave the patient utterly exhausted and with the most agonizing horror of their repetition.

In some cases death occurs early in the disease from asphyxia during a spasm, but more commonly as the symptoms increase in severity the patient is rapidly exhausted; the pulse becomes feeble, frequent, and irregular, and as the spasms are more prolonged, he may die from gradual asphyxia or exhaustion.

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[1] Pasteur's experiments show that inoculation with a diluted virus affords protection from the actual disease. Not over seventy per cent. of those bitten by rabid animals become hydrophobic, owing, doubtless, in many cases to the cleansing which the fangs receive as they pass through the clothing.

In rare cases a *paraplegic stage* is said to occur, in which the paralysis is most marked in the under jaw and lower limbs.

The "hydrophobia" which is so characteristic of the disease as to give it a name, is due entirely to fear of the distressing spasm which every effort at swallowing produces, and is generally absent in dogs and other animals. For the same reason the patient is continually hawking and spitting out the thick, ropy mucus which is so abundantly secreted.

The peculiar characteristics of the disease are the intense hyperæsthesia of the skin and organs of special sense; the exalted reflex irritability of the nervous centres, which results in the peculiar spasms; and the paroxysmal rabid impulses that lead the patient to injure, it may be, his dearest friends, even when he is conscious of the nature of his frenzy and is struggling against it.

Differential Diagnosis.—Hydrophobia may be confounded with *tetanus*, but in tetanus the mind is clear throughout, there is no fear of liquids, the spasms are tonic and the hyperæsthesia is not so acute, nor does it involve the special senses.

In *hysteria* the difficulty in swallowing is the only symptom of hydrophobia, and the expressions of fear are out of all proportion to the other symptoms. A spurious rabies may be developed by the imagination in patients, who suppose they have been bitten by a rabid animal, but the course of the disease, its milder symptoms and favorable termination, will readily distinguish it.

Prognosis.—Most authors regard the disease as absolutely fatal, and in tables of cases which record a small per cent. of recoveries the possible hysterical nature of these cases must be considered. I have never known a case to recover.

The duration of the disease is from two to ten days, but in rare cases may be extended to two or three weeks. Death usually occurs from asphyxia, rarely from exhaustion or inanition.

Treatment.—When it can be done immediately, if the injury is upon a limb, a tight ligature should be applied above the wound, which is then to be widely excised and the part cupped. Venous hemorrhage should be encouraged.

Of the many remedies proposed, curare offers the most encouragement. It should be given hypodermically, in doses of one-third grain every fifteen minutes, increasing until the spasms are controlled. Recovery has been reported in one case where it was used. Pasteur claims that the developments of the disease may be prevented and its progress arrested by inoculations with an attenuated hydrophobic virus derived from the spinal cord of an affected animal. His claims have received the support of a large number of careful scientific observers.

MALARIAL FEVER.

Introduction.

The different varieties of malarial fever are like different branches of the same tree; they have many things in common, yet differ from each other so widely in the phenomena which attend their development, that they may be regarded as distinct diseases. They have a common origin in a poison which has received the name of miasm.[1] All varieties of malarial fever depend upon one and the same poison, which is subject to certain variations in quantity. The concentration of this poison determines the severity and, to a certain extent, the type of the fever. It is possible to arrange the different types in a progressive scale, from the mildest to the most severe, beginning with simple intermittent and passing on to pernicious fever. The extent of the morbid processes and the rapidity with which they are developed depend upon the intensity of the malarial poison, the length of time the individual has been under its influence, and, to some extent, upon individual idiosyncrasies.

Many theories have been advanced as to the nature of this *miasm* or malarial poison. By some it is regarded as gaseous in its nature; others believe it to be a living vegetable organism; and again, others think it a specific poison, having no tangible, chemical or microscopical constituents. No one of these theories, nor any of the many others which at different times have been advanced, has been sustained either by fact or by reliable chemical or microscopical analysis. In 1881 Lavaran, and later, in 1883, Marchiafava and Celli, described certain protoplasmic bodies in the red blood corpuscles of malarial patients, to which the name "plasmodium sanguinis malariæ" was given. It was claimed that these bodies were found only in connection with malarial poison. Their presence in a proportion of cases seems established, but it is entirely undetermined whether they are normal, or are the cause or result of the malarial infection. While we have *no positive knowledge* in regard to the true nature of malaria, we do know something of the circumstances which are necessary for its production, and the laws which regulate its development.

First.—There must be a certain amount of vegetable matter, either on the surface or in the substance of the soil where the malarial poison is generated. It is not necessary that the quantity be large, but a certain amount is a necessity.

Second.—A certain amount of moisture must be on the surface or in the substance of the soil; it need not be excessive; but some is indispensable.

Third.—A certain average degree of temperature is necessary for its production. It cannot be developed below an average temperature of 58° F. for the twenty-four hours, and will not prevail as an epidemic unless the average temperature ranges as high as 65° F. for the twenty-four hours.

In regions where these fevers prevail, their type, form and intensity, to a great degree, depend upon the height of the temperature. As a rule, malarial fevers are endemic, rarely extending over large sections of country in the form of an epidemic. We also have some knowledge concerning the regions in which malarial fevers are most likely to prevail, and which seem most favorable to the development of malarial poison.

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[1] "Telluric poison" is a term which has recently come into vogue.

First.—Marshes are especially favorable to the development of this poison, and may generate it for an indefinite period. The Pontine marshes have been malarial for more than two thousand years. Yet all marshes are not malarial; their power to generate the malarial poison varies with the amount of water they contain. Where there is an abundance of water, malarial fevers are rare; when they are covered only with a thin sheet of water, and are exposed to the direct rays of the sun, malarial poison will abound. Marshes that have dried up are especially favorable to the development of this poison, yet as soon as heavy rains submerge the previously parched surface, the power to generate the poison is for a time diminished or entirely arrested. Scattered here and there over our own continent are districts which have been malarial ever since the white man has held possession of them; whether such was the case in earlier times, history is too uncertain to determine.

As a rule, salt-water marshes are especially free from malaria, but when salt and fresh water are mixed in the marsh, the most favorable conditions for the abundant development of malaria occur. Marshes that rest on a substratum of sand are far less malarial than those resting on limestone, clay, or mud. There are marshes in the higher latitude of New York and other States which often, during the heat of summer, become dry, yet no malarial poison is generated (although during the day the thermometer may reach 90° F.), since during the night the temperature falls below 50° F. There are some quite extensive marshes in which, apparently, every condition of development of malaria exists, and yet none is generated. We cannot account for this fact unless we accept the theory that the ozone which is claimed to be present in such marshes arrests or prevents its generation. Damp "bottom lands" that are exposed to an annual overflow, such as are found along the southern shores of the Mississippi River, are as fruitful as swampy regions in the generation of this poison.

Second.—Another condition which seems to favor the development of malaria is the upheaval of new alluvial soils, such as obtains when new lands are first brought under cultivation. This same state of things also occurs throughout the middle and southern portions of this State, and in the New England States. Where railroad excavations are made, malarial fever is very frequently developed. In New York City, while the Fourth Avenue improvements were being made, the entire region along the avenue was rendered highly malarious by the excavations. Such excavations bring decomposing vegetable matters to the surface; these, under the influence of heat and moisture, generate miasm.

The fact that fevers of this type appear in regions previously free from them, as soon as the conditions favorable to their development exist, is confirmed by the testimony of many careful observers.

Third.—Regions otherwise non-malarial may have malarial poison brought to them by the waters of rivers which have their source in, or flow through, malarial districts. Examples of this kind are found along the banks of our Western rivers, where some of the most pernicious types of this fever are developed; while in places only a short distance from these rivers it is unknown.

Fourth.—Non-malarial regions may be rendered malarial from poison transmitted by the wind. There has been considerable discussion as to whether this poison can be transmitted in such a manner, and if it can be, to what distance; there is no reliable account of its transmission over a greater distance than four and three-quarter miles.[1] The wind may also carry malarial poison up along the sides of mountains, to an elevation of one thousand feet; some writers say no higher than six hundred feet. American writers give an account of its being carried higher than six hundred feet, while some German writers give well-authenticated cases which show that it must have been carried to the height of one or two thousand feet.

The circumstances which are inimical to its production are:

First.—High latitude. In this country malarial poison is not generated in higher latitude than that of Quebec. The limit of its development is 63° north and 57° south latitude. Between these two parallels of latitude, both on the eastern and western hemispheres, malarial fevers may be developed; the nearer the approach to the equator, the more severe the type. They do not prevail over the entire region embraced between these parallels of latitude, but it is possible for them to be developed at any point where the altitude is not too great.

Second.—High elevation is another condition inimical to its development. As a rule it is not generated above an elevation of one thousand feet above the sea. There are, however, some remarkable exceptions to this rule. We find recorded cases of malarial fever which have been developed upon plateaus among the Pyrenees, at an altitude of five thousand feet, and in South America at ten and eleven thousand feet above the sea-level. Among the Pyrenees, there is a marsh which has a clay bottom, and there malarial poison is developed which is very persistent.

Third.—Drainage is another means which diminishes, and in certain conformations of soil entirely destroys, malarial generation. In the majority of marshes, this generation can be arrested or prevented by free drainage. Yet there are marshes upon which millions have been expended in drainage and which still remain pestiferous. Perhaps it is possible to drain the Jersey flats so as to render them non-malarial in their character, out it is hardly probable that this change can be effected, for they have a clay bottom, and contain both salt and fresh water, conditions which are most favorable to malarial generation. Years of labor and large expenditures of money have been bestowed upon the Pontine marshes to render them non-malarial, yet they are as pestiferous as they were twenty centuries ago.

Fourth.—Cold is a powerful agent in arresting malarial generation. If, in a pestiferous region, the temperature should fall below the freezing point, only for one night, nothing more need be feared in that region from malaria, until the average temperature shall have again reached 60° F. This law holds in all malarial districts. In these districts, after the temperature has fallen below the freezing point, persons may have the fever, but it is the result of previous poisoning. Again, the generation is less rapid and the poison is less virulent during the day than at night. This is the uniform testimony of those who have seen most of, and written most on malarial diseases. It is also almost universally conceded that malarial districts are most pestiferous during months when the atmosphere is hot and dry, with little or no wind, especially when this state of atmosphere has been preceded by long, heavy rains, and that the virulence of the poison is greatly diminished as soon as fresh, strong winds clear the atmosphere.

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[1] Malarial fever broke out in the crew of a ship, which was anchored just four and three-quarter miles from shore where this fever was prevailing. No cases were on board when the anchor was cast, nor did any of the crew go on shore. So long as the wind blew from the ship towards shore, the crew remained well, but when the wind changed its direction and blew from the shore towards the ship, within six days from the time of change, cases of well developed malarial fever appeared on board. This seemed to prove conclusively that the fever was brought to the ship by the wind.

The question arises : How does malarial poison gain entrance into the human body ? The most reasonable view is that this is effected through the respired air. Certain facts seem to show that it may be introduced through the intestinal tract with the food and water. There seems to be scarcely a doubt but that it may be taken into the stomach with foul drinking-water. When this poison has once been introduced into the circulation, it undoubtedly has the power of reproducing itself. From this fact, which must be regarded as well established, these who regard this poison as a living organism, claim that these organisms may reproduce themselves indefinitely, but this has never yet been demonstrated.[1]

It has also been claimed that certain races are more exempt than others from malarial fever, also that there are idiosyncrasies of constitution which render certain individuals exempt from diseases of this type, for in districts where these fevers prevail there are persons who never have the fever. This exception, both in races and individuals, is due to the greater physical power of the individual, which enables him to resist these noxious atmospheric influences. In a district where malarial influences prevail, the weak and anæmic are the most liable to be attacked, and all those influences which tend to lower vitality, and to render feeble the powers of resistance, must be regarded as special predisposing causes. A strong man may resist for a long time, while the old and children very quickly succumb to the influence of the poison. Women are more susceptible than men. We can no more account for the fact that one person can take in large doses of malarial poison without being affected by it, while another is affected by a very small quantity, than we can account for the fact that one person can take large quantities of alcoholic stimulants without showing any signs of intoxication, while a very small quantity will intoxicate another, supposing, in both instances, the individuals to have apparently an equally vigorous constitution. Some claim that when a person has been poisoned with malaria, complete recovery never takes place; others that even in the worst cases recovery is possible. My own experience leads me to believe that when an individual has once suffered from malarial poisoning, he is much more susceptible to the poison than one who has never been so poisoned. Some unknown physical change has taken place which renders him a fit subject for malarial manifestations upon the slightest exposure.

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[1] 1880. Recently Crudell (of Rome) and Klebs (of Prague) examined the lower strata of air, the soil, and the stagnant water, and in the two former they found a "microscopic fungus," consisting of numerous movable shining spores, long and oval, nine micro-millimeters in diameter. This fungus was cultivated and when inoculated into animals they all had a regular typical *chills and fever* with an *enlarged spleen*. They call this the *bacillus malaria*. Roman physicians now claim that they have found this in the *human subject*. It is said to be always in the blood during the period of invasion. The spleen and the marrow of the bones are its favorite seats.

The doctrine of latency of malarial poison in the human body is an interesting and at the same time a very obscure subject. That there is a period of incubation, or rather that a certain time elapses between the exposure and the development of malarial fever, seems to be settled. For a certain period, often a long one, always elapses before new-comers in a malarial district have their first attack of the fever; sometimes the poison remains latent until after they have removed from the district. It is on the basis of the latency of the malarial poison that the relapses can be accounted for which occur in those who, having lived in a malarial district, remove and remain in a non-malarial one. This reawakening of the malarial poison may depend upon a variety of causes, such as taking cold, over-fatigue, sudden changes of temperature, etc. From my own observation, I am convinced that it is impossible to bring one wholly from under the influence of the poison while remaining in a malarial district, though he may become exempt from its influence if he remains beyond the malarial belt. Undoubtedly, an individual may become so acclimated as to resist malarial influences, and live for a long time in a malarial district without suffering any evil effects from it. There can be no question but that those living in such districts suffer less from the acute manifestations of the poisoning than new-comers. But those changes which are called chronic malarial manifestations are constantly going on in those who are supposed to be acclimated.

Malaria acts like any other poison: after a time the system reaches a certain degree of tolerance. This tolerance, or immunity from its manifestations, amounts to nothing more than the accommodation of the system to its influence. Let the acclimated person, as he is called, be taken sick with any active form of disease, such as diphtheria or pneumonia, and it usually proves fatal, not that there is anything unusually severe in the diphtheria or pneumonia, but death is due to the fact that the system is depressed by the malarial poison, and its power of resistance to disease is lessened.

INTERMITTENT FEVER.

Like typhoid fever, intermittent fever is met with in all parts of the world, although the region of its development may be said to be limited by 63° north and 57° south latitude. Within these parallels it is the more prevalent nearer the equator.

Morbid Anatomy.—Its anatomical changes are few. None of those changes in the blood which are present in the more severe forms of infectious diseases are found, those which are present in the pernicious type of malarial fever, such as pigmentation and marked diminution in the red globules are much less prominent. Some disintegration of the hæmoglobin and corpuscular pigmentation, however, will be developed when the fever is distinctly established. But the blood clots imperfectly, and is of an abnormally dark color, and if the fever has continued for a long time there may be slight diminution in the number of the red globules and a decrease in the fibrin-factors ; but these changes, to a great extent, are due to the high temperature which attends its paroxysms.

The only constant pathological lesion of intermittent fever is congestion of the internal organs. The spleen and liver are always more or less enlarged, but the enlargement is due to simple hyperæmia; no structural changes occur in these organs until the intermittent paroxysms have been often repeated, and the malarial poisoning has been of long duration. There is also more or less hyperæmia of the kidneys and the mucous membrane of the intestines, but it is not attended by any signs of gastric or intestinal catarrh. As yet no one has been able to prove that any structural change takes place either in the nerve tissue or in any other tissue of the body; nor from the structural or functional disturbances that occur during the fever has any one been able to find satisfactory answer to the question: why is it a paroxysmal and not a continued fever? During a paroxysm of the fever the white blood-globules are very rapidly increased in number.

Etiology.—This subject has just been considered. All agree that simple intermittent fever is due to malarial poisoning, and that the poison is introduced into the body either through the lungs or through the intestinal tract. Whatever tends to depress the mental or physical powers of an individual renders him more susceptible to malarial influences, and consequently these depressing influences must be regarded as predisposing causes. Among these may be included intemperance, exposure to night air, excessive fatigue, bad hygiene, and a long list of like debilitating causes.

Symptoms.—This fever is paroxysmal, and differs in its types according to the period of time which intervenes between the paroxysms. The *first*, and most common, is the *quotidian* type, in which the paroxysm occurs every day, and there is an interval of twenty-four hours between the paroxysms.

The *second* is the *tertian* type, in which the paroxysm occurs every third day, with an interval of forty-eight hours between the paroxysms.

The *third* is the *quartan*[1] type, in which the paroxysm occurs every fourth day, with an interval of three days or seventy-two hours between the paroxysms. These are the regular and more common types.

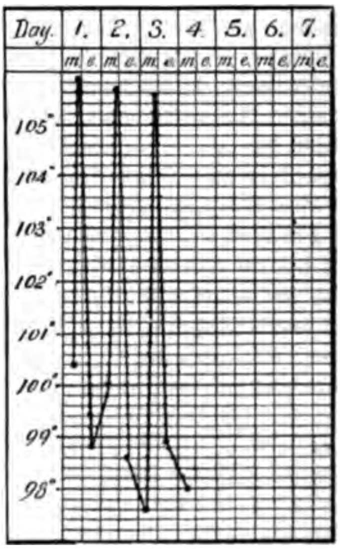

FIG. 172.
Fever Curve in Quotidian Intermittent.

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[1] In 98,287 cases in the U. S. army, only 1,737 were quartan.

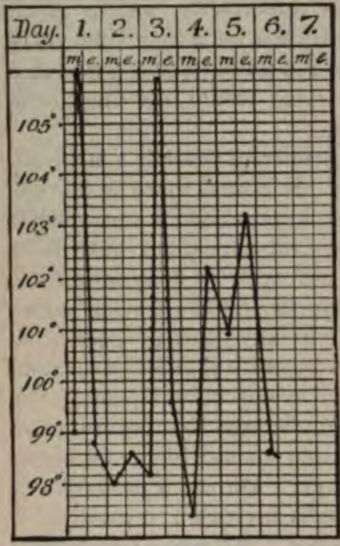

FIG. 173.
Fever Curve in Tertian Intermittent.

Other types exist, which, although irregular, are unquestionably modifications of those already mentioned. Among these is the *double quotidian*, in which two paroxysms occur daily. Usually one paroxysm is severe, the other mild; the severer one generally occurs in the morning, the milder in the afternoon or evening. There is also *double tertian*, in which a paroxysm occurs daily, but it differs from the quotidian as the paroxysms that resemble each other occur at intervals of forty-eight hours. For instance, the paroxysm of to-day is characterized by the occurrence of a severe chill and mild fever; to-morrow it is characterized by a short chill and severe fever; the following day there occurs the severe chill and mild fever, as on the first day.

A form of intermittent fever is met with in which the paroxysm occurs on the seventh, fourteenth, twenty-first day, etc., with an interval of seven days between the paroxysms. The types most frequently met with are the quotidian, tertian and quartan. In the quotidian variety the paroxysm occurs in the morning, in the tertian it occurs about noon, while in the quartan it occurs in the afternoon or evening. The duration of the paroxysm varies with the type of the fever. In the quotidian it lasts from eight to ten hours, in the tertian it lasts from six to eight hours, in the quartan from four to six hours. There are many exceptions to these rules, but it is a question whether there would be any if the disease was permitted to run its course without treatment.

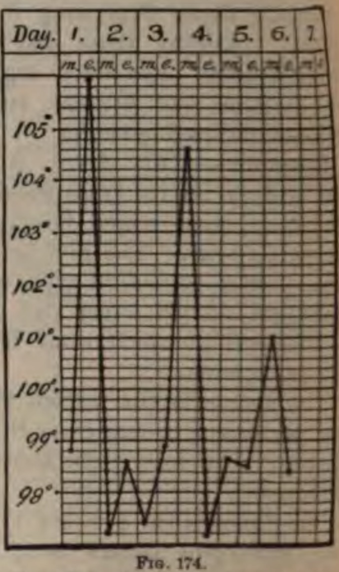

FIG. 174.
Fever Curve in Quartan Intermittent.

Paroxysms.—A paroxysm of intermittent fever has three stages—the *cold* stage, the *hot* stage, and the *stage of sweating*. In most cases these are readily distinguished from one another. In the true type of intermittent fever regular intervals between the paroxysms of fever occur. The phenomena which usually precede the cold stage are pain in the head, a sense of languor, and some nausea.

Cold Stage.—Passage into this stage is first marked by a sensation of coldness along the back, which soon extends to the extremities, and an uncomfortable sensation of coldness gradually creeps over the entire body. The skin becomes shrivelled, the finger ends and lips become blue, the face is pale, the eyes are sunken, chills rapidly follow each other, the teeth begin to chatter, any voluntary motion is attended by trembling, until finally, as one chill after another in quick succession passes over the body, the teeth chatter so that the noise can be heard some distance from the patient, and there is a shaking of the entire body. The surface of the body becomes rough, the blood seems to recede from it, and it assumes the appearance of *goose-skin* or *cutis anserina.* The temperature of the surface of the body is lower than normal, but if the thermometer be placed in the axilla or under the tongue the temperature marks 104° or 105° F. The voice of the patient is weak and husky; the respirations are rapid, short and sighing, but the mind remains clear. The urine is increased in quantity and paler than normal, and there is frequent desire to empty the bladder. Usually these symptoms last from half an hour to two or three hours; the length of time depends upon the severity of the case. Children do not have a regular chill; they merely grow cold, blue and livid.

After the cold stage has continued for a longer or a shorter period, the patient begins to have flashes of heat alternating with the chilly sensations. Usually these are first felt at the extremities, but they rapidly extend over the whole body, and the *hot stage* is established.

Hot Stage.—The skin is now no longer shrivelled, but becomes red, swollen and turgid, and there is a recession of the blood from the central organs to the surface of the body. That the temperature is elevated cannot be ascertained simply by laying the hand upon the surface. If, however, the thermometer is placed in the axilla, in most cases the temperature marks 106° or 107° F. Thirst is intense. The uncomfortable sensation which the patient experienced while passing from the cold to the hot stage, has given place to great restlessness, the patient tossing from side to side, with face flushed and eyes red and fiery. Sometimes herpetic vesicles appear about the mouth. The tongue becomes dry, the carotids pulsate, the radial pulse becomes firmer and more rapid than in the cold stage, and nausea is marked. It may have been present in the cold stage, but in the hot stage nausea and vomiting become the prominent symptoms. As a rule these symptoms last from half an hour to two hours. In exceptional cases they may continue for a much longer time, the ordinary duration of a paroxysm of a quotidian intermittent being from eight to ten hours; that of a tertian, from six to eight hours; and that of a quartan, from four to six hours. It is possible, especially in those forms of malarial fever in which the poisoning is intense, for the hot stage of a quotidian to continue twelve hours.

There is no condition in which, for the time, there is more intense fever than in the hot stage of intermittent fever. The urine, which during the cold stage was abundant and of a pale color, now becomes highly colored and scanty. Not infrequently it is almost suppressed during the hot stage. Complete suppression of urine occurs only in the pernicious type of the disease. When the fever has continued for a longer or shorter time, a slight moisture appears upon the forehead, which gradually spreads over the entire body, and the patient becomes bathed in a profuse perspiration. He is now in the *sweating stage.* In children, just before the sweat, coma or convulsions may occur.

Sweating Stage.—As this stage comes on, restlessness and uneasiness decline, and a feeling of comfort is experienced as the perspiration makes its appearance. The temperature rapidly falls; the pulse rapidly diminishes in frequency and force; the pulsation of the carotids ceases; the face assumes its normal appearance; the congestion of the conjunctivæ disappears; and the patient rapidly passes from a high state of fever into one in which he falls asleep, and awakens after a period ranging from one to three hours, with a sense of exhaustion.

Interval.—During the interval between the paroxysms at first the patient may feel perfectly well, but if there is a frequent repetition of the paroxysms, there will very soon be a marked loss of vitality; he becomes pale and feeble, and all the symptoms of malarial cachexia are present. There will be more or less of a jaundiced hue to the skin, enlargement of the spleen and liver, and pigmentation of the tissues. It is true that many paroxysms of simple intermittent may occur before any such general disturbance of the health of the patient manifests itself; yet, in the interval between the paroxysms, we cannot call the patient's condition one of perfect health. Usually, in the quotidian type, the day previous to the development of the first paroxysm, unnoticed by the patient, there is a slight rise in temperature, perhaps to 101° F. At the same time he experiences a sense of lassitude, and is disinclined to make any exertion, either mental or physical. The temperature commences to rise in the morning, and by noon it has reached its maximum; then it begins to fall, and by evening it may have fallen to nearly its normal standard. Thus the course of the temperature is quite characteristic, and may be summed up as a rapid ascent, a short and intense stationary period, and a critical defervescence constituting the paroxysm, with a perfectly normal temperature in the interval. The following day another rise in temperature will be noticed; now the rise does not occur in the morning, but after midday, perhaps as late as in the evening. Usually in the quotidian type of intermittent fever the highest temperature is reached a little earlier each day; if it is reached a little later, the fever is being modified or controlled by treatment.

When the paroxysm comes on a little earlier each day, it is called *anticipating*, and indicates that the fever is not being controlled; when it comes on later each day it then indicates that the fever is being controlled, and is called a *postponing* intermittent. The types of intermittent fever which occur most frequently in temperate climates are the quotidian and the tertian. In those who have suffered repeatedly from intermittent fever, the disease is liable to run an irregular course, the paroxysms occurring on irregular days, and with irregular intervals. In children this fever shows certain deviations from the ordinary course. The paroxysms may be ushered in by convulsions, or by a period of stupor. Children rarely have the distinct chill; after a period varying from ten minues to half an hour, we have the hot stage of regular intermittent fever coming on, with all its attendant phenomena. The intermissions are rarely complete. The child loses his appetite and flesh, becomes irritable, and has a pale, waxen look, suffers from gastric and intestinal disturbances, and the intermittent very soon lapses into the remittent form.

Differential Diagnosis.—The differential diagnosis of simple intermittent fever is never very difficult. There are only two diseases which are liable to be mistaken for it, namely, *remittent fever*, and *pyæmia.*

It is readily distinguished from *remittent fever,* for in remittent fever there is never a complete *intermission,* whereas in intermittent there is always a period in which there is no fever. A careful thermometrical observation for twenty-four hours settles all questions in regard to it. There is also a regular development of the paroxysm in intermittent, which does not occur in remittent. In remittent, there is usually but one chill, while in intermittent a chill precedes each paroxysm of fever. When the chill and sweat are absent, but a sense of heat, malaise, headache and lassitude come on at pretty regular periods in a malarial district, the thermometer showing a pyrexia of 102° to 104° F., the patient is said to have "dumb-ague."

Prognosis.—The prognosis in simple intermittent fever is good. The possibility of the development of malarial cachexia must enter into the prognosis. When this occurs the case is more than one of simple intermittent fever; there is enlarged spleen and liver, with pigmentation of the tissues.

Treatment.—The treatment of intermittent fever is divided into that for the *paroxysm* and that for the *interval.*

The treatment for the paroxysm, in most cases, is simply to render the patient as comfortable as possible while passing through its various stages. At one time it was proposed to tourniquet the limbs, so as to prevent congestion of internal organs, and thus arrest the paroxysms. Again, it has been proposed to apply cold to the surface for the purpose of giving a shock to the nervous system, and in that manner to arrest the paroxysm. Some propose to cover the surface of the body with sinapisms, in order to irritate the cutaneous surface. Some have claimed that if an individual is brought fully under the influence of alcohol the regular development of a paroxysm can be prevented. It has also been claimed that opium, given in full doses at the usual time for the recurrence of the paroxysm, has power to prevent it. Experience does not lead me to accept any of these statements. It is true that, in some instances, a sudden shock to the nervous system may prevent the development of an intermittent paroxysm when the paroxysms have become a habit.

If there is anything in the entire list of means (either remedial or hygienic) which has power to prevent the full development of a paroxysm, it is opium. When this is administered hypodermically, early in the cold stage, it will diminish the severity of the cold and hot stages. Whether, in the treatment of the milder forms of intermittent fever the combination of opium with quinine is advisable, is still an unanswered question, though it seems to me that in such cases much comfort can be afforded, and the patient be much less injuriously affected by the paroxysm if opium be administered in moderate doses. Patients with intermittent fever should be kept in bed during the entire paroxysm, however mild it may be.

During the cold stage, cover them with blankets, surround them with bottles of hot water, and let them drink freely of hot water. All these means will hasten the hot stage of the disease.

During the hot stage, the extra clothing and external heat should be gradually removed and cold instead of hot drinks should be administered. If nausea and vomiting are present in this stage, opium, administered hypodermically, affords great relief. When the patient reaches the sweating stage, let him alone ; within a few hours he will be entirely relieved and in a state of convalescence.

The treatment of the *interval* is to prevent the occurrence of another paroxysm. A patient should never be allowed to have a second intermittent paroxysm ; for if the system once becomes accustomed to these paroxysms, they will be repeated upon the slightest provocation. This will be found to be the case with those who for a long time have not been subjected to malarial influence, and yet upon the least nervous excitement or fatigue will have a paroxysm. The great remedy at this time is the *sulphate of quinine.* Skilfully used, it is all-powerful to accomplish this result. How and why it arrests the development of these paroxysms is still unknown. Our knowledge of its antiperiodic power is purely empirical. There is much difference of opinion as to the mode in which it should be administered. In commencing the treatment of a case of intermittent fever, after the occurrence of the first paroxysm it is always safe to assume that the fever is of the quotidian type. At least thirty grains of quinine should be administered between the termination of one paroxysm and the hour when another is to be expected. The first dose of ten grains should be given toward the close of the sweating stage, and twenty grains about two hours before the time of the expected paroxysm. If possible, give the quinine in solution. If irritability of the stomach causes rejection of the quinine, it may be administered hypodermically, or by enema. Three grains administered hypodermically has about the same antiperiodic power as ten grains administered by the stomach. If one succeeds in preventing the occurrence of a second paroxysm much has been accomplished. Having prevented the occurrence of a second paroxysm, it is important that a moderate degree of cinchonism should be maintained for a number of days, by the daily administration of quinine in moderate doses. About two hours before the time of day at which the first paroxysm occurred, from ten to fifteen grains of quinine should be daily administered.

A patient should visit his physician *one month from the date of the first paroxysm*, for although he may not have had a fresh malarial exposure, there will be a strong tendency at this time to a repetition of the paroxysm, and it is important that at that time he should be again brought fully under the influence of quinine. If it is possible for him to remove from a malarial district a second paroxysm will almost certainly be prevented. If, however, the patient is not seen in his first paroxysm, and if he lives in a malarial district, sulphate of quinine, administered in the manner I have just recommended, may only prevent for a time the return of the paroxysm, and even complete cinchonism may fail to control it. The case should now be carefully examined in order to ascertain if there is not some condition present which interferes with the antiperiodic action of the quinine, such as hepatic or splenic hyperæmia. When careful percussion shows that the liver and spleen are increased in size, even after the administration of full doses of quinine, the administration of full doses of calomel with the quinine will increase the antiperiodic power of the latter, and diminish the percussion areas of these organs.

Occasionally, when full doses of quinine combined with calomel have failed to prevent a recurrence of a paroxysm, I have noticed an unusual excitement attending its development, and believing from this circumstance that owing to individual idiosyncrasies the malarial poison had a more than usual irritating effect upon the nervous system, I have accomplished the desired result by administering full doses of opium with the quinine. In fact, if the patient is of a highly sensitive, nervous organization, I never allow a second paroxysm to pass without administering a full dose of opium before the time when its return is to be expected. In all those cases which are called obstinate, we must ascertain why we fail to control the disease by the use of quinine. I rarely have administered arsenic in simple intermittent fever. If I fail to control the fever with quinine, after I have reduced splenic and hepatic congestion, controlled nervous irritability, and increased nutrition by the administration of iron and the moderate use of stimulants, I never succeed with arsenic. In some of the chronic forms of malarial manifestation, I have found arsenic of great service, but never in simple intermittent fever. Salicin, strychnia, piperine, eucalyptus, and hydrastia sometimes act antiperiodically when quinine fails.

Masked Intermittent.—In this connection should be mentioned a form of intermittent fever which has been designated as *masked* intermittent fever. For example, to-day a patient has a regular intermittent paroxysm, but to-morrow, instead of its recurrence, perhaps he suffers from the most intense neuralgia. This neuralgia may have its seat in an intercostal or in the sciatic nerve, or, perhaps, more frequently in the frontal branch of the ophthalmic division of the trigeminus. Some one nerve becomes involved and no other seems to be affected. In some cases, an intense hemicrania takes the place of the paroxysm. As a rule, these neuralgiæ have distinct intermissions, and so come to be regarded as masked forms of intermittent fever. Instead of a neuralgia, the patient may have an attack of asthma, or an attack of indigestion. Diarrhœa, dysentery, and sometimes hæmaturia and apparent suppression of the urine may take the place of a distinct intermittent fever paroxysm. Again, a patient may have a single well-defined chill, or even two chills, followed by most intense hemicrania, and then have no more for a long time ; but sooner or later he will have a well-defined intermittent paroxysm which will reveal the real nature of the disease.

REMITTENT FEVER.

This is a *continued fever, with diurnal exacerbations.* It is known by different names, such as Southern, Western, African, continued, bilious, acclimative, and remittent fever. The term remittent fever is the one more generally accepted.

Morbid Anatomy.—The anatomical lesions of remittent fever resemble those of intermittent fever; and the differences are in degree rather than in kind.

Unquestionably, both these types of fever are the result of malarial poisoning, and the same diminution of the red globules and the same changes in the fibrin factors occur in remittent as in intermittent. Yet there are other changes in the blood which are usually present in the former, and quite rare in the latter, namely, the presence of free pigment-granules. These pigment-granules are met with in some of the pernicious forms of intermittent fever; but in all cases of well-developed remittent fever they are present at some time during the progress of the disease. This pigmentation is due to hæmoglobin which has been liberated from the blood-corpuscles within the blood-vessels, and the coloring matter may remain either within the blood-corpuscles, which, after a time, become transformed into pigment-granules, or remain free in the fluid portion of the blood, or infiltrate the adjacent cells and tissues. It may be transformed into granular or crystalline hæmatoidin.

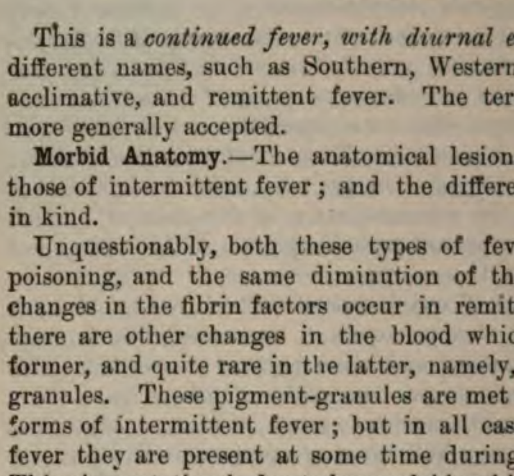

Fig. 175.

Section of the Liver from a case of Remittent Fever, showing part of a Lobule.

A. Central vein of the lobule.
B, B. Intralobular capillaries densely pigmented.
C. Hepatic cells, also containing pigment.

The *spleen* is not so much enlarged in remittent as in intermittent fever, and the increase in size seems to be of a different nature. The enlargement is evidently the result of congestion, and the organ sometimes presents very nearly the same appearance as it presents in typhoid fever, except that there is more pigmentation. There are also structural lesions found in the liver, in the stomach and in the intestines, which are not present in intermittent fever. The liver is not very much increased in size, and is of a *bronze* hue. The principal change is in color, which is uniform throughout its entire substance. This varies in degree in different types of the disease, and in different cases of the same type.

eculiar color is due to pigmentation of the liver-tissue, and varies ing to the amount of pigment deposited. Pigmentation may occur er tissues of the body, but not to the same extent as in the liver. On roscopical examination of the liver, pigment is found throughout its structure—not only in the hepatic cells, but in the nuclei of these and in the walls of the blood-vessels. This discoloration is of such m occurrence that it has been recognized as the characteristic path- al lesion of remittent fever. Consequently, the "*bronzed liver*" is of as the characteristic lesion of this fever. Occasionally this lesion in intermittent and pernicious fever, but this is so seldom, and its ce is so constant in remittent fever, that if met with at an autopsy ent fever may be suspected.

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re is probably no form of endemic disease the geographical bounda- which are more extensive than those of remittent and intermittent In general terms they may be said to encircle the earth parallel with uator, circumscribing a broad belt, limited by 63° north and by 57° latitude. The boundaries of this belt are quite irregular, now ap- ing the line of the tropics, now receding from it. The remittent which occurs within the temperate portions of this belt is much less than that which occurs in the tropical regions. From the localities ch this fever prevails, it would seem that a higher average tempera- required for its development than is required for the development of ittent fever. As already stated, a remittent fever during its con- nce may become an intermittent, and, conversely, an intermittent, from new exposure to malarial influences or to the influence of high rature, may become a remittent. From this fact, the conviction is upon us that both types of fever are developed from a common poi-

son. Usually certain atmospheric changes will have taken place to change the type of the fever. Intermittent fever may prevail early in the season, but as the season advances, and the temperature ranges higher, the fever which prevails will assume the remittent type. Those who go from a non-malarial district into one where remittent fever is prevailing are likely to have it, while the old inhabitants only suffer from the milder form of intermittent.

Symptoms.—Its ushering-in symptoms are usually well marked. The most constant as well as the most urgent of the premonitory symptoms is oppression in the epigastrium. This may be present for forty-eight hours, or even a longer time, prior to the development of the fever. There is also a certain lassitude, nausea, and loss of appetite ; and with these feelings uneasiness and perhaps pain in the head and limbs. It does not come on gradually, like typhoid fever, but abruptly, usually with a chill. It is not difficult to determine when the patient began to be sick. The chill is neither so complete nor so long continued as in intermittent fever or pneumonia. During the chill the thermometer will indicate a temperature two or three degrees above the normal. With the chill there is a most intense headache, and pain in the back and limbs. As a rule the chill is not of so long duration as the chill of intermittent, neither does it begin like it, by creeping down the back and gradually extending over the body, but there is general coldness over the entire surface at the very commencement of the chilly sensation. Again, there is not that tremulousness and shaking of the body, nor that chattering of the teeth, which are so frequently experienced in intermittent fever. Following the chill there is fever, during which the temperature rises very rapidly. The fever increases in severity, and, within twelve hours from the time of its commencement the temperature may reach 105° or 106° F. As soon as the temperature commences to rise, the pulse is increased in frequency, and perhaps beats 100 or 120 a minute. The face becomes flushed, the eyes are usually suffused, and the conjunctivæ are somewhat congested. The patient is restless, tossing in bed, in the vain search of an easy posture. As the hot stage advances, nausea and vomiting are always present, and the sense of oppression in the epigastrium increases, and is not relieved by vomiting, which is persistent and distressing.

In the febrile stage of remittent fever the patient suffers from pain in the epigastrium, to such an extent that quite commonly it is the only thing of which he complains. The epigastric distress is often accompanied by the most extreme tenderness upon pressure. The material first vomited simply consists of the contents of the stomach, next follows the vomiting of a greenish matter, and finally, in severe cases, there may be a slight amount of black vomit. The quantity of fluid vomited is greater than the quantity taken into the stomach. Vomiting of stringy mucus tinged with green is always present. Sometimes the patient's stomach rejects everything taken into it, and the vomiting is accompanied by intense pain in the head. Usually at the commencement of the fever, the bowels are constipated.

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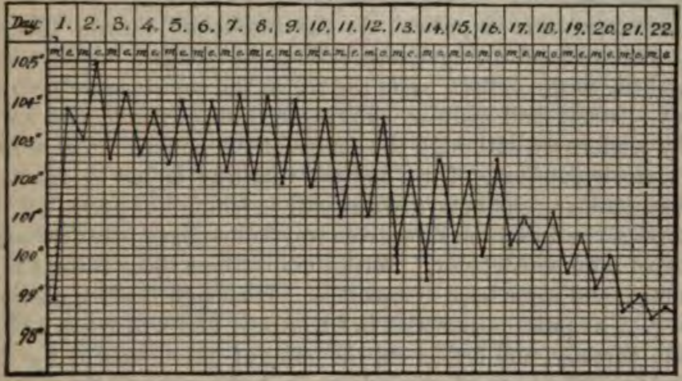

FIG. 176.
Temperature Record in a case of Remittent Fever ending in recovery.

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The peculiar color is due to pigmentation of the liver-tissue, and varies according to the amount of pigment deposited. Pigmentation may occur in other tissues of the body, but not to the same extent as in the liver. On a microscopical examination of the liver, pigment is found throughout its entire structure—not only in the hepatic cells, but in the nuclei of these cells, and in the walls of the blood-vessels. This discoloration is of such uniform occurrence that it has been recognized as the characteristic pathological lesion of remittent fever. Consequently, the "*bronzed liver*" is spoken of as the characteristic lesion of this fever. Occasionally this lesion occurs in intermittent and pernicious fever, but this is so seldom, and its presence is so constant in remittent fever, that if met with at an autopsy remittent fever may be suspected.

The mucous membrane of the *stomach* is more or less congested, thickened, and softened. Changes similar to those in typhoid are found in the mucous membrane of the intestines; it is more or less congested, and presents very much the appearance seen when a moderately severe catarrhal inflammation is present. The Peyerian patches are usually enlarged, and quite frequently present the shaven beard appearance. In some cases there are ulcerations, not, however, as extensive or of the same nature as the ulcerative processes of typhoid fever. The mesenteric glands are not enlarged, and there is none of that granular infiltration so noticeable in typhoid fever.

Etiology.—The great predisposing and exciting cause of this fever is malarial poisoning. There can be no question but that the same malarial poison which gives rise to intermittent fever can produce a remittent fever. In other words, a remittent can pass into an intermittent fever, and an intermittent into a remittent fever. While it is possible for this to occur, the two diseases do not, as a rule, prevail in the same locality at the same time. Endemics of one form may occur and be followed by endemics or sporadic cases of the other form. In some localities remittent fever is almost the only form of malarial disease, intermittent fever only occasionally occurring.

There is probably no form of endemic disease the geographical boundaries of which are more extensive than those of remittent and intermittent fever. In general terms they may be said to encircle the earth parallel with the equator, circumscribing a broad belt, limited by 63° north and by 57° south latitude. The boundaries of this belt are quite irregular, now approaching the line of the tropics, now receding from it. The remittent fever which occurs within the temperate portions of this belt is much less severe than that which occurs in the tropical regions. From the localities in which this fever prevails, it would seem that a higher average temperature is required for its development than is required for the development of intermittent fever. As already stated, a remittent fever during its convalescence may become an intermittent, and, conversely, an intermittent, either from new exposure to malarial influences or to the influence of high temperature, may become a remittent. From this fact, the conviction is forced upon us that both types of fever are developed from a common poi-

son. Usually certain atmospheric changes will have taken place to change the type of the fever. Intermittent fever may prevail early in the season, but as the season advances, and the temperature ranges higher, the fever which prevails will assume the remittent type. Those who go from a non-malarial district into one where remittent fever is prevailing are likely to have it, while the old inhabitants only suffer from the milder form of intermittent.

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The febrile symptoms increase in severity for ten or twelve hours, when a slight perspiration appears upon the forehead. In a short time, it extends over the entire body, not profuse, but a slight moisture upon the surface. With the perspiration there will be a fall of one or two degrees in temperature, and a fall in the pulse of ten or twenty beats in the minute. The thirst will diminish, the vomiting grow less, there may now be ability to retain fluids in the stomach, and the patient falls into a quiet, refreshing sleep, and is relieved from all the severer symptoms of the paroxysm. If, however, the thermometer is placed in the axilla, it will indicate fever, and although the temperature may show a marked decline, it is still above the *normal* standard. At no time is there a complete interruption; the fever is continuous. This is termed the period of remission. At the same hour on the following day all the active febrile symptoms return, the range of temperature is higher, the gastric disturbance is more marked, the countenance assumes an anxious expression, and all the symptoms are more severe.

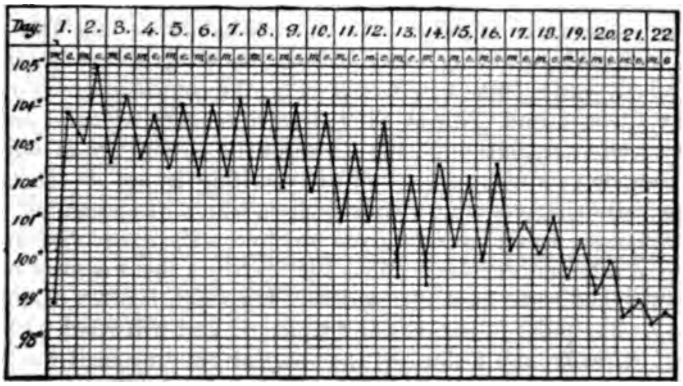

FIG. 176.
Temperature Record in a case of Remittent Fever ending in recovery.

This return of the severe febrile symptoms constitutes what is called the exacerbation, and the period between the time when the fever abates and the development of the exacerbations is called the period of remission. Remissions and exacerbations are the characteristic symptoms of a remittent fever when it is fully developed, at which time a morning remission is the rule, though the time of the first paroxysm varies. The morning remission is so invariable that it is regarded by many as a diagnostic sign. If the exacerbation begins at noon it will usually decline about midnight, and the remission will last until about noon the next day. In very severe cases there may be a double exacerbation, one at noon, the other at midnight, the remissions being in the evening and morning. The second exacerbation is similar to the primary in its attendant phenomena, except that it is more severe and of longer duration, ends in a less profuse perspiration, and the remission is not so well marked as the first. On the third day at about the same hour, or a little earlier, there is another exacerbation, which has a still longer duration, is of greater severity, and is followed by a more incomplete remission.

If the disease continues, the remission from day to day becomes less and less distinct. By the end of the first week the remission can no longer be detected, and the fever becomes continuous, without any marked daily variation in temperature or pulse. As the remissions become less and less distinct, with each returning exacerbation, the tongue becomes more and more parched, sordes collect upon the teeth, the countenance becomes dull and heavy, distress and pain in the epigastrium continue, and are accompanied by tenderness, although the senses of the patient are so dulled that he may scarcely complain of it; and the vomiting is not so constant, and is of a less distressing character; constipation, which was present at the commencement of the fever, has now given way to diarrhœa, the discharges usually being of a brownish color. With the diarrhœa there is some fulness of the abdomen, and some local tympanitis. Hiccough is often obstinate and distressing. The pulse is increased in frequency, and will reach 120 or 130; it is small, thready, and feeble—at the onset of the disease it was full and compressible. The patient slips down in bed, picks at the bed-clothes; there is subsultus and difficulty in deglutition, and the tongue is protruded with difficulty, as in the severer forms of typhoid fever. The *urine* is scanty, acid, dark colored, but *very* rarely is it albuminous. It may be bloody. The patient passes into a condition closely resembling that of one in the third week of typhoid fever, with the exception that there is no eruption.

The diarrhœa, abdominal disturbance, and tympanitis, and often the tenderness over the ileo-cæcal region, the typhoid tongue, and the low muttering delirium, closely ally this stage of remittent fever to typhoid fever. After these symptoms have continued a week or ten days, if the case is to terminate in recovery, remissions occur and become more and more distinct, until finally there is no exacerbation, and the patient passes into a state of convalescence. If, however, a fatal termination is to take place, the remissions will not recur, but the typhoid symptoms become more marked, and the patient finally dies from exhaustion or from complications. Of all the symptoms which attend remittent fever, nausea and vomiting are the most constant and the most distressing. I have seen patients, after the temperature has fallen to its normal standard, suffer for weeks from gastric disturbance, attended by more or less jaundice.

If, in the progress of a remittent fever, the exacerbation occurs a little earlier each day, then treatment is not controlling it; the fever is then said to be *anticipating*, and the disease is almost certainly passing from a distinct remittent to a continued remittent. If, on the other hand, the exacerbation occurs a little later each day, the fever is said to be *postponing*, and it is under control, the remissions become longer, the exacerbations become shorter and less severe, until the patient reaches complete convalescence. The thermometer will indicate to what extent the disease is being controlled.

Bilious Remittent Fever.—In a certain proportion of cases in all endemics of remittent fever, vomiting of "bilious" material, and *jaundice* are prominent symptoms, the skin often becoming so yellow that the patients present an appearance similar to that of those suffering from yellow fever; with this yellow discoloration of the skin there is an unusual tenderness on pressure over the hepatic region. Under such circumstances this fever has been named "*bilious remittent.*" By some of the older writers it has been described as an idiopathic fever, distinct from remittent or any other form of malarial fever. Medical literature, however, contains no facts in support of such a view. The pathology and symptomatology of the fever described by writers under the head of *bilious remittent fever* differ in no respect from those of simple remittent, except that the fever is accompanied by symptoms of more than usual hepatic and gastric disturbance. My own experience leads me to regard it as a form of simple remittent, accompanied by a more than usually severe gastro-hepatic catarrh, and that it is not entitled to a separate place in the nosology of fevers.

Infantile Remittent Fever.—It is a matter of every-day experience that children are subject to certain gastric and intestinal derangements, which are attended by more or less fever, which is very apt to assume a remittent type. Such fevers cannot, however, be regarded as specific diseases, for they are developed independent of any specific fever poison, and are only symptomatic of some local irritation. There is a form of mild typhoid fever which is often met with in children, especially in the autumn, which has also incorrectly received the name of infantile remittent fever. In this class of cases, the usual symptoms of typhoid fever are so modified by age that the fever assumes a remittent type. The presence of rose-colored spots, and the characteristic typhoid lesion of the intestines, will determine the true nature of these fevers. Simple malarial remittent in children does not differ from the remittent of adults. Remittent fever in children is more liable to be followed by malarial cachexia than in the adult.

Differential Diagnosis.—The rules by which a remittent is distinguished from an intermittent fever have already been given.

The differential diagnosis between remittent and *typhoid* fever is often attended with difficulty, if the patient is not seen until the second week of the disease, but if he is seen at the very onset of the fever, it is hardly possible to confound these two forms of fever. The sudden advent of a remittent is in marked contrast to the slow development of a typhoid fever. Besides, they widely differ in the range of temperature during the first week of their development. In remittent there is a distinct remission, and there can be no doubt as to the type of fever after the first, certainly not after the second remission has occurred. Gastric symptoms are much more severe in remittent than in typhoid. By these symptoms alone a differential diagnosis can be made. If, however, the fever has been protracted to the third week, and the remissions are slight or altogether absent, although many of the symptoms of typhoid fever are present, the absence of the rose-colored spots, taken in connection with the previous history of the patient, is sufficient to establish the diagnosis.

Remittent fever may be distinguished from *yellow fever* by its high range of temperature, by its daily exacerbation and remission, by the presence of pigment in the blood, and by the absence of albumen in the urine, which is present in yellow fever. In remittent fever, hemorrhage from the mucous surfaces, especially from the mucous membrane of the stomach, indeed from any source, is of rare occurrence, while in yellow fever it frequently occurs from mouth, nose, eyes, ears, bowels, and urinary passages. Death often occurs on the third day in yellow fever, but in the severest cases of remittent fever not before the seventh day. Yellow fever is portable and contagious; remittent is neither. Remittent fever may be confounded with pyæmia and septicæmia, but their differential diagnosis has already been sufficiently considered.

Prognosis.—The prognosis in simple remittent fever is good. Even cases of the severe types of this fever should terminate in recovery, if skilfully managed, especially if they are seen in the early stages. Its type varies very much according to locality. The remittent fever in New York City is of a mild type. In that form which prevails in our Western and Southern States a fatal termination is of frequent occurrence. There is a type which soon loses its remission, and becomes a pernicious malarial fever, the prognosis of which is unfavorable. The complications which may render the prognosis unfavorable are meningitis, pneumonia, gastritis, enteritis, diarrhœa, dysentery, and splenitis. The prognosis will also be modified by the condition of the patient at the time of the attack, and by the character of the endemic which is prevailing.

The symptoms which indicate that recovery is to take place are the fact that the exacerbation is delayed or rendered less severe, the early subsidence of gastric symptoms and headache, and a decrease in the frequency of the pulse, and the appearance of vesicles about the lips. Distinct remissions, accompanied by moderately free perspiration, indicate an approaching favorable change. On the other hand, if the fever is more continuous than paroxysmal, with a pulse becoming daily more feeble and more frequent, if there is a tendency to collapse at the close of the exacerbations, and suppression of urine, with signs of extreme exhaustion, danger is indicated. The average duration of this fever is two weeks.

As this fever varies so greatly in severity at different times and in different localities, it is impossible to accurately determine its average rate of mortality.

Treatment.—In this disease, we have means at our command by which, in the majority of cases, it can be controlled, and by which, in most instances, its duration may be much shortened. It is hardly necessary to refer to such remedial agents as blood-letting, emetics, cathartics and diaphoretics, which have all been employed in the treatment of this fever, for they have all been supplanted by a single remedy. Experience has proved that the poison which causes the fever cannot be removed from the system by any of the so-called eliminative methods of treatment. If this class of patients are depleted to any extent, the development of those typhoid symptoms which are especially to be avoided will be hastened. Those living in malarial districts are never up to the normal standard of vigor, and, consequently, are in a condition to be affected unfavorably by any plan of treatment or by any remedial agents which shall enfeeble the vital powers.

The first thing to be done in the successful management of this fever is to place the patient under the best possible hygienic surroundings. The same care should be exercised in the arrangment of the sick-room as has already been proposed in the management of typhoid fever. Those who have seen most of remittent fever in its severer form recommend that the treatment of each case be commenced by administering a mercurial purge. They claim that there is always more or less engorgement of the liver, spleen, and mucous membrane of the stomach and intestines, and that, so long as these organs remain in this condition, no plan of treatment will be successful. However great may be the differences of opinion in regard to this, all agree that the sulphate of quinine should be used in its treatment. Practitioners differ as to the mode of its administration, but all advocate its use. Some maintain that it has greater power over the disease when administered in small doses, repeated at short intervals; others, that it should be given in one or two large doses during the remission, an hour or two before the commencement of the expected exacerbations. Others, again, claim that the quinine has its greatest power over the fever when administered during the activity of the febrile excitement.[1]

From these reports, and from my own experience, I do not hesitate to administer quinine at any time during the period of the exacerbation or remission. My rule is to give ten or twenty grains at a dose, according to the severity of the fever, and repeat it every two hours until cinchonism is produced. When cinchonism is reached, although the fever may not be controlled, it is well to stop its administration until twenty-four hours have elapsed; by doing this one can better determine the antiperiodic power of the drug. If the exacerbations do not disappear, but are delayed and are less severe, the fever is being controlled. If, notwithstanding this free use of quinine, the exacerbations are more severe and longer in duration, and the remissions less frequent, and typhoid symptoms are manifesting themselves, stimulants may be demanded. Even large doses of stimulants may be required to sustain the patient while he is passing through this period of the disease.[2]

Remittent fever is not, like typhoid fever, a disease of days or weeks.

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[1] This subject was carefully studied by those engaged in the English Medical Service in India. Under the direction of the Surgeon-General in that department quinine was administered at different periods in the course of the fever, one surgeon giving quinine at the commencement of the exacerbation, another immediately after the exacerbation had passed its height and as the sweating stage was coming on, another immediately preceding the exacerbation, and still others giving it during the remission. This plan was adopted to determine with positiveness when the smallest amount of quinine would have the greatest controlling effect over the fever. From the various branches of the department reports were made, whence the conclusion was arrived at that quinine, administered during the time of the exacerbation, had not only a greater influence in diminishing the severity of the disease, but it also more completely controlled the fever, and more markedly shortened its duration than when it was administered during the remission.

[2] Livingstone and other African travellers advise bitter ale as about the best stimulant, and the one best borne by the irritable stomach in this fever.

In its severer forms no time should be lost while waiting for the action of cathartics or other remedial agents which are supposed to be of importance, but the administration of quinine should be at once commenced. When the disease has reached its second or third week, and there is no evidence that the patient is passing on toward recovery, administer a second time large doses of quinine; in this way the progress of the fever may be arrested. If, after a second cinchonism is produced, the fever is not arrested, omit again for a few days the administration of quinine; then repeat the large doses a third time. It is much better to proceed in this way than to keep the patient in a continued state of cinchonism. It is not necessary to enumerate the long list of drugs which at different times have been proposed as specifics in this fever, all of which, by common consent, are now regarded as far less reliable than quinine. The important thing is to know how and when to administer quinine.

There are certain palliative measures which it is sometimes important to employ. If the exacerbations are very intense, the headache very severe, and the restlessness or other febrile symptoms are not relieved by full doses of quinine, cold may be employed for its antipyretic effect, as in typhoid fever. Full doses of the bromide of potassium promote sleep. Frequently, in mild cases, sponging the surface with tepid water is not only grateful to the patient, but it has a controlling influence over the fever. If vomiting is constant, severe, and exhausting, hypodermics of morphine will be found of service. Some advise Fowler's solution to check the distressing vomiting. The treatment of this fever is expectant, save in the use of quinine.

CONTINUED MALARIAL FEVER.

I have included this fever in the list of the malarial fevers, although it is not altogether malarial in its origin; malarial poison, however, is essential to its development. As it has many elements in common with typhoid, and many which ally it to remittent fever, it has been called "typho-malarial."[1] During the late civil war it was called camp and Chickahominy fever.

In its etiological aspect it partakes more of the character of typhus than of typhoid. The name typho-malarial fever has been employed by one class of observers to indicate the presence of malaria, and the specific poison of typhoid fever. By another class the term has been employed to indicate the presence of malaria and a *septic* poison. Many doubt the existence of such a form of fever, and regard the so-called typhoid element as nothing more than a *typhoid condition*, liable to be developed in connection with remittent fever, as well as with many other diseases. The term typho-malarial is a convenient one for the first class of observers, and is one which can be employed by them without confusion; whereas to the second class of observers it is exceedingly objectionable, and gives rise to confusion.[1]

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[1] Wood (Prac. of Med.) calls it entero-miasmatic, and Drake (Dis. of Mississippi Valley) gives it the name Remitto-Typhus.

This fever is produced by the combined action of a *septic* and a *malarial* poison. In some the septic element predominates, and in others the malarial. The preponderance of the one or the other will determine with a good degree of certainty the course, prognosis, and treatment of each individual case. The distinguishing lines, however, between these two elements are not always sharply drawn ; both may be modified in their manner of development and in their morbid anatomy, by the development of intercurrent complications, such as scurvy, pneumonia, etc.

Morbid Anatomy.—The changes which take place in the constituents of the blood are a decrease in the albumen and fibrin-factors, and an increase in white blood-globules. In connection with these blood changes there are more or less extensive parenchymatous changes in the internal organs similar to those met with in other forms of acute infectious diseases.

The *liver* is increased in size, and its cut surface presents an appearance which closely resembles that known as nutmeg liver. Sometimes it presents the peculiar bronzed color of the liver in remittent fever ; at other times it very closely resembles the liver of yellow fever. A microscopical examination shows free fat and pigment granules, as well as lymphoid, fusiform and stellate cells—which are perhaps derived in great measure from the spleen ; no pigment is found in the hepatic cells, but they are stained with bile, as is also the inter-lobular tissue.

In most cases the *spleen* is enlarged, softened, and of an almost black color. The Malpighian bodies are prominent, and present the appearance on the torn surface of the spleen of little tumors, varying in size from a pin's head to that of a pea. The organ is rarely as much enlarged or softened as in typhoid or remittent fevers. It is always the seat of more or less pigmentation. The pigment is in the lymphoid cells of the spleen chiefly, but it also accumulates about the veins.

No uniform change will be noticed in the *kidneys*, except hyperæmia, which will be most marked in their cortical substance.

The *lungs* are the seat of more or less extensive hypostatic congestion. Splenization of the lungs is not frequent.

The *heart* is pale and flabby. Its muscular fibres are the seat of granular or vitreous degeneration similar to that which takes place in the heart in typhoid fever. Exsanguinated clots more or less firm may be found in its cavities, but they have nothing peculiar about them. They closely resemble those found in persons who have died from failure of heart power. They are rarely, if ever, the direct cause of death.

The intestinal changes, when present, resemble those of typhoid fever.

While, therefore, no pathological lesions which can be regarded as characteristic of this type of fever are found, and while the lesions very closely resemble those of typhoid fever on the one hand, and remittent fever on the other, still its clinical differences are sufficient to distinguish it from both, and to stamp it as a distinct type of fever.

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[1] In 1847 Wood stated that "remittent or bilious fever, as it was then popularly called, was sometimes of a low adynamic character, from co-operation of a typhoid epidemic influence with miasmata." Forty years ago the term *gastric fever* was given to that variety of marsh fever where the stomach was deranged and irritable from the onset.

Etiology.—It is difficult to determine the true etiology of this fever. That malarial poison is necessary for its development there can be no question. It is equally certain that some other poison besides malaria is in operation whenever it prevails. Its infection does not follow the laws of development governing the specific cause of typhoid fever, for it is in no way connected with the excrements of one suffering from the fever. There are a few facts connected with its development which are now well established:

First. It is met with only in malarial districts.

Second. In the majority of instances, when this fever has prevailed, its development has been preceded or attended by marked and easily recognized anti-hygienic conditions, such as overcrowding, bad sewerage, and other conditions favorable to the development of septic poison.

Third. That it is a *non-contagious disease*, and is never propagated from the affected to the healthy, either directly by personal contagion, or indirectly by morbid excretions.

Fourth. In its morbid anatomy and symptomatology it is a combination of malarial and typhoid fever. The special symptoms and lesions of one or the other of these fevers stamps its character. In large cities in which malarial diseases are prevalent, *sewer gases seem to furnish the element which is so essential for its development.* The history of disease in New York city during the past few years furnishes striking examples of the combination of these two poisons in developing a type of fever which must be classed under the head of continued fever.

Symptoms.—It is difficult to present a typical picture of this fever. To give even an outline of its symptoms which shall be approximately true of all, or even the majority of cases, is impossible. Its clinical history varies as the malarial or septic element predominates. Besides, there is a large number of cases in which neither of these elements predominates, for the patient almost insensibly passes from a malarial into a typhoid condition. There are also certain anti-hygienic conditions which may be present, which give to the fever an unusual and peculiar type. For example, when those conditions exist which favor the development of scurvy, as the patient enters upon the second week of the fever, the scorbutic phenomena will become prominent. At times the dysenteric element may be engrafted on this fever, and greatly modify its course, and lead to a train of symptoms and morbid changes which closely ally it to epidemic dysentery. The course of this fever may also be greatly modified by certain local complications which are especially liable to occur during the second or third week. The presence of any of these conditions will greatly change its clinical history, but the phenomena which attend its early development will always be sufficient to determine its true character.

In considering the symptoms in detail, that class of cases in which the *malarial element is predominant* will first be described. This type of fever is usually ushered in by a distinct chill.[1] In some instances no premonitory symptoms are present; in others the chill is preceded by wandering pains in the limbs and back, headache, loss of appetite, and a feeling of great exhaustion. In a large proportion of cases in the early stage, the countenance has a peculiar waxy, clay-colored or yellowish tinge. The chill varies in duration from half an hour to an hour, and in character closely resembles the chill of simple remittent fever. It is immediately followed by active febrile symptoms, the temperature rising in a few hours to 103° or 104° F. The pulse reaches 100, and is full and forcible. The excretions are all checked, and there is mental disturbance and sometimes delirium.

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[1] In Gibb's account of a malignant epidemic in Nicaragua, Central America, none of the cases began with a chill.

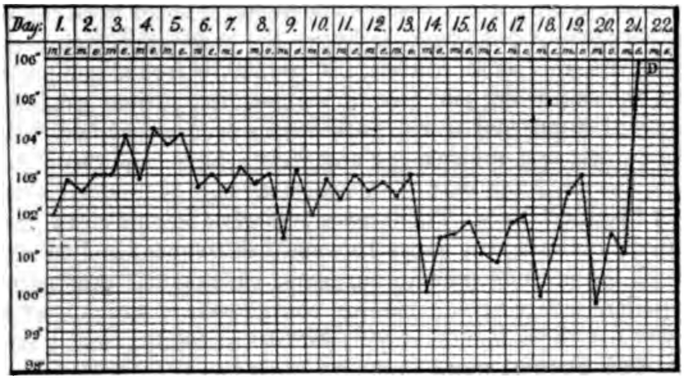

FIG. 177.

Temperature Record in a case of Continued Malarial Fever, Remittent type.

When once established, the fever pursues a variable course. At its onset, and for the first few days, its phenomena often closely resemble those of simple remittent fever, though the remissions are never so well defined, and there is at the very onset of the fever an amount of intestinal disturbance which is rarely present in simple remittent. The existence of abdominal tenderness, especially in the right iliac fossa, is a strong point in its diagnosis. As the temperature rises, nausea, vomiting, and epigastric tenderness are present in a greater or less degree. These gastric symptoms bear a close resemblance to those which attend the development of remittent fever, while the intestinal and abdominal symptoms are similar to those of typhoid. Diarrhœa may precede the chill; in most cases it is present during some portion of the fever. At first the tongue presents a pale, flabby appearance, with a smooth surface; soon it becomes covered with a white or yellowish-white coating; later it becomes red, and the coating becomes brownish; in severe cases it may suddenly become clean, red and shining, and sordes may collect upon the teeth and lips.

In those cases in which a scorbutic element exists, the tongue is enlarged, pale, and flabby, its surface smooth and covered with a white fur, which is thickest on its edges, the gums are swollen and present the characteristic appearance of scurvy; the skin is covered with petechiæ and irregular discolorations, and mental and bodily prostration is early marked.

In those cases in which a dysenteric element is present, as the fever develops, the dysenteric symptoms become prominent, the discharges from the bowels are blood-stained and watery. The tongue soon becomes dry and brown, and the patient shows signs of extreme exhaustion, with a few of the gastric symptoms which are usually so well marked in the early period of the fever. Throughout the whole course of the disease there is a marked tendency to periodicity, the exacerbations usually assuming a tertian type.[1] In fatal cases, as the patient reaches the second or third week the symptoms are very like those of fatal typhoid fever; the prostration becomes more and more complete, the pulse reaches 130 or 140, is feeble, compressible and irregular, the skin is hot and cold in patches, the patient gradually passes into a state of stupor and coma, involuntary evacuations take place, there is subsultus, deafness, a blackened and rigid tongue, and death ensues. In cases that recover, symptoms of amendment may be noticed between the tenth and twentieth days. The tongue begins to become clean, the abdominal symptoms subside, the pulse becomes less frequent and fuller, the disturbance of the nervous system disappears, the appetite gradually returns, and the patient enters upon a tedious convalescence, which is attended by more or less diarrhœa, mental stupor, cardiac irritability, and a slow return of mental and physical vigor.

The train of symptoms thus briefly sketched may be greatly modified by a variety of complications. Not infrequently pulmonary complications develop during its second week, and so change its phenomena that the fever element may be overlooked and the pulmonary element alone engages the attention of the physician. Suppurative inflammation of the cervical and inguinal glands sometimes complicates it, and leads one to the mistake of regarding it as a purely suppurative fever. Enlargement and suppuration of one or both parotids are not uncommon events. Again, scurvy under certain anti-hygienic conditions may so modify its phenomena as to lead one to regard it as an entirely new type of fever. The scorbutic element in this class of cases is developed in connection with the malarial exposure.

The prominent symptoms present in the *septic type* of this fever, such as lassitude, headache, pains in the back and limbs, resemble those of typical typhoid fever; either a distinct chill or a complete intermittent or remittent paroxysm ushers in the febrile symptoms. The rise in temperature following the ushering-in chill has no typical range; in some cases the rise is gradual, not reaching its maximum before the middle of the second week; in other cases the rise is sudden, reaching 104° or 105° F. within twenty-four hours after the occurrence of the chill. In typhoid fever during the first week, there are indistinct forenoon remissions and afternoon exacerbations, but in this fever the remissions are well-marked, especially on every second or third day, causing the fever to assume a more or less distinct tertian or quartan type. *One of its earliest symptoms is*

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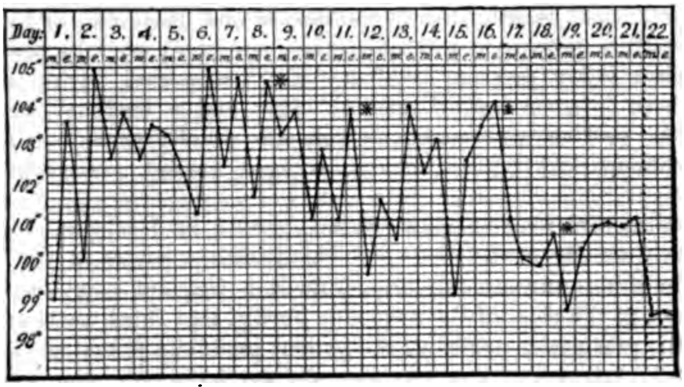
[1] As the disease progresses the exacerbations become shorter; the remissions longer, and at times a normal or even a subnormal temperature may be reached, temporarily, as the adynamic condition becomes pronounced.

well-marked hepatic tenderness; with the hepatic tenderness there is enlargement of the spleen, which, as the fever progresses, reaches a much larger size than is ordinarily met with in typhoid fever. During the first week the pulse is full and rarely more than 100, but during the second and third weeks it is small and compressible, and in severe cases ranges from 110 to 130 per minute.



* Sponge Bath.

FIG. 178.

Temperature Record in a case of Continued Malarial Fever. Septic variety.

The appearance of the tongue varies with the period of the fever. At first it is swollen, with red projecting papillæ, and has a light white coating. As the typhoid condition becomes more marked its appearance changes; it becomes dry and brown, and frequently the brown coating cracks and fissures are formed in the mucous membrane underneath. Should the tongue become moist and begin to clean, it is an indication that convalescence is being established. The coating is removed in two ways, either gradually from the edges to the centre, or it is thrown off in flakes. In the latter case, after the removal of the coating, the tongue assumes a beefy red appearance, and after a short time may again become brown and dry. Under such circumstances there will be a renewal of the fever-symptoms. After the fever has continued a few days the surface becomes dry and harsh, and the skin assumes a dingy hue, which is quite characteristic; sometimes there is a well-marked jaundice. The urine gradually diminishes in quantity and deepens in color until convalescence commences, when it increases in quantity. It is rarely albuminous.

Diarrhœa may occur at any period. It is not usually excessive until the second or third week. There is nothing characteristic about the discharges. They are usually of an exceedingly fetid odor, watery and dark-colored; in the later stages of the disease they sometimes contain blood. In some instances the character of the stools is termed bilious and an excessive hepatic secretion is then indicated; at other times they are of dark clay color, showing a deficiency of the biliary secretion. With the diarrhœa there is usually more or less abdominal tenderness, especially in the right iliac region ; but tympanitis is rarely well-marked. In many cases there is retraction of the abdomen.

As already stated, headache is very constant in the early period of the fever. It often precedes the ushering-in chill. As the fever progresses it gives place to a delirium, which is never violent, but which is muttering in character, and is attended by restlessness and insomnia, or by drowsiness, subsultus, picking at the bed-clothes, and great nervous prostration. If delirium is not present, or after it has disappeared during convalescence, there is great lack of mental vigor and a tendency to apathy. The other nervous phenomena, which are usually present in any condition when marked typhoid symptoms exist, are not prominent in this fever.

The subsequent phenomena which may attend its development will vary with the intensity of the fever and the resisting power of the patient. Epistaxis is not uncommon ; bronchitis is a frequent complication. In fatal cases, at the close of the second or during the third week, symptoms of extreme prostration come on, the patient gradually passes into a state of stupor, which lapses into one of coma, and death ensues. In cases that are to recover, usually by the end of the second week the tongue begins to clean, the gastric and intestinal symptoms, with the exception of the diarrhœa, begin to subside, the pulse becomes slower, the nervous disturbances disappear, the appetite returns, and the patient enters on a convalescence which is usually protracted. Its phenomena may be modified by certain anti-hygienic surroundings, to which those suffering with this fever may have been subjected prior to, and during, its development. Thus, when it prevails among those who have suffered privations, been badly fed, badly clothed, overcrowded in badly ventilated apartments, or surrounded by decomposing animal and vegetable substances, although the fever is attended by the same general phenomena, there are certain variations which ally it to relapsing fever. Prominent among these are neuralgic and arthritic pains in various parts of the body, especially in the back and limbs ; hemorrhagic tendencies marked by bleedings from the gums and mucous surfaces ; and not infrequently large ecchymoses occur in various parts of the body.

In this class of cases the fever is of a low type from the commencement, with quotidian exacerbations and remissions. Diarrhœa usually precedes the development of the febrile symptoms. Frequently during the second week a muttering delirium comes on, accompanied by drowsiness and a tendency to stupor. Despondency, indisposition to make any exertion, and a state of utter indifference as to the future are frequently met with during the entire period of the fever ; in fact, mental and bodily prostration is more marked here than in any other fever. In fatal cases death may be the result of hemorrhage from the mucous surfaces, or from exhaustion. In cases that recover, convalescence comes on late, and is slow and tedious. Diarrhœa frequently follows the subsidence of the fever, and leads to a fatal result.

Differential Diagnosis.—The affections with which continued malarial fever is likely to be confounded, are *typhoid, remittent, relapsing, typhus,* and *yellow fever.*

The advent of continued malarial fever is usually marked by a distinct chill, while typhoid comes on insidiously and is attended not by a distinct chill, but by a chilly sensation. The rise of temperature in continued fever is sudden and follows no typical range, while in typhoid the typical range of temperature during the first week is diagnostic of the fever. In typhoid fever, on the sixth or eighth day, rose-colored spots appear; in the other, although an eruption may be present, it has none of the characteristics of the typhoid eruption, is not rose-colored, does *not* disappear on pressure, and remains visible throughout the whole course of the fever. Besides the absence of these characteristic symptoms of typhoid fever, there is, in continued malarial fever, a distinct periodicity in the febrile action, a certain icteroid hue of the skin, hepatic tenderness, and great gastric disturbance, conjoined with which the appearance of the tongue, the character of the diarrhœa, and the non-infectious character of the stools will serve as important aids in the differential diagnosis of these two forms of fever. If, upon microscopical examination of the blood, free pigment is found, the diagnosis of continued malarial fever is established.

The malarial type resembles *remittent fever* in its ushering-in symptoms. In both cases there is a chill followed by fever, attended by one or more distinct exacerbations and remissions. The early appearance of the enteric symptoms, attended by other well-marked typhoid phenomena by the end of the second week, establishes the diagnosis, and as the fever progresses the typhoid condition becomes more and more apparent. Besides, remittent fever yields more promptly to quinine.

Severe cases which are complicated by scorbutic tendencies marked by petechiæ and great prostration of the vital powers may be confounded with *typhus fever;* yet the severity of the attack, the higher range of temperature, the greater frequency of the pulse, the dusky countenance, the absence of diarrhœa and all other abdominal symptoms in typhus fever, render it easy to make the differential diagnosis between the two types of fever. Besides, typhus fever has a characteristic eruption, is only propagated by contagion, and, if it prevails, does so epidemically.

Occasionally *yellow fever* has been confounded with continued malarial fever. The range of temperature is lower in *yellow* fever, and on the third or fourth day it falls suddenly, and there is a more or less complete remission. The circumorbital pain, the appearance of the eye, the pulse rarely ranging over 110, the peculiar color of the skin, the character of the matter vomited, the absence of diarrhœa, and the shorter duration of the disease, will enable one to make the diagnosis of yellow fever. The urine is rarely albuminous in continued fever; nearly always so in yellow. Again, yellow fever usually prevails epidemically, and is confined to certain localities and certain seasons of the year. It is a portable disease, and the yellow fever poison may be conveyed from an infected to a non-infected district by means of clothing or merchandise, while the poison of continued fever is of endemic origin, and cannot be carried beyond the infected district.[1]

Prognosis.—The ratio of mortality in continued malarial fever varies greatly in the different regions in which it occurs, and as the malarial or septic element predominates. The hygienic surroundings of the patient and the range of atmospheric temperature will also very greatly influence the prognosis. Statistics of this fever in different localities and in different years give the ratio of mortality from eight to ten per cent. The septic type is more fatal than the malarial. Great caution should be exercised in prognosticating the result of any case, for the mildest cases sometimes suddenly assume a severe type and terminate fatally, while very severe and apparently hopeless cases unexpectedly improve, and recovery takes place.

The average duration of those cases which terminate in recovery is from three to four weeks. The period of convalescence is prolonged; three or four weeks often elapse before the patient is completely restored to health. A fatal relapse may occur at any period during convalescence.

Its most frequent complication is inflammation of the respiratory organs, the development of which is marked by those symptoms which usually attend the development of the different acute pulmonary affections. In the majority of instances the signs of bronchitis are not present until the fever is well established. The bronchitis resists treatment, and does not disappear until convalescence is fully established. When pneumonia occurs it is catarrhal in character, and few of the strongly marked rational symptoms of ordinary pneumonia are present. The physical signs, however, will always enable one to determine the presence of pulmonary complications, and any great irregularity in temperature during the course of the fever should be an indication for a careful physical examination of the chest.

It is sometimes difficult to distinguish between the cerebral symptoms of this fever and those symptoms which attend meningeal complications, but the meningeal complications are of so very rare occurrence that it is safe to assume they are not present until some of the diagnostic symptoms of meningitis occur. Serious abdominal complications, such as intestinal perforation, peritonitis, and hemorrhage are rare, but when they do occur their advent is marked by such urgent symptoms that one loses sight of the ordinary symptoms of the fever. It is hardly necessary to refer to those modifications in the clinical history of this fever which follow the development of abscesses, bed-sores, gangrene, etc. The occurrence of any of these complications will very materially influence the prognosis in any given case. Capillary bronchitis and pneumonia are especially dangerous when they develop during the third week of the fever. Anti-hygienic surroundings, such as overcrowding and improper food, materially affect the prognosis. If continued malarial fever prevail among those who are crowded into badly-ventilated apartments, who from filth and improper nutrition have septic and scorbutic tendencies, the ratio of mortality is much greater than among those who are free from such complicating influences.

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[1] The points of differential diagnosis between this disease and relapsing fever are considered under the head of relapsing fever.

The symptoms which may be regarded as indicating an unfavorable termination are a continued high temperature, showing little or no tendency to remission; a very frequent, feeble, fluttering pulse; continued hiccough; profuse diarrhœa, the discharges at times being involuntary and containing mucus, pus, and blood; a dry, red, cracked, and fissured tongue; great drowsiness, with a tendency to stupor and coma; and the appearance of petechial spots on the surface of the body, attended by frequent hemorrhages from the lips, gums, and tongue. In a severe case, the occurrence of any of these phenomena renders the prognosis more unfavorable.

The character of the prevailing fever will also greatly influence the prognosis in any given case. If the type of the prevailing fever is mild, or if comparatively few deaths have occurred, though the symptoms in a given case may appear unfavorable, yet recovery is probable. If, on the other hand, the type is severe, and many deaths have occurred, cases apparently mild will suddenly become severe, and the prognosis becomes unfavorable. As already stated, the hygienic surroundings and the previous habits of the patient very greatly influence the prognosis. With drunkards, and those enervated by vicious habits, a mild type of this fever will probably prove fatal.

Treatment.—No plan can be presented which will be applicable to all cases.

The first question which meets us is: Cannot the development of this fever be prevented? It has been stated that its development was principally due to three causes—namely, malarial poison, overcrowding, and improper diet. In a large proportion of instances it is possible to do away with the last two causes. The overcrowding and the faulty diet may be prevented, and thus the septic poison which gives to this fever its "typhoid" type may be destroyed, or its development prevented. The strict observance of hygienic laws in the localities where this fever prevails has, in some instances, entirely changed the type of the disease. Even after the fever symptoms have been well developed, the removal of patients from anti-hygienic surroundings has frequently been attended by the most satisfactory results. When isolated cases of this fever are met with in localities apparently free from such sources of infection, a careful search should be instituted in order to find the source of the infection. Defective sewerage and faulty drainage have been found to be fruitful sources of infection.

There are no specifics. In those cases in which the malarial symptoms predominate, the administration of quinine will in many instances arrest its progress or shorten its duration; but in those cases in which the septic element predominates, while quinine may act as an antipyretic, it has little power to arrest its progress or to shorten its duration, but it will, in many instances, render the course of the fever milder. Warburg's tincture in many cases will have a controlling power over the fever when quinine fails.

It has been claimed by some that arsenic has a specific influence over the fever, and that it exercises a peculiar and most beneficial effect upon the intestinal lesions. There is little doubt but that arsenic, like quinine, acts beneficially in many cases of the malarial type of this fever; but unquestionably this beneficial effect is due to its acknowledged power over malarial affections, and not to any specific influence which it has over the fever. As an antiperiodic it is inferior to quinine. Eucalyptus does not act as beneficially in continued malarial fever as in the simpler forms of malarial fever.

It is of importance to remember that this class of patients do not bear well the prolonged application of cold to the surface, either by means of the cold bath or the cold pack. The danger resulting from the injudicious use of cold baths is greater in this than in any other infectious disease.

The rules for the administration of stimulants are the same as those given for their administration in typhoid fever. The effects of the first few doses should be carefully watched. They should never be given indiscriminately, for there is greater danger of over-stimulating in this than in any other fever. Their use is indicated whenever signs of heart-failure are present, such as a feeble pulse and an indistinct first sound of the heart. The use of stimulants is not necessarily contra-indicated when delirium is present. Frequently after their administration the delirium will pass away, and only when it is decidedly increased by their use should they be abandoned.

The state of the bowels, skin and kidneys demands the closest attention. If, early in the disease, the bowels are constipated, a calomel purge combined with ten or fifteen grains of quinine will often be followed by marked benefit. In any stage of the disease brisk purgation should be avoided. If diarrhœa is present, it should not be interfered with unless it becomes exhausting; then it should be checked by small doses of opium combined with astringents. Symptoms referable to disturbance of the nervous system sometime require special treatment. If there is extreme restlessness, muscular twitchings, or active delirium, opium may be administered in full doses. The effect of the first dose must be carefully watched. If sleep soon follows its administration, and the delirium gradually subsides without any aggravation of the other symptoms, its use may be continued; if, instead of producing sleep, the patient becomes more wakeful, and the delirium is increased and more active, and the other symptoms are greatly aggravated, its use must be immediately abandoned. Under these circumstances chloral may be tried with great care.[1] Quain advises gr. xv.-xx. of bromide of potassium under similar conditions.

Some claim that spirits of turpentine in the treatment of this form of fever has almost a specific power, while others regard it useful only as a stimulant. My own experience leads me to employ it only as a stimulant during the second and third week of the disease, when there is great prostration and marked typhoid symptoms. It may be given as an emulsion in doses of twenty drops every two hours. The *diet* best suited to patients with this fever is milk administered in the same way as was proposed in the case of typhoid fever patients. Special complications occurring during the non-septic variety must be met with such remedies as the condition of the patient and the peculiar complications may require.

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[1] Wood recommends Hoffman's anodyne and spts. æth. nitrosi for restlessness; and musk, asafœtida camphor, and similar drugs for the hiccough.

PERNICIOUS MALARIAL FEVER.

This form of fever has received other names, at different times and in different localities. It has been called *congestive fever, ardent fever, tropical typhoid fever,* and *pernicious fever.* The latter name seems most appropriate, and at the present time is generally adopted.

It is true that in the majority of cases there is more or less congestion of the internal organs, and sometimes the patient is overwhelmed by these congestions, but in a large number of cases no such congestions exist, and under such circumstances the designation pernicious is to be preferred. In its severe and dangerous form it may be remittent or intermittent in character, and may assume any of the types of periodical fever, but the quotidian and tertian types are the most common. Sometimes its pernicious character is clearly marked at the onset of the fever, during the first paroxysm; at other times it comes on insidiously, and its pernicious character is not suspected until after the occurrence of two or three paroxysms.

There are several well-marked and distinct varieties of pernicious fever—the most common and most important of which are the *comatose,* the *delirious,* the *algid,* and the *gastro-enteric.* It is the locality in which pernicious fever prevails that gives the fever its distinctive peculiarity. Pernicious fever not infrequently appears as an epidemic; sporadic cases are met with in those regions where simple intermittent and remittent fevers prevail.

Morbid Anatomy.—Its anatomical lesions are similar in kind to those of intermittent and remittent fevers, but they differ very much in degree. For instance, the pigmentation is more abundant. The abundance of the pigment, and the extent of the pigmentation will vary with the severity of the fever. The other changes in the different organs and tissues of the body are very similar in character to those described in connection with intermittent and remittent fever. The post-mortem appearances in pernicious fever vary with the intensity of the malarial infection and the peculiar atmospheric conditions under which the fever is developed. In some instances there will be evidences of intense engorgement of the blood-vessels of the brain, and the entire brain substance will be more or less thoroughly pigmented. In others, minute blood-extravasations will be found scattered here and there throughout the substance of organs. Small blood-extravasations into the spinal cord, accompanied by more or less pigmentation, are very apt during life to be attended by tetanic spasms. In persons dying of pernicious fever after the third attack, I have found all the organs of the body pigmented. Sometimes there is intense engorgement of the liver, that is, the most marked post-mortem changes will be found in that organ, and the amount of pigmentation present will correspond with the intensity of the congestion. With intense engorgement of the organ there are usually blood-extravasations.

Occasionally, infarctions occur in the spleen, and around each there will be a mass of pulpy material. The spleen is more invariably found softened than in any other fever. In connection with this softening, which is very extensive and similar to that found in typhoid fever, the organ rapidly becoming a soft, pulpy, bloody mass, it is also enlarged even beyond what it is in typhoid fever and is darker in color than normal. It is unnecessary to describe in detail the enlargement of the capillary vessels which occurs as a necessary result of this intense engorgement. Sometimes the kidneys and the lungs are the seat of intense hyperæmia, as the result of which the functions of these organs are more or less extensively interfered with. Hemorrhagic infarctions in the lungs are not infrequent. A low form of pneumonia is sometimes present. The heart is pale and flabby.

Etiology.—The exciting and predisposing causes of pernicious fever differ from those of the simpler forms of malarial fever only in degree, not in kind, but a higher range of temperature is requisite for the development of pernicious fever. It prevails only in those localities where the average range of temperature, for a time, reaches 65° F.

Symptoms.—Pernicious fever may commence abruptly; generally the premonitory symptoms which mark its development do not differ from those which mark the development of intermittent and remittent fever. In most varieties the attack commences with a chill, which is usually severe and prolonged. The attack may commence with distinct intermittent paroxysms of the quotidian type, but rarely more than two of these intermittent paroxysms will occur before it assumes the pernicious type; or a remittent fever with a distinct exacerbation and remission may go on for four or five days before its pernicious character will be developed. The milder form either gradually passes from a simple intermittent into a pernicious fever by a progressive increase in the severity of the paroxysm, or a single paroxysm of not unusual severity is suddenly followed by a pernicious one; a fatal result rarely occurs until the third paroxysm is passed. Again, a distinct chill may be followed by a condition that will at once be recognized as one of the varieties of pernicious fever. The ushering-in symptoms will always vary with the type of disease which is about to be developed. I shall not describe the phenomena that attend all these different varieties, but only those most commonly met with.

As the varieties in type of this fever are as numerous as the localities in which they occur, and as the type in any locality may change with every succeeding year—that is, the type of one year may be very unlike that of the preceding or following year—it is very difficult even to classify its different forms. The slight variations which are met with in the pathological lesions of the different varieties are still more difficult of description and classification. For instance, there is one variety which is characterized by a tendency to coma, called the *comatose variety;* another is characterized by a tendency to a peculiar form of delirium, termed the *delirious variety;* still another is characterized by a marble-like coldness of the surface, called the *algid variety;* again, we have one which is characterized by vomiting and purging, or choleraic symptoms, termed the *gastro-enteric variety;* then one in which there is acute jaundice, termed the *icteric variety;* then one in which there are profuse hemorrhages, termed the *hemorrhagic variety;* and still another in which there is profuse diaphoresis, termed the *colliquative variety.*

Comatose Variety.—A patient has a distinct paroxysm of one of the simpler forms of malarial fever (intermittent or remittent), with no special phenomena attending it, except that he has had a more than usually severe headache; with this there has been perhaps vertigo, stammering and indistinctness in the speech, an inability to talk with freedom, and a more than usual tremulousness during the hot stage. From this condition he passes as usual into the hot stage of an intermittent, or rapidly into an exacerbation of remittent, then into a state of stupor and unconsciousness, and finally lies upon his back, with a flushed face, congested conjunctivæ, dilated pupils, slow, deep, stertorous respiration, and perhaps a very slow pulse, or, if slow at first, it may soon become frequent. The axillary temperatures range from 105° to 107° F. The patient is now partially unconscious; he is apparently paralyzed; the urine is retained in the bladder, and the bowels move involuntarily. If the pulse is slow, it is full and hard. The respiration becomes more and more stertorous, and unconsciousness more and more complete. Usually a moisture makes its appearance within twelve hours from the commencement of the first paroxysm, and the patient awakes to consciousness perspiring profusely. The headache and giddiness pass off, and if the fever which preceded it was remittent, there may be a well-marked remission; if it was an intermittent, there may be a distinct intermission. With the next remittent exacerbation or during the hot stage of an intermittent, the pain in the head, giddiness, unconsciousness, and all the symptoms already described will return with greater intensity than before. With the second attack the patient may pass into a fatal coma.

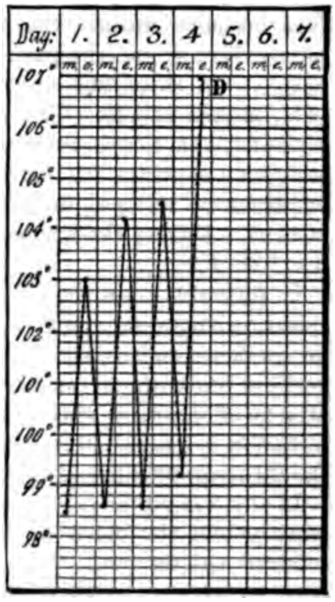

FIG. 179.

Temperature Record in a case of Pernicious Fever.

(*Comatose variety.*)

In this variety patients sometimes pass into a condition of apparent death, which may last for hours. Some are, nevertheless, perfectly conscious, seeing and hearing everything which occurs around them, although unable to move or utter a sound; others are unconscious. Even though the strongest counter-irritants are applied to the surface, there is no sign of life, until, at the beginning of the sweating stage, the patient comes to consciousness. If a patient survives the second paroxysm, quite probably he will die during the third. With each successive paroxysm the prognosis becomes more and more unfavorable; patients sometimes lie in a comatose condition for days, and finally die apparently from cerebral congestion.

Delirious Variety.—In this variety, the patient, after passing into the hot stage of an intermittent or into the exacerbation of a remittent, becomes delirious. Mild delirium is not uncommon during the progress of an intermittent or a remittent fever, but the delirium now referred to is of a more active character. If delirium is developed during the exacerbation of a remittent or during the hot stage of an intermittent, which has been preceded by severe headache, dizziness, ringing in the ears, and great restlessness, one may be quite certain that he has to deal with a case of pernicious fever, especially if it is prevailing in the locality. In this variety of pernicious fever there will also be more or less headache during the interval, and perhaps other peculiar cerebral phenomena. The delirium which appears is always violent in character; perhaps the patient will require restraint; he may be disposed to jump out of the window, or in some way to do injury to himself or those around him. During the paroxysm of delirium the face becomes flushed, the eyes brilliant, the conjunctivæ injected, the pupils dilated, and the patient is constantly crying, singing, and trying to escape. In those who are extremely anæmic the countenance assumes a pale, sunken aspect. The pulse is full and hard, and the carotids beat violently, the temperature often reaches 107° or 108° F. This delirious state may continue for hours. Suddenly the patient passes from it into a condition of collapse, or gradually sinks into a coma from which he never wakens. During the whole period the axillary temperature rarely falls below 105° F.

In favorable cases the delirium gradually becomes milder, a profuse perspiration comes on, and the patient falls into a prolonged sleep, from which he awakens conscious, though weak and exhausted, with headache and vertigo, but without the slightest recollection of what has passed. These attacks of delirium may be repeated three or four times before a fatal termination is reached, but so much danger attends them, that a second attack should never be allowed to occur if it can be prevented.

In this variety of pernicious fever, other nervous phenomena may accompany or take the place of the delirium, such as epileptiform convulsions, tetantic spasms, etc. The tetanic spasms sometimes resemble the phenomena of hydrophobia. That form of tetanus which occurs in various malarial districts, and is sometimes called sporadic tetanus, I believe will be found to have many things in common with this type of pernicious fever.

Gastro-Enteric Variety.—In this variety the patient, after he has passed into the hot stage of an intermittent, or the exacerbation of a remittent, is seized with almost incessant vomiting and purging. The vomiting and purging are peculiar, altogether unlike that which is sometimes present in the simpler forms of malarial fever. There is blood-stained material, both in the matter vomited and in that discharged from the bowels. In some instances, the discharges may be so reddened as to look like beef-brine or the washings of raw meat; sometimes the proportion of blood is so great as to cause the discharges to have the appearance of clear blood. In some endemics the discharges assume the appearance of rice-water, having no odor, and similar in appearance to those of Asiatic cholera. The patient has no abdominal pain or tenderness, but has a sense of weight and burning in the stomach, accompanied with cramps in the calves of the legs, coldness and blueness of the surface, with a small, almost imperceptible pulse, sunken eyes, and the "facies" of cholera. So closely do these patients resemble in appearance those of Asiatic cholera that this disease has frequently been mistaken for it. During the attack the thirst is most intense. The respiration is peculiar; it consists of a double inspiration, followed by a double sighing expiration. The restlessness is very great, the patient is constantly tossing from one side to the other; sometimes, an hour or two before death, he suddenly springs up and walks across the room.

The usual length of the fatal paroxysm is from three to six hours. Patients die in a state of collapse. After the vomiting and diarrhœa have assumed the characteristic appearances already described, very few patients recover. As death approaches, the pulse becomes more frequent, feeble, irregular, and fluttering in character. The respiration is more and more prolonged and sighing, the skin cold and shrivelled, and covered with a cold, clammy perspiration. It frequently happens when all these symptoms are present that the patient cannot be convinced that he is seriously ill, and wishes to get out of bed and go out of doors.

Algid Variety.—This variety is characterized by coldness of the surface of the body, while the rectal temperature may range from 104° to 107° F. The attack begins with a chill of not unusual severity or duration, but soon after the patient enters into the hot stage of the paroxysm, or, during the exacerbation of a remittent, the surface of the body begins to grow cold, while at the same time he complains of a sensation of burning and intense thirst. A cold perspiration soon covers the surface. The pulse becomes slower and slower, falters, and disappears at the wrist. Alternately the extremities and face become cold; only the abdomen retains its normal temperature. The surface has a cold, marble-like feel, and the temperature in the axilla may fall to 88° or 84° F. In the comatose and delirious varieties the temperature rises higher than normal, and may reach 106° or 107° F., but in this variety it sometimes falls two or three degrees below the normal. The tongue becomes white, moist, and cold; the breath is cold, and the voice feeble and indistinct. The action of the heart is feeble, often perceptible only on auscultation. The mouth is clean, and the patient seems to himself to be in a comfortable

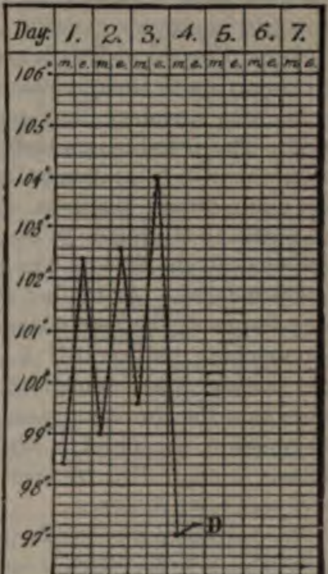

FIG. 180.
Temperature Record in a case of Pernicious Fever.
(*Algid variety.*)

condition, except that he feels exhausted and is sensible of great internal heat. The mind is clear. The expression of the countenance is that of death.

This variety is very insidious in its progress. To one not familiar with it, the calm which follows the febrile excitement will be mistaken for relief, perhaps attributed to some plan of treatment pursued, or to some remedial agent which has been employed. If a patient in one of these paroxysms is to pass on to recovery, the pulse gradually returns in the wrist, and the surface regains its normal feel and temperature. As the warmth returns to the surface, the patient passes on to convalescence in the same manner as patients recover from a comatose or delirious paroxysm. An *algid* pernicious paroxysm is rarely preceded by a distinct intermission, and it rarely has any appreciable remission. Once established, it marches steadily on to a fatal issue, unless arrested by treatment.

There is another variety which will occasionally be met with, in which a profuse perspiration, called a "*colliquative sweat,*" comes on at the end of the fever stage and continues through the succeeding intermission, accompanied by great prostration, feeble heart action, and labored respiration. Upon the second or third return of this sweat the patient sinks and dies, apparently from exhaustion. Again, severe hemorrhages from the stomach, bowels, or kidneys may occur during the sweating stage of a pernicious paroxysm and endanger the life of the patient from sudden syncope.

A mild form of hæmaturia sometimes occurs independent of a pernicious paroxysm in chronic malarial poisoning.

Icteric Variety.—This is always endemic, confined to certain localities, and occurring in them whenever any form of pernicious fever prevails. It begins with a violent, long-continued chill, during which jaundice shows itself. The jaundice gradually deepens, and extends over the whole body. Intense nausea accompanies its development, with a copious vomiting of bile, and a bilious diarrhœa. The patient suffers with a most intense headache, pain in the region of the spleen and over the kidneys, and a feeling of numbness in the limbs. The pulse is small, frequent, and hard. The urine is dark colored. As the hot stage comes on, the pulse becomes fuller and more frequent, the respiration is labored, the skin very hot, the temperature reaching 106° or 107° F., and the thirst is most intense. This stage lasts three or four hours, and often terminates in death. If the patient passes into the sweating stage, recovery usually takes place. During the intermission the mind is clear, but the jaundice continues. Unless the disease is controlled by treatment, each succeeding paroxysm becomes more and more severe.

This variety is incorrectly called pernicious bilious remittent fever. If the attack is mild, there is only a slight staining of the skin, but in that form in which there is an apparent arrest of the functions of the liver, the patient may die deeply jaundiced within two or three days after the first discoloration appears. The mild form of so-called bilious remittent fever, in which the febrile movement is constant, is very different from the form under discussion, and is better classed under the head of simple remittent. All these different varieties depend on the same blood-poisoning, differing in its manifestations according to its intensity and the predisposing atmospheric or septic conditions which may exist in the localities where they are developed.

Differential Diagnosis.—The diagnosis of pernicious fever is sometimes very difficult. In determining whether a given case is, or is not, one of pernicious fever, the first inquiry will be in regard to the character of the prevailing fever. If pernicious fever is prevailing in the locality, a diagnosis will easily be made ; if, however, the first case in the locality falls under observation, probably great difficulty will be experienced in making a diagnosis, and this difficulty, to a certain extent, will vary with the type of the fever. If, for example, a case belongs to that class in which there is a tendency to coma, delirium, etc., it may be confounded with some form of cerebral disease. This form of pernicious fever has been mistaken for *cerebral apoplexy*, *meningitis*, and *acute uræmia*.

As a rule, it is not difficult to draw the line between apoplexy and pernicious fever of the comatose or delirious variety. The constant and prominent symptom of *apoplexy* is hemiplegia, which is of rare occurrence in pernicious fever. It may occur, but if it does, it is developed slowly. Neither coma nor hemiplegia is ever reached suddenly in pernicious fever. Rise in temperature, rapid pulse, and all the phenomena of intense febrile excitement are present before the occurrence of either. On the other hand, in apoplexy the hemiplegia is of sudden development, attended by a slow pulse, irregular contracted pupils ; or, perhaps, one pupil is dilated and the other contracted, and its occurrence is preceded by a sudden loss of consciousness, and not attended or preceded by high febrile excitement.

As regards pernicious fever and *meningitis*, although in both diseases the patient reaches a condition of coma, yet in *meningitis* days elapse before the coma is reached, and during those days there has been pain in the head, photophobia, and delirium, extending over a considerable period of time ; whereas, in pernicious fever the patient reaches his condition of coma within twelve hours. Besides, in pernicious fever there will be a history, not only of the prevailing type of malarial disease, which will indicate its character, but the attack of coma or delirium will be preceded by a distinct malarial paroxysm—perhaps two of these paroxysms ; then the patient will pass rapidly into a state of coma. In meningitis the fever rarely ranges above 102° or 103°, the face is pale, the abdomen retracted, and the pulse is tense and wiry—all markedly contrasting with delirious pernicious fever.

The gastro-enteric and cold or algid varieties of pernicious fever closely resemble *cholera*. They may be distinguished from it by the character of the primary discharges. There may be a time in this type of pernicious fever when the discharges will very closely resemble those of cholera ; but they will always have been preceded by one or two bloody discharges. In cholera there is albumen in the urine, the occurrence of which is comparatively rare in pernicious fever. In cholera there are the peculiar surroundings of the patient, the prevalence of cholera in the locality, etc.

When the endemic is at its height it is almost impossible to make a differential diagnosis between the two diseases from the clinical history of the cases; but, when we take the early history of the endemic, at which time the cases at their commencement were marked by distinct intermittent or remittent paroxysms, the true character of the disease is very readily determined. If in any given case there is still a question whether it is or is not one of pernicious fever, it may be determind with positiveness by a microscopical examination of the blood, which will be found to contain free pigment.

The icteric variety of pernicious fever which often, in many of its phenomena, so closely resembles *yellow fever*, may be distinguished from it not only by the history of its development, but by the fact that when it prevails as an endemic, those are seized with the fever who have been longest under the influence of malarial poison, whereas new-comers are not usually attacked; in yellow fever districts new-comers are almost certain to contract the disease. The symptoms in icteroid pernicious fever tend to become typhoid and adynamic, while in yellow fever the symptoms are active and there is little tendency to a typhoid condition. Then the jaundice of yellow fever appears late in the disease, while the jaundice of this form of pernicious fever comes on early, even before the chill passes away. Again, bloody urine is frequently present in this type of pernicious fever, while in yellow fever hæmaturia rarely occurs without the accompanying evidences of nephritic inflammation. The presence of free pigment in the blood will aid in settling the question of diagnosis in difficult cases.

Prognosis.—In all varieties of pernicious fever the prognosis is unfavorable, unless it can be controlled before the occurrence of the second paroxysm. The prognosis will depend in a great degree upon the character of the prevailing endemic or epidemic, as also upon the stage of the epidemic, for the ratio of mortality is always greater during the earlier period of an epidemic than during its decline. All agree that the prognosis is better in every variety of pernicious fever if there are distinct intermissions, however short may be their duration. If the paroxysm does not last more than twelve hours, and terminates in a distinct remission, the prognosis is far better than when one paroxysm follows another without any distinct remission. Unquestionably the most favorable cases are those of the tertian type. Those varieties in which the cases most frequently terminate fatally are the gastro-enteric and the algid; those in which recovery is most likely to occur are the comatose and delirious.

The prognosis is also much influenced by the age and condition of the patient and by the presence or absence of complications. The mortality is greatest among the very young and very old, and among the intemperate. Patients with pernicious fever may die suddenly during a paroxysm, or the paroxysms may be prolonged and run into each other, and the patient may finally pass into a typhoid or collapsed condition. If the second or third paroxysm is not attended by signs of intense visceral congestion, if it declines with profuse warm sweats, if it has been preceded by distinct intervals, if the urine is free, and the appetite early returns, a speedy recovery is at hand. On the other hand, if the second or third paroxysm is protracted and accompanied by great anxiety and restlessness, with active delirium and a tendency to coma, with coldness of the surface; if there is intense pain in the epigastrium, with tingling of the surface, and scanty and high-colored urine; if there is profuse vomiting and purging, bleeding at the nose, and cold, colliquative sweats; if the pulse becomes small and feeble, or the radial pulse is imperceptible, the danger is very great, and a fatal issue is almost certain. Sometimes severe and fatal dysentery comes on at the end of a paroxysm; at other times, as the paroxysm subsides, the fever assumes a typhoid type, and, after a period of continued fever ranging from ten to twelve days, it terminates fatally.

Treatment.—The expectant plan of treatment cannot be practised in the treatment of pernicious fevers. The alarming symptoms crowd upon one another with great rapidity, and it is only by prompt and vigorous measures that in the severe forms of the disease the patient can be rescued from impending death. The issue of life or death often hangs upon a single hour. Some have proposed, before administering the only specific which we possess capable of controlling this disease, to produce free purgation by the administration of cathartics; others to bleed and freely vomit the patients. If the case is one of the gastro-enteric variety, emetics and purgatives are certainly very plainly contraindicated. It is now a well established fact that in no variety of pernicious fever do patients bear depletion. In India, where the most severe types of this fever prevail, the English surgeons are very positive in their testimony upon this point. All forms of depletion have been abandoned by them. Although stimulating enemata and friction to the surface may act as aids in the management of the algid and delirious varieties, they must not be relied upon as having any controlling influence over the disease.

Those who have had the most extended opportunities for testing the different remedies and plans of treatment which have been employed in the management of this fever are united in the opinion that quinine and opium are the only agents which can be relied upon for controlling its different varieties. In fact, the hypodermic use of these drugs has inaugurated a new era in its treatment, for in a large proportion of the severer forms it is impossible to get the full effect of either of these remedies by the ordinary methods of their administration, the usual avenues for their introduction being closed.[1]

Whatever solution may be used, administer from five to seven grains of

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[1] The solution of quinine commonly employed by the English surgeons for this purpose is made by adding one hundred and fifty grains of quinine and fifty drops of dilute hydrochloric acid to four ounces of water, and then evaporating the solution to two ounces. Of this, thirty drops may be administered at each injection. Some add carbolic acid to a solution of quinine in dilute sulphuric acid; the carbolic acid is added to prevent abscess at the point where the injection is introduced.

The formula for this solution is as follows:

Quiniæ disulphatis	gr. l.
Acidi sulphuric. dil	♏ v.
Acidi carbolici	♏ ij.
Aquæ destillat.	℥ i.

M.

Thirty minims is the quantity usually administered at each hypodermic injection.

It may be distinguished from *rheumatism*, as it prevails epidemically, and a period of febrile excitement precedes the arthritic phenomena.

It may be distinguished from *relapsing fever* by the eruption and by the character of the remissions.

Prognosis.—The prognosis is always favorable, although the symptoms which attend its development may be alarmingly severe. The prognosis is only unfavorable when it occurs in the very aged or in feeble infants.[1]

Treatment.—This fever always runs a definite course, and its treatment is the symptomatic treatment of fever, combined with well-recognized anti-rheumatic remedies. It is claimed that emetics and free purgation diminish its severity. A favorite combination is ipecacuanha, calomel, and colchicum—which is to be administered every night in cathartic doses. Calomel should never be administered alone, nor in combination with other drugs, if its specific effect is likely to be produced. The administration of colchicum with spirits of nitre and nitrate of potash, in such proportion that profuse diaphoresis may be produced, in connection with effervescing draughts, will usually afford relief from the pain in the head and limbs. Should the arthritic pains persist, opium may be administered in sufficient quantity to afford relief. Salicylate of soda or salol are of great benefit where arthritic pains are severe.

During the remission the bowels should be kept freely opened with salines, and quinine combined with an alkali should be given at stated intervals. Narcotics may be given in small doses to produce sleep, should the patient be wakeful. By the employment of these measures a return of fever may be prevented and the arthritic pains will gradually subside. If this plan is pursued, should the fever return, it will be mild in character, attended by little constitutional disturbance. The weakness and exhaustion which attend convalescence may be combated by the free use of wine or malt liquors.

The diet should be most nutritious. Nourishment should be administered at stated intervals, during the night as well as during the day. The lymphatic enlargement, especially in the inguinal region, should be treated locally with iodine. Citrate of iron and quinine will be found of great service during convalescence. If a single joint remains swollen and tender for a considerable period after the subsidence of the fever, the occasional application of a blister is recommended.

In some epidemics, relapses after an interval of two or three weeks have been of frequent occurrence. They run a milder course than the primary fever. The relapses more closely resemble an attack of articular rheumatism than the primary fever. Quinine is said to furnish great protection against a relapse.

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[1] Among sequelæ Thomas records heart affections, but does not say what these are; though he says they are not the same as in rheumatism. Peripheral paralysis of the forearm may occur. Catarrh of the fauces, trachea or œsophagus is mentioned as a sequela.

CHRONIC MALARIAL INFECTION.

Malarial cachexia, or *chronic malarial infection*, may be a sequela of any form of acute malarial disease. It may be developed in those who have never suffered from any form of malarial fever, but who have resided for some time in a malarial district. One who has had repeated attacks of intermittent or remittent fever, or has simply lived for some time in a malarial district and has become exceedingly anæmic, with an enlarged spleen and enlarged liver, may be regarded as in a condition of chronic malarial cachexia.

Morbid Anatomy.—The morbid anatomy of chronic malarial infection does not differ from that of the severer types of malarial fever, except in the more advanced stages of the tissue-changes. Thus the spleen is often ten or twelve times its normal size, tough, firm and resistant. Its surface is uneven, its capsule thickened and more or less adherent to the adjacent organs. Its substance is rich in pigment matter, and presents the minute changes, either of simple hyperplasia or amyloid degeneration. Similar tissue-changes take place in the liver and kidneys. In some instances the muscular tissue of the heart undergoes fatty or amyloid degenerative changes. Œdema of the subcutaneous cellular tissue, and an accumulation of fluid in the serous cavities, are common attendants of chronic malarial cachexia.

Etiology.—It may be the result of prolonged exposure in a district only slightly malarial, or of a short exposure in a district strongly malarial.

Symptoms.—Those who are the subjects of chronic malarial infection complain of vertigo, ringing in the ears, loss of memory, disturbances of sight, loss of appetite, nausea, dyspeptic symptoms, and pain and oppression in the epigastrium. The bowels are rarely constipated; diarrhœa is often present in the morning. The sleep is disturbed; it may be profound, but it is unrefreshing. The patient awakes in the morning with a confused feeling about the head and a general feeling of discomfort. Some complain of pains in the back and loins and along the sciatic nerve; others of pain and tenderness in the joints and stiffness of the muscles of the limbs and back; they become easily fatigued on exertion, have shortness of breath and palpitation of the heart.

The nervous system seems to suffer most severely. One of the most common nervous manifestations is local anæsthesia, which usually shows itself upon the outer surface of the thighs. Itching, burning, and a sense of formication, tingling, or numbness are very common symptoms. Not infrequently numbness of the arms and fingers and tickling and burning of the feet are complained of, and a patient will fear that he is about to have an attack of paralysis.

Hemiplegia sometimes occurs. I remember one case in which there was complete loss of power of the right arm and leg, yet no facial paralysis. This patient had never had a paroxysm of malarial fever, and for that reason the possibility of malarial infection had been excluded.

Chronic malarial infection may be unattended by any nervous manifestations. This form shows itself in catarrhal inflammations affecting the mucous membrane of the stomach, intestines and bronchial tubes. Patients have a form of bronchitis which is really a chronic malarial affection. A gastro-enteritis, in which there is marked interference with digestion, may be developed. If this is treated with the ordinary remedies for dyspepsia, no good result is accomplished, while a few doses of quinine will relieve the patient and establish the diagnosis. The chronic intestinal catarrh which results from chronic malarial infection may give rise to a troublesome diarrhœa which will assume all the characteristics of chronic diarrhœa. As already stated, anæmia is a very common result, and palpitation of the heart is a very frequent and sometimes distressing accompaniment of such anæmia. It often gives rise to temporary attacks of melancholia and hypochondriasis. Persons imagine they have disease of the heart, kidney, or spine, etc.

Another nervous manifestation of chronic malarial infection is neuralgia. Certain nerve-trunks or their roots seem to be directly involved, while the nerve-centre connected with the affected nerve-trunks escapes. The first branch of the fifth nerve is most liable to be affected. This neuralgia follows a periodic course. Persons over forty are most liable to be affected by it. Usually the nerve-trunks first affected are the ones involved in successive attacks; thus if a certain intercostal nerve is the seat of the primary neuralgic paroxysm, at each subsequent attack this particular nerve will be the seat of the neuralgia.

In some instances chronic malarial infection manifests itself by hemorrhages from the mucous surfaces, such as epistaxis, hæmatemesis, hæmaturia, etc. The most troublesome cases of menorrhagia (occurring independent of a mechanical cause) often recover after the administration of large doses of quinine, when all the remedies ordinarily employed in such cases have failed to produce the desired result.

Differential Diagnosis.—The first question that now arises is: How can we decide whether the manifestations are malarial or non-malarial? In the majority of cases there will be some enlargement of the spleen. There is not necessarily any rise in temperature. The manifestations will be more or less paroxysmal. If the patient has localized anæsthesia or hyperæsthesia, it will be found to be more severe at some particular hour in the morning or evening. If the patient has lost power over one portion of the body, he will find that the loss of power is more marked at a certain period of the day. The patient may not observe this periodic tendency, and it is frequently elicited only after careful examination and close questioning by the physician. In the severer cases of chronic malarial infection, when there is hemiplegia or some structural change affecting the mucous membrane of the stomach, intestines, bronchial tubes, etc., there are also evidences of pigmentation of the tissues. Free pigment is frequently found in the blood.

The diagnosis of chronic malarial infection, to a certain extent, depends upon the circumstances which attend its development. If the individual has repeatedly suffered from malarial paroxysms, or if he has resided for some time in a malarial district without having had a distinct malarial paroxysm, and although the peculiar malarial cachexia which is so characteristic of malarial poisoning is not present, yet it is always well to carefully consider the question of malarial infection.

While the manifestations of chronic malarial poisoning are legion—and in many instances they very closely simulate the phenomena of other diseases—still, with a history of possible malarial exposure, and after excluding all other conditions, we determine that the patient is suffering from some form of blood poisoning, and then the nature of the poisoning is readily determined. In very doubtful cases one may confirm an uncertain diagnosis by treatment.

Prognosis.—The prognosis in chronic malarial infection depends upon the severity of its manifestations, and the degree of enlargement of the spleen and liver. When the symptoms are mild and the spleen is but slightly enlarged, and when neither ascites nor œdema of the lower extremities is present, the prognosis is generally good. If the patient is very anæmic, the spleen very greatly enlarged, and the area of hepatic dulness very much increased, the prognosis is unfavorable. When distinct tumors can be detected in the spleen and liver, they indicate an exceedingly grave form of malarial infection; if the tumors are large, they can rarely be reduced. If the individual in whom these tumors are found removes from a malarial district, a long time may elapse before they very much interfere with his health and comfort.

The possibility of a patient being able to take up his permanent residence in a non-malarious region must be taken into consideration before a prognosis is given in any case. So long as such a patient is under malarial influences, however slight, the progress of the disease cannot be permanently arrested; and when the manifestations of the graver forms of malarial infection are present, there is little prospect that the disease can be temporarily relieved while he remains in a malarial district.

Treatment.—The first and most important thing to be accomplished in the treatment of chronic malarial infection *is the removal of the individual from a malarious district to a high, warm, mountainous region.* It is of the greatest importance that all exposure to wet and cold, and the damp air of the evenings and nights, should be avoided; the sleeping apartments must be dry and airy, and flannel should be worn next to the skin. So long as the thermometer shows even a slight febrile movement, quinine must be given in full doses. If anæmia is present, iron must be combined with the quinine, and administered immediately before or after taking food. In those cases in which the spleen and liver are very much enlarged, when no febrile excitement is present, *iodide of iron combined with cod-liver oil* will be found of great service.

It is claimed by some that muriate of ammonia has a very beneficial effect in this class of cases, but my own experience does not lead me to favor it.

One-half an ounce of Warburg's tincture taken daily for ten days, two hours before breakfast in the morning, is often efficacious when quinine fails. If the bowels are constipated, aloes or rhubarb should be given in connection with some of the saline mineral waters.

In those cases in which the measures already referred to fail to produce any improvement or afford any permanent relief, arsenic may be resorted to, but the effects of the drug must be carefully watched, and on the appearance of œdema or of gastric disturbance, it must be promptly discontinued. It must be borne in mind that the use of all these therapeutic agents is not sufficient; proper attention must be paid to hygienic measures.

The neuralgiæ which are such frequent manifestations of this infection are best treated by combining a full dose of opium with large doses of quinine. If paralysis is a manifestation of the malarial poisoning, strychnine, iron and quinine may be combined in its treatment, in connection with cold douches and friction to the paralyzed limbs. A most nutritious diet and a liberal use of good wine are indicated in all cases. The daily use of brandy in small quantities is often of great service.

In regard to the use of quinine in this class of cases, I am convinced that its indiscriminate use does harm. After fairly testing its effects, if no relief is obtained, it should be discontinued for a time, or at least until the beneficial effect of a removal from a malarial district is tried, or until, by the use of mild cathartics and daily administration of cod-liver oil and iron, the patient is in a condition to be benefited by it. Quinine seems to have no effect upon many who suffer from the severe manifestations of this infection, so long as they remain in a malarial district, but as soon as they remove to a non-malarial district it acts promptly. It is of the greatest importance that one should be familiar with the condition in which quinine is indicated in the treatment of this class of affections. Avoid depressing remedies in all forms of chronic malarial infection: drastic cathartics, exhausting diaphoretics, and other depressing remedies must not be used. They do great harm by exhausting the already enfeebled vital powers. Especially is this true in regard to the free use of mercurials, which are so commonly resorted to in this affection. Unquestionably, an occasional cathartic dose of calomel is of service, but the administration of small doses repeated after short intervals to produce its constitutional effects, will always be followed by the more serious manifestations of the malarial infection.

The exhausted system of this class of patients needs rest, concentrated nutrition, and the influence of a change of climate.

SECTION V.

CHRONIC GENERAL DISEASES.

1. Rheumatism.
2. Gout.
3. Lithæmia.
4. Diabetes.
5. Anæmia.
6. Chlorosis.
7. Progressive Pernicious Anæmia.
8. Leucocythæmia.
9. Pseudo-Leukæmia.
10. Addison's Disease.
11. Ammonæmia.
12. Hæmophilia.
13. Scurvy.
14. Purpura.
15. Myxœdema.
16. Scrofula.
17. Rickets.
18. Alcoholism.
19. Trichinosis.
20. Syphilis.

RHEUMATISM.

Rheumatism is a term still vaguely used to cover all inflammatory and painful affections of the fibrous tissues about the joints and in the muscles which depend upon some constitutional morbid state. There are five distinct varieties :—

1. Acute Articular Rheumatism, or Rheumatic Fever.
2. Sub-acute Articular Rheumatism.
3. Chronic Articular Rheumatism.
4. Arthritis Deformans.
5. Muscular Rheumatism, "Myalgia."

ACUTE ARTICULAR RHEUMATISM.

Acute articular rheumatism, or rheumatic fever, is the most acute manifestation of the morbid constitutional state.

Morbid Anatomy.—The blood when drawn from the vessels coagulates rapidly, the fibrin which can be derived from it is in excess of the normal ; sometimes it reaches ten per cent., and it is readily separated from the other constituents. The number of red discs is diminished and the serum is alkaline.

The joints are the chief points of attack ; yet in many cases where they have been greatly enlarged and excruciatingly painful during life, *no change* has been detected after death. The synovial membrane is usually injected ; the capillaries are dilated, and the reddening is best marked where the membrane joins the cartilage. The cells of the synovial fringes multiply ; the epithelial cells are enlarged and often surrounded by fat. The lymphatics of the synovial adventitia are enlarged ; and the cartilage-cells proliferate and the fundamental substance segments. Sometimes the articular cartilages are œdematous. Small hemorrhages may occur in, or a thin fibrinous exudation may cover, the synovial membrane. The ends of the bones have been found abnormally vascular in a few cases. Inflammatory œdema of the peri-articular tissues is very common.[1] The fluid in the joint-cavity may be normal, or it may be increased in amount and slightly turbid. Flocculi of lymph are sometimes seen floating in it; and at times it contains an abnormal number of cell elements. These elements often undergo fatty change, and may resemble Gluge's corpuscles. The color of the fluid varies; its reaction is alkaline. Albumen and fibrin are found in abundance. Urate of soda is *never* found. If the temperature has been high, the liver and other internal organs may show *cloudy swelling*, or parenchymatous degeneration.

Etiology.—Acute rheumatism may be regarded as a constitutional disease,—"a specific inflammation of joint structures attended with fever." Some claim that there is an excess of sulphur in the blood of rheumatic patients; others regard the disease as due to a change in the normal relations of the salts. Another view is that *lactic acid*—the normal product of nutritive changes in the tissues—accumulates in the blood in excess on account of a change in the blood salts, consequent upon some change in the albumen.[2] It is a disease of temperate climates, occurring mostly between December and March. In common with gout, diabetes, and arterial sclerosis, its etiology is very closely connected with disturbances of the hepatic function.

There is an hereditary tendency in about thirty per cent. of cases. It attacks persons between fifteen and thirty oftener than those of any other age. In old age it is exceedingly rare.[3] It is most frequently met with in those exposed to wet and cold, as cabmen, laborers, and maid-servants. It attacks men oftener than it does women. Insufficiency, or a poor quality of food, and prolonged residence in a damp atmosphere or dwelling predispose to it.

Any impairment of the general health from defective nutrition renders one more liable to a rheumatic attack. Erysipelas, dysentery, scarlatina and gonorrhœa are named among its exciting causes. Pregnancy is said to have caused rheumatic arthritis, and prolonged lactation may induce it. Scrofula, phthisis, and cancerous affections so often precede rheumatism that a connection between them cannot be denied. The exciting causes in one predisposed to it are exposure of the unprotected surface to sudden changes of temperature, to wit: cold and suddenly checking perspiration.

Symptoms.—In many instances dyspeptic symptoms precede a rheumatic attack, which usually comes on suddenly at night. A distinct febrile movement may precede the articular symptoms for twenty-four or forty-eight hours. Uneasiness and restlessness, a vague feeling of *malaise*, or slight and indefinite pain in the joints that are to be affected, may also precede its development. In all cases there is aching pain and stiffness in some one of the larger joints, which rapidly increase in severity, accompanied by a rise in temperature proportional to the rapidity of the development of the articular symptoms and the number of joints involved. The temperature commonly ranges from 102° to 103° F.; but it occasionally rises to 105° F., and 109° and 110° F. have been reached before death in severe cases. There are distinctly marked exacerbations and remissions, the minimum temperature being *nocturnal.* The pulse is full, bounding and compressible, as a rule keeping pace with the temperature; it often reaches 120 per minute *independent* of the fever, seemingly on account of the severe pain.

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[1] Garrod states that the only change found in some cases is a lax state of the ligaments and opacity of the cartilages.

[2] Richardson's experiments consisted in injection of lactic acid into the peritoneal cavity of a cat. The next day peri- and endocarditis were developed. Canstatt regards the articular affection as the result of a disturbance of innervation, consequent upon peripheral irritation set up by a chill.

[3] Diseases of Old Age.—Charcot and Loomis. N. Y., 1881, pp. 150, 151.

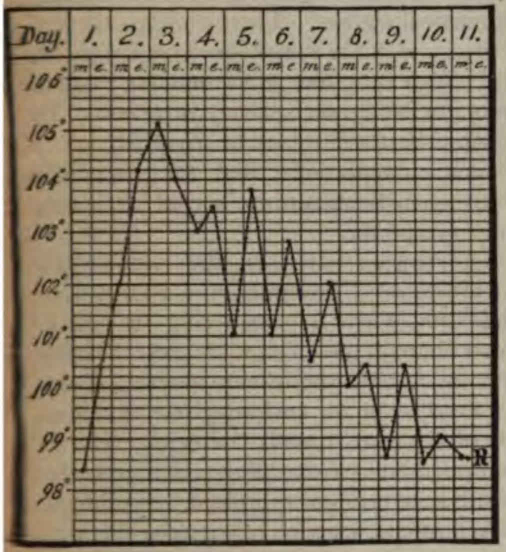

FIG. 182.
Temperature Record in a mild case of Acute Rheumatism.

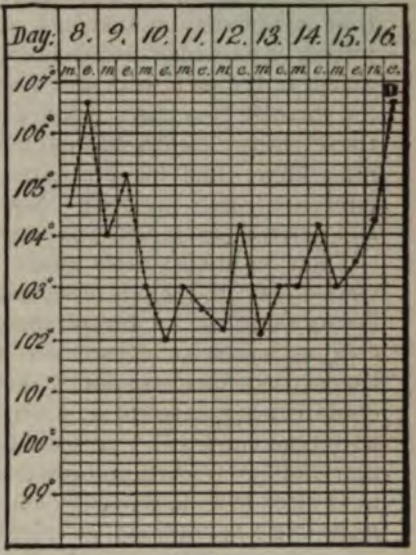

FIG. 183.
Temperature Record from the eighth day in a fatal case of Acute Rheumatism.

The joints attacked become red, swollen, and so tender that the weight of the bed-clothes cannot be borne, nor can the patient bear the shaking of the bed by the footsteps of attendants. Fluctuation can soon be made out in those joints whose situation permits examination. Muscular cramps usually accompany the joint symptoms. The face is flushed and the body is bathed in a copious, sour smelling, and acid sweat, especially about the affected joints. Sudamina are common. The saliva is said to become acid, and inflammatory fluids poured out into serous cavities during rheumatic fever have been found distinctly acid in reaction. There is great anxiety and restlessness, but the sufferer dares not move, so agonizing is the pain in the joint.

The urinary secretion is diminished, its color being darker than normal. On cooling it deposits a brick-dust sediment of urates. Its specific gravity varies from 1.025 to 1.030. Urea and coloring matter are in excess, while the chlorides are diminished. Sulphates and albumen are not infrequently found. Uric acid is increased.[1] The tongue is covered with a thick creamy fur. There is anorexia and great thirst, the bowels are usually constipated and the fæces are dry.

The large joints are usually involved first. The joint involvement is remarkably symmetrical : thus one ankle joint swells, and as soon as this subsides its fellow is immediately attacked, and the affection travels rapidly from one joint to another in a symmetrical course. There is little œdema, pitting or cuticular desquamation after the acute symptoms have subsided, except in the *very* weak and feeble. As a rule, one joint remains swollen for three or four days, while others are being successively involved. By the seventh or eighth day half a dozen articulations may be involved. The hip-, finger- and toe-joints, the spinal articulations, and the symphysis pubis are rarely involved. Not infrequently sudamina, herpes urticaria, and miliaria accompany the articular symptoms.

There is usually little mental disturbance ; but when the temperature reaches 105° F. there is often great restlessness, insomnia, and a mild wandering delirium. I regard this delirium (when not occurring in drunkards) as indicative of extensive blood changes and a sign of great danger. One of the characteristics of rheumatic fever is the intense anæmia developed in a few days after its onset, even when no antiphlogistic remedies have been employed. Auscultation of the heart reveals "blowing" hæmic murmurs, even when no cardiac complications exist. The articular symptoms often subside suddenly, and cardiac or cerebral symptoms as suddenly make their appearance. Cerebral symptoms are always attended by high temperature. The cerebral and cardiac phenomena are not due to a metastasis in the strict sense of the term, nor yet are they complications. There is some bond of connection between these cerebral and heart lesions and the joint affections,—the exact nature of the connection is not known. The fibrous tissue of the heart is as liable to be involved as the joints ; and in many young subjects the heart involvement precedes that of the joints.

Differential Diagnosis.—The connection between gout and *rheumatism* is considered under the head of gout.

Pyæmia may be mistaken for rheumatic fever ; but the *recurring* chills, sickly, *sweet* breath, slow development, jaundiced skin, previous history, and finally the presence of infarctions, multiple abscesses and thrombi are sufficient to distinguish pyæmia from rheumatic fever. Suppurative synovitis may occur in pyæmia, but not in acute rheumatism.

Non-rheumatic arthritis is to be distinguished by its *persistence in one joint*, by the absence of the characteristic sweats, by the graver local and more trivial constitutional symptoms, and by the absence of cardiac complications.

Prognosis.—Apart from the complications, acute polyarticular rheumatism is *not* a fatal disease ; three per cent. is its average death rate. The disease lasts from three to four days in one joint ; the whole duration of the fever and attendant arthritis is from three weeks to thirty days. The rule is that no crippling of the joints follows the acute attack. If the course of the disease is prolonged, complications are usually present; in many cases its course is erratic in that just as convalescence is apparently established all the acute symptoms reappear. It runs no cyclical course,—there is no definite limit to its duration; patients may recover in two or three days or a week, or it may persist for a month or longer; generally the milder the attack the shorter its duration. I have seen six or seven weeks elapse before the joints have returned to their normal state; in such cases all the joints become involved in succession and the articular phenomena are persistent. The more robust the individual the sooner is convalescence established. Sometimes the fever subsides and the joint affections persist.

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[1] Parkes and Garrod, respectively, found thirteen and eleven and one-half grains in a quart of urine.

The frequent occurrence of endo- and pericarditis leaves little doubt as to their intimate connection with the rheumatism. The younger the subject the more liable are the cardiac affections to occur. Statistics are most diverse on this point; some say five, others seventy-five per cent. of all cases of rheumatic fever are accompanied by cardiac inflammations. Another, graver, sequel is ulcerative endocarditis. The worst legacy acute rheumatism leaves is a crippled valvular apparatus in the heart.[1] Endarteritis, pleurisy, pneumonia, cerebral and spinal meningitis, laryngitis, bronchitis and peritonitis are its principal complications. A strange sequel of rheumatic fever is *chorea*.

Instead of complete recovery, acute rheumatism may become subacute or chronic. Muscular rheumatism frequently follows an attack of acute articular rheumatism.

Treatment.—The hygienic surroundings of rheumatic patients should be very carefully attended to. The temperature of the apartment should range from 68° to 70° F.; all draughts should be avoided, and the patient should be clothed in flannel and covered with flannel sheets. The *diet* should be milk and seltzer-water. If this is not well borne, concentrated food, *other than animal*, can be given. Animal food and alcoholic stimulants are contraindicated during the active period of the disease. As soon as the fever declines, nutritious and easily digested animal food may be freely given.

Only a few of the many *external* applications which have been made to the affected joints will be referred to. Cold by the means of ice-bags to the joints has been strongly recommended. Friction, with chloroform or opium in glycerine combined with alkalies and the tincture of aconite is a favorite plan with some. "Hot-packs" by means of flannel compresses wrung out in water as hot as the patient can bear, or bathing the joints in warm laudanum and then covering them with oiled silk is always grateful to the patient. Wunderlich and Niemeyer advocate, respectively, ethyl chloride and ether to be rubbed over the affected joints. The "blister plan," which consists in surrounding all the affected joints with fly blisters, temporarily relieves the pain, but the rheumatic attack is not shortened, nor do they afford any permanent benefit. My experience leads me to the opinion that if the affected joints are protected from changes of temperature by cotton batting and oiled silk, all is accomplished that can be by local applications.

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[1] Two cases of acute rheumatism were recently (July 31st, 1882) reported by Mayer in "Henoch's Klinik," where peculiar and significant complications were observed. The patients were children; and both had *little tumors* develop about the joints (malleoli, elbow, etc.), that on section, after death, proved to be connective-tissue neoplasms which had a tendency to necrobiosis or to osseous-like change.

Internal Medication.—Innumerable remedies have been brought to the notice of the profession as specifics in the treatment of rheumatism, yet it is still the most unmanageable of all diseases. Garrod is of the opinion that colored water is about as potent as anything. He claims that rheumatic fever is a "self-limiting disease," sometimes running a long, sometimes a short course. Bleeding, mercury, and antimony and mercury are no longer employed. Some advocate the use of iodide of potassium; some colchicum, some veratria, some guaiacum, and some quinine. Garrod's "quino-alkaline" plan, which combines quinine with the alkaline treatment, is a favorite method with many. It is claimed that the so-called alkaline treatment shortens its duration and diminishes the frequency of cardiac complications. It consists in the administration of the soda salts in from ʒv to ʒx daily until the urine becomes alkaline.

My experience inclines me to the opinion that while alkalies in the early stages relieve the pain in the joints, they do not shorten the duration of acute rheumatism, and if long continued they do positive harm.[1] The tincture of the chloride of iron, in one-half drachm doses, is especially serviceable as soon as the temperature reaches 100° F. Within the past few years salicin, salicylate of soda, and salicylic acid have been very extensively employed. It is claimed that immediate relief follows the administration of the salicylates—that the temperature falls, that the pain and swelling of the joints subside, and that the duration of severe attacks has been limited to thirty-six or forty-eight hours. But it causes great depression of the heart, increases the liability to cardiac complications, causes irritability of the stomach, and places the patient in a weak and debilitated state; for the past few years I have seldom employed it, for my experience shows that while in some cases it may relieve the urgent symptoms of the disease in two or three days, relapses are almost certain to follow, and the duration of the disease is not shortened, and I have seen very serious toxic effects follow its use. Salol is to be preferred to salicylic acid. Benzoic acid and benzoate of soda are claimed by some to act more efficiently than salicylic acid and to be less harmful. I have not seen any good results follow their use.

During the past few years I have obtained the most satisfactory results in the use of antipyrine during the acute stage of articular rheumatism. I have come to employ it almost to the exclusion of all other remedies for the relief of the arthritic pains, and in many cases it has seemed markedly to shorten the duration of the disease. The treatment of the various complications is considered under their appropriate heads.

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[1] Gull, Sutton and Lebert found marked diminution in the duration of rheumatism from steady administration of lemon juice.

SUB-ACUTE RHEUMATISM.

Sub-acute rheumatism is usually a sequela of acute, or occurs in one who has at some time had an acute attack ; it is attended by slight if any fever ; the pain in the joints is not severe, except on motion ; and the swelling and redness are not excessive, and are limited to one or two joints, usually the large ones. There is *no* tendency to disorganization or permanent crippling of the affected joints; and although it may last six or seven weeks, or three or four months, the joints usually return to their normal condition. The blood changes are the same as in acute. The articular symptoms are less metastatic than in acute ; anæmia is always well-marked, and cardiac complications are not infrequent.[1] The treatment is a milk diet, iron and cod-liver oil, and a warm climate, and heat to the affected joints. The so-called anti-rheumatics have failed in my hands to relieve or cure.

CHRONIC ARTICULAR RHEUMATISM.

Chronic articular rheumatism usually involves only a few joints ; it occurs most frequently in those who have had rheumatic fever in early life. Although it may be of long duration it rarely induces extensive changes in the joints.

Morbid Anatomy.—The parts affected are the fibrous tissue around the joints, the fibrous envelopes of the nerves, the aponeurotic sheaths of the muscles, the fasciæ, and the periosteum. The synovial membrane is thickened, the fringe-like processes are hypertrophied and very vascular, and the synovial fluid becomes turbid and cloudy. The ligaments are thickened, the cartilages relaxed, shaggy, and sometimes in a state of fatty degeneration. The more protracted the disease the greater the thickening of the peri-articular structures, and the thicker and scantier will be the fluid in the joint. The fibrous tissue developed about the joints causes more or less stiffness and loss of motion, but not ankylosis.

Etiology.—It is a disease of adult and advanced life, occurring oftenest in those whose hygienic surroundings are bad, and who are exposed to wet, cold, or sudden changes of temperature. A residence in dark and damp dwellings predisposes to it. Previous attacks of acute articular rheumatism develop a tendency to chronic rheumatism. It is often hereditary, and then there may be no appreciable exciting cause to its development. Both sexes are equally liable.

Symptoms.—There is aching and constant pain in some one or more of the larger joints, usually the knee or ankle, but sometimes those of the upper extremities. The affected joints are tender and slightly swollen, and their movements are constrained. No fever is present. The aching and deep-seated pains are often worse at night. When it is the result of *exposure*, heat will give a grateful sense of relief; when a rheumatic *diathesis* exists, the heat of the warm bed-clothes increases the pain, and relief is often obtained by exposure of the joint to dry cold. Old people with rheumatic joints are great "weather prophets," often being able to foretell the coming of a storm. After rest, motion gives great pain, but use renders the joint temporarily more supple and less painful. The pains undergo exacerbations and remissions, and the disease may continue for years without causing much deformity or great crippling of the joints. The muscles near the joint usually undergo more or less atrophy, and as a result the affected joints appear larger than they really are.

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[1] Barwell, who calls this disease "Sub-acute Rheumatic Synovitis," says it may lead to *hydrops articuli*. —Diseases of the Joints, 1881, p. 138, *et seq.*

Palpation may reveal fluctuation in the joint, and *auscultation* gives a rough, grating crepitus during motion.

Differential Diagnosis.—Chronic articular rheumatism may be distinguished from *arthritis deformans* by the absence of deformity of the affected joints, the history of previous acute rheumatic attacks, the large joints being mainly involved, the partial or complete recovery during warm weather, and its return on exposure to wet and cold ; arthritis deformans is a steadily progressive disease, one joint after another being involved and never recovered from.

Prognosis.—There is little chance of complete recovery after middle life; it is only possible in those cases treated at the onset under favorable hygienic conditions. In long-standing cases, wasting of the muscles may lead to great crippling of the joint. Muscular rheumatism is a frequent accompaniment of articular. Chronic rheumatism never affects the duration of life, except as it may deprive the patient of exercise and sleep, and thus interfere with nutrition.

Treatment.—This form of rheumatism is benefited mostly by local treatment, such as blisters, iodine, belladonna, aconite, opium, and chloroform liniments, veratrum ointment, etc., etc. Most rheumatic patients have their favorite prescriptions for local use, which they claim afford them almost instant relief. If there is but little pain in the joints, ammonia and turpentine liniments are of service. Thick flannels should always be worn about the joints. Some advocate, for both pain and stiffness, sponging the joints with hot water. The Galvanic or Faradic current will temporarily reduce the swelling and pain, and sometimes improves the mobility of the joint. Local or general baths form an essential part in the treatment of this form of rheumatism. Hot air, or vapor baths are not so efficacious as hot water. Many of the hot saline springs for bathing have acquired a great reputation in the treatment of this form of rheumatism, cures being effected in cases that had resisted all other methods of treatment.[1] Massage and rubbing are always beneficial ; and undoubtedly much of the good that is claimed for certain liniments is due to the rubbing and manipulation of the joints during their application.

Internally, tonics should be employed, such as iron, quinine, strychnine, etc. Cod-liver oil is the most useful of all internal remedies, and should be administered continually. All the means for improving the general health of the patient should be carefully considered, and if possible he should reside in a dry, warm climate. Colchicum, arsenic, iodide of potassium, and *guaiacum* have gained reputation in its treatment, as have also the turpentine and cajeput oils combined with sulphur; but I have been unable to find sufficient proofs of their beneficial effects to strongly recommend their use. Recently, Rawson advises guarana, and Heller liquor ammonii.[1]

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[1] Those which I have found most efficacious in this country are the Hot Springs of Arkansas, the Virginia Hot Springs, and the Richfield Springs in New York State.

The diet must be highly nutritious and absolutely non-stimulating; I am convinced that errors in diet and "fits of indigestion" prolong, and are powerful to develop this disease. Exercise is important, and if possible a sea voyage in a warm climate should be taken.

ARTHRITIS DEFORMANS.

(*Rheumatoid Arthritis.*)

Arthritis deformans is a chronic inflammation of the synovial capsule, the ligaments and tissues of the joint, unattended by suppuration and with little fluid accumulation in the articular cavity.

Morbid Anatomy.—The synovial membrane and articular cartilages are first involved. The fringes of the former are increased in number, and are very vascular: they are called "the destructive vegetations" of the synovial membrane. The central portion of the articular cartilages becomes roughened or villous, gets gradually worn down, and finally disappears, and the bones thus laid bare undergo eburnation. The ivory-like surfaces are striated, the striæ running in the direction of the articular movements.[2] While the *central* portion of the cartilage is disappearing, its margin forms nodular irregular outgrowths. The synovial fringes take part in the hypertrophic processes, and form pyriform excrescences, which, after a time, become converted into fibrous tissue. These outgrowths in some cases blend with the osseous structure of the epiphysis, and in others become detached and are free. Lateral expansion of the joint surfaces with enlargement of the ends of the bones takes place and leads to deformity, dislocation, and immobility. All the joints may, in course, be involved, especially those of the hands and feet. The thickened ligaments sometimes undergo partial cartilaginous, osseous, or fatty degeneration.

The *tendons* are sometimes thinned and ossified. The fluid in the joint cavity is thick, turbid and yellow, and alkaline in reaction. There are no blood changes; no *urates* are found in the joints. The adjacent muscles undergo atrophy.

Etiology.—This disease is regarded by some as a peculiar form of chronic rheumatism, while others regard it as an essentially different disease. It may occur at any *age;* but the tendency to it increases with advancing years. Women are more liable to it than men. The smaller joints are most often involved in women; the larger in men. It is *very* rare in dry, hot climates. Damp dwellings, poor food, and mental depression are powerful predisposing causes. Thin people suffer oftener than the corpulent. It is met with oftenest in the poorer classes.[1]

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[1] *Wien. Med. Presse*, Dec., 1875.

[2] Charcot calls eburnation a "sclerosis of bone, accompanied by vascularization of the deep parts."

Symptoms.—The symptoms of arthritis deformans are all referable to the changes which occur in the joints. It may come on insidiously, neuralgic pains sometimes preceding stiffness and deformity. As the soft parts atrophy, the joints stand out distorted and rigid from the flabby muscular surroundings. There are usually no severe inflammatory symptoms, the joints are only slightly tender to pressure, but motion always gives pain. Months and years may elapse before the articular changes are completed; but once started, they are progressive, until the joint is ankylosed or deformed.[2] The immobility of the joints depends upon the osteophytes or fibro-cartilaginous transformation of the synovial membrane or fibrous union of the surfaces of the bared bone.

In some cases the disease begins with all the signs of rheumatic fever, with inflammatory symptoms of a mild type. No cardiac signs are present, and *no excess of uric acid can be found in the blood.* The acute symptoms gradually subside, leaving the joint irreparably crippled. The small joints are usually first involved. The metacarpo-phalangeal articulations of the index and middle fingers are usually first attacked. In forty-five cases, the smaller joints of the hands and feet alone were involved in twenty-five; the great toe in four; the hands and feet with a large joint in seven; the large joint first, then the fingers in nine. After a time large joints, the temporo-maxillary articulation, or the articular processes of the vertebræ, especially the cervical, become involved. The hip, shoulder, elbow, knee, and hands are its favorite sites. In many joints *abnormal* mobility is developed, *i. e.*, the hip may slip up and down in its socket. Subluxations are common, in the fingers especially.

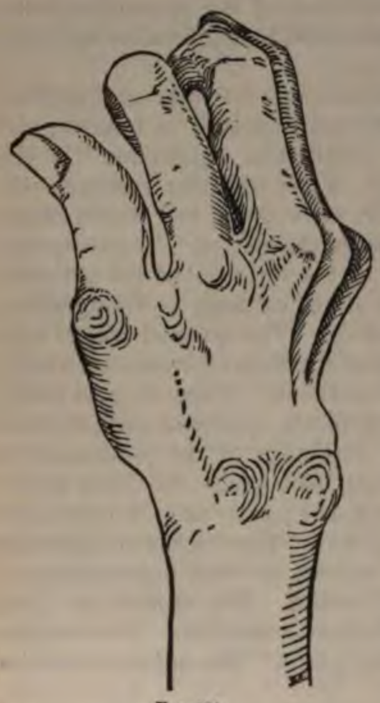

FIG. 184.

Deformity from Articular Rheumatism.

Early in the disease a friction crepitus is heard as the articular surfaces are rubbed upon each other, which becomes coarser as the disease advances. There are painful spasms of the muscles in the affected limb, more marked at night and just before a storm. When the disease is localized in the hip joint, it has been called "morbus coxæ senilis." In such cases the limb is shortened and the patient limps. The greatest variety of deformity takes place in the hands of those who have been long the subjects of this disease. The constitutional disturbance is never commensurate with the local signs. The skin becomes dry and harsh; there is great acidity of the stomach, cold extremities and a condition of extreme anæmia.[1]

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[1] Charcot advocates a nervous origin, especially when it begins in the smaller joints. Barwell regards it as due to *colitis*, originating in "some constitutional cachexia," which, in some of its tendencies, resembles the rheumatic diathesis.

[2] Remak believes that the painful swellings on either side of the joint are "*neurotic nodes.*"

Differential Diagnosis.—Arthritis deformans may be mistaken for *chronic articular rheumatism* without deformity, and *chronic gout.*

Gout is hereditary, and occurs more in males. Arthritis deformans is rarely hereditary, and occurs oftenest in females. Attacks of gout are periodic, and the small joints are found involved. Arthritis is progressive, and both large and small joints may be attacked. Kidney complications are common in gout and rare in arthritis. Chalk-stones develop in the joints in gout and are never present in arthritis. Uric acid is always in excess in gout, and never in arthritis; deformities and ankyloses are less marked and extensive in gout than in arthritis.

Prognosis.—Arthritis deformans never destroys life, and is never recovered from; patients with this disease may attain very old age. The greater the number of joints involved the more deplorable will become the condition of the patient. There is rarely complete ankylosis. When false joints form there is a possibility that such patients may walk or move about with comfort.[2]

Treatment.—Treatment of this disease is very unsatisfactory; for the most part we must trust to local measures for relief, and to such constitutional measures as shall improve the general health. Quinine, iron, cod-liver oil, arsenic and strychnia are indicated. The diet should be nutritious and easily digestible. Alcoholic stimulants, if they improve nutrition, are of service. Change of climate and habits of life is often followed by an arrest in its progress. Flannels should always be worn next the skin. Mineral waters and warm saline baths, either artificial or natural, often temporarily arrest its progress and relieve the pain in the joints. The preparations of *iodine* and the acute rheumatism and gout remedies have seemed to me to do more harm than good. Local frictions with iodine, mercury, and iodoform sometimes relieve. If the pain is so severe as to prevent sleep, it must be relieved by anodynes. Great care must be exercised in their use that the patient does not become addicted to them. The constant or Faradic current may be cautiously tried; in many cases it is of great benefit.[3] The parts should be moved as much as possible if the joints are not painful. In the so-called acute attacks rest is necessary; and then leeches and blisters to the joints are indicated.

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[1] The only *urinary* change is diminution in the amount of phosphoric acid by nearly fifty per cent.—(*Drachmann.*)

[2] Charcot describes numerous cases complicated by asthma, megrim, cystitis, and such skin diseases as eczema, nummular psoriasis, lichen, and arthritic prurigo; but these complications are the result of the long confinement and inability to excercise, rather than necessary sequelæ.

[3] *Remak* and *Althaus.*

MUSCULAR RHEUMATISM.

(*Myalgia.*)

Muscular rheumatism is a rheumatic affection of the voluntary muscles, the fasciæ, periosteum, and other fibrous structures, accompanied by pain and tenderness, but by *no other* evidences of inflammation. It has been named according to its seat, *torticollis* (*wry-neck*), *cephalalgia*, *pleurodynia*, *lumbago*, etc., etc.[1]

Morbid Anatomy.—The negative results of autopsies lead to the conclusion that there are no constant anatomical lesions, except those due to transient hyperæmia, or to scanty serous exudations into the muscles. In a few cases there is evidence of inflammation of the fibrous sheath of the muscles and of muscle-degeneration. Thickenings and adhesions of the neurilemma of the *nerves* supplying muscles that have long been subject to chronic rheumatism have been found.

Etiology.—Muscular rheumatism is not uncommon in the children of the gouty or rheumatic. It is often intimately associated with articular rheumatism, which sometimes precedes, sometimes follows it. Exposures to cold and damp draughts are often the exciting causes of an attack, especially after the muscles have been over-fatigued. Sudden straining of a muscle may induce it. It often seems to have a malarial origin. It may come on suddenly in a rheumatic or gouty subject without any appreciable exciting cause.

Symptoms.—An attack usually comes on suddenly with severe, deep-seated pain in the group of muscles affected. The pain is of a stretching or tearing character, increased by movement or pressure; it is always more severe at night, and remits or disappears during the day. It may be migrating or remain fixed in certain muscles or fasciæ. It is usually acute when the muscle is in action, and dull when the parts are at rest. Certain positions mitigate the pain. In many instances it will wholly disappear in a few moments, and the sufferer, who perhaps has been for hours enduring excruciating cramp-like pain, feels a sudden sense of relief. Such attacks are followed by lassitude.

Lumbago, *pleurodynia*, and *wry-neck* are the most common forms.

Lumbago, or rheumatism of the muscles on either side of the lumbar spine, usually is the result of straining the lumbar muscles, or sitting on the damp ground, or is excited by a current of air across the back. The patient is unable to bend backward or forward; if the pain comes on while he is in a sitting posture, he is compelled to walk with the body bent at the hips. Lumbago comes on very suddenly, and the pains are more intense than in any other form.

Intercostal rheumatism, or *pleurodynia*, is attended by many of the symptoms of acute pleurisy. There is pain in the side, which is increased by every respiratory movement; the sufferer leans to the affected side. Coughing, sneezing and defecation render the pain more intense.

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[1] These different varieties are grouped by some in the list of the symptoms of *chronic rheumatism*; others regard them as *neuralgias*.

In *wry-neck* the muscles on one side of the nape of the neck are involved. The patient holds his head toward the muscles that are affected, so as to relax them, and, in attempting to turn his head, turns his whole body like a pivot.

If the frontal, occipital, or temporal muscles are involved, it is termed *rheumatic cephalalgia.*

If the abdominal muscles are involved, it is termed *abdominal rheumatism.* In all cases there is *pain* and rigidity of the muscles or groups of muscles involved, accompanied by a fixed position. There is no fever or constitutional symptoms.

Differential Diagnosis.—Lumbago may be mistaken for *renal colic.*

In *renal colic* there is no tenderness on either side of the lumbar spine, which is always present in lumbago. The position of the patient is not fixed as in lumbago. In renal colic the pain radiates along the ureter, to the end of the penis, and is often accompanied by retraction of the testicle on the affected side. The urine is diminished during, and copious and bloody after, the attack.

An examination *per vaginam* will decide between uterine disease and lumbago.

Lumbago may be distinguished from *disease of the spine* by the fact that in the latter affection pressure *on* the ends of the spines will produce pain, while *lateral* pressure gives negative results; in *rheumatism of the muscles* the reverse is the case.

Pleurodynia may be mistaken for *pleurisy* and *intercostal neuralgia.* Pleurisy is accompanied by fever, increased pulse-rate, cough and—on physical examination—by physical signs of pleurisy. *None* of these conditions are present in *pleurodynia.* In *intercostal neuralgia* there are three (diagnostic) points of tenderness: at the exit of the nerve from the spine; at its termination near the sternum; and midway between these points, while there is *no* tenderness over the muscles. In pleurodynia these points of tenderness are absent, and the intercostal muscles are tender.

Abdominal rheumatism may be mistaken for peritonitis; but in *peritonitis* there will be fever, increased pulse-rate, and well-marked constitutional symptoms which are absent in abdominal rheumatism. In the latter, deep, firm pressure affords relief.

Trichinosis is accompanied by symptoms that resemble those of muscular rheumatism; but the history of the case, the œdema of the feet, and a microscopical examination of a portion of the muscle, will decide the diagnosis.

Prognosis.—No danger attends this form of rheumatism. An acute attack may last a few hours or days. If it become chronic, the muscular pains may last for months. Wry-neck is the mildest and lumbago the severest variety. One attack generally favors the occurrence of a second.

Treatment.—In the young, if there is an hereditary tendency to rheumatism, cod-liver oil acts as a prophylactic. At the commencement of an attack a hot-air or Turkish bath will be of service. Guaiacum, sulphur and arsenic are the favorite drugs in chronic cases. Quinine is almost a specific in the malarial form. In vigorous persons subject to muscular rheumatism the surface should be warmly covered with flannel, and the individual should accustom himself to a morning rub-down with a dry, coarse towel after a cold sponging. The bromides are useful in some cases.

In *lumbago* hot anodyne fomentations and anodyne liniments will often give relief if vigorously applied. Hypodermics of morphia may be given for temporary relief. These patients should remain quiet in the position that gives them most relief.

In *intercostal neuralgia*, cupping, blisters, and hot poultices will often relieve. But in severe cases hypodermics of morphia must be resorted to.

In *wry-neck*, the cervical region should be swathed in warm flannel. Gentle traction likewise aids in this. The constant and Faradic currents may be passed alternately through the affected muscle. Showering with water as hot as can be borne is very efficacious in some cases. Acupuncture affords relief in many instances. Veratria and aconite are often used in ointments. Manipulation by a skilled "rubber" is one of the most efficient means of local treatment. If anæmia is present chalybeate waters and tonics are indicated.

GOUT.

Gout is a constitutional disease of mal-nutrition, characterized by an excess of uric acid in the blood and the deposit of urates in the cartilages and fibrous structures of joints and throughout the body. The constitutional condition is generally described as the "gouty diathesis,"—lithæmia; and the term gout is applied only to the phenomena, which may be either acute or chronic, attendant upon the elimination of the urates and their deposition in the joints.[1]

Morbid Anatomy.—The primary changes, so far as we are able to appreciate them, are in the blood and consist in an excess of uric acid.[2] In just what way the nutritive processes are at fault, and whether the urates found in the blood are there as the result of an excess or a deficiency of activity, are points as yet undetermined. In acute gout this excess occurs only just previous to, and during the paroxysm, when the proportion of urates may reach 1 in 20,000 or even 1 in 6,000, and the corpuscular elements remain unchanged, but in chronic gout it is permanent though in much smaller ratio, and is accompanied by anæmia. Traces of oxalic acid are occasionally found in the blood, and there is always decrease of alkalinity and increase of the fibrin-factors.

Although the blood change is the essential one, the articular are the more manifest and characteristic, and consist in the deposit of the urates in the joints with the attendant inflammatory and ulcerative processes. The primary deposit occurs *in* the cartilage capsule and cells at the point of least vitality and most remote from vascular supply, and gradually in subsequent attacks invades the cartilage, periosteum, synovial fringes, and fibrous tissues of and about the joint. where it is found between the connective-tissue fibres. This deposit consists principally of the urate of soda in the form of minute needle-shaped crystals, but to the unaided eye appears amorphous; chloride of sodium, urates of lime, magnesia or ammonia, hippuric acid and phosphate or carbonate of lime are present in small proportion. In the earlier stages the articular surfaces are slightly granular in appearance, with a thin amorphous incrustation. But as the acute attacks are repeated, or in the later stages of chronic gout, the incrustation increases, the surfaces are decidedly roughened; gradually erosions occur from epithelial degeneration and attrition, and all the adjacent structures are infiltrated and covered with a thick deposit of urates. The articular changes may go on to entire destruction of the cartilages and articular surfaces, while the extra-articular deposits form large concretions or *tophi*. These may induce ulcerative and suppurative processes until they finally protrude through the skin, are discharged from abscesses, or produce fibrous and osseous ankylosis. The veins about the joint become dilated, varicosed, and filled with thrombi which may at any time cause embolism. All these changes are gradual in their development, but each attack of gout, even the first, leaves its permanent mark in the affected joint. Aside from the acute attack, inflammatory processes of a low grade are eventually engendered by the irritation of the deposits, and molecular changes enter as an element in the destructive processes, or the inflammation may result in the production of ecchondrosis.

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[1] It has received the names of podagra, chiragra, gonagra and arthritis according as it affects the foot, hand, knee, or several joints.

[2] The precise combination in which uric acid occurs in the blood is undetermined. It is probably either as the acid or neutral urate of soda.

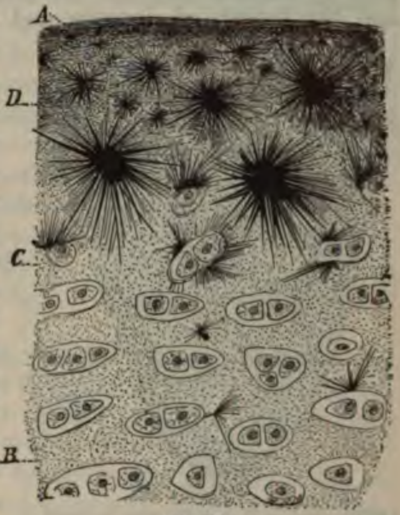

FIG. 185.

Section of a Gouty Cartilage.

A. Articular surface.
B. Normal Cartilage.
C. Crystals of urate of soda in the stroma.
D. Dense deposit of urates. × 300.

The small joints of the body are most frequently affected, and of these the metatarso-phalangeal articulation of the great toe is so unquestionably first as to render it the classical seat of acute gout. In order of frequency after this are the joints of the fingers, knee, elbow, hip, and shoulder. Other joints are but rarely affected. But all the cartilages and fibrous tissues throughout the body as well as those of the joints may be the seat of gouty manifestations. Tophi are frequently found in the cartilages of the ear, nose, or eyelids, and are then of the highest clinical significance.

They may be present also in the larynx, sclerotic, tendons of the hands, and even on the spinal dura or outer coat of the arterial sheaths.

The most important visceral lesions are found in the kidney, and consist in a similar deposit of urates and subsequent cirrhotic changes. The primary deposits here are probably in the tubules and their epithelium, and the interstitial deposits and fibrous changes are secondary. The kidney presents a granular appearance, and on section the fine striæ of urates may be seen throughout the tubular structures. More abundant deposits may aggregate in the pelvis as calculi and incite pyelitis. The heart is eventually hypertrophied and may present signs of fatty degeneration, while the arteries present varying stages of atheroma from the primary fibroid changes to distinct calcareous deposits. Fibroid and cirrhotic changes also occur in the liver and stomach, and finally uric acid is present in both the normal and pathological fluids found in the body.

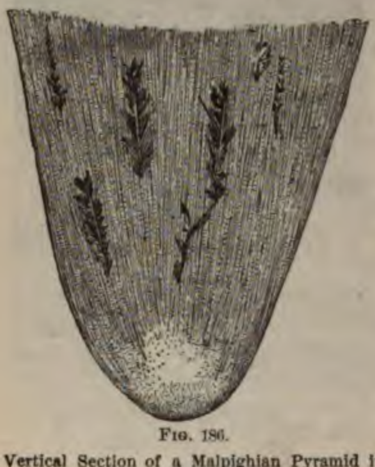

FIG. 186.

Vertical Section of a Malpighian Pyramid in Gouty Nephritis.

The striæ, consisting of urates, follow the general course of the tubules. Much of the deposit has been washed out of the specimen in hardening. × 30.

Etiology.—The exact nature of those changes which result in the gouty diathesis is still undetermined, but certain etiological relations are quite clearly established. Gout is pre-eminently a disease of middle life, and although it can be developed in any constitution, heredity can be traced in fully sixty per cent. of cases, and almost invariably so when it is present in children or early adult life.

Clinical experience proves that the direct exciting cause is the product of the following factors in varying proportions.—*First.* An excess of nitrogenized material in the system from over-eating and the use of alcohol. Excess as applied to eating is a relative term, and in certain constitutions an amount of food, which in others would be very moderate, may so exceed the demands of the system as to be the cause of gout. Of alcoholic beverages the sweet wines and malt liquors are considered more gouty than spirits.

Second. Deficient or suddenly arrested oxidation. Lack of exercise and its consequent abundant supply of oxygen are the most frequent causes of such deficiency of oxidation, but it is also probable that the constitutional and hereditary tendency exerts a powerful influence over the assimilative function, and that impaired nervous energy may be the ultimate cause of defective oxidation.

Third. Failure in the excretive power of the kidney. Such failure may result either from deficiency of the eliminative power of the renal epithelium or mechanically from obstruction of the tubules by deposits of urates, and is the prime factor in producing the acute gouty attack. In a system already laden with urates, a few glasses of wine, a fit of dyspepsia, exposure, severe mental effort, or a fit of anger may be sufficient to bring on an attack of gout.

Symptoms.—Gout may be either acute or chronic, and appear as "regular" in the joints, or as irregular, misplaced, retrocedent or anomalous gout in the non-articular structures and the internal organs.

In most cases of *acute gout* premonitory symptoms precede the first paroxsym ; there will be a history of occasional pains in one or both great toe-joints, a feeling of malaise with sleeplessness, constipation, dyspepsia, and perhaps pain in the side, a dry, hot skin, scaly eruptions and scanty urine. Sometimes an attack is preceded by a peculiar sense of well-being, even excitation ; at others by great anxiety, irritability, and depression of spirits. Asthmatic symptoms often precede the outbreak of an attack. Usually between midnight and four or five in the morning the individual wakes with a burning, throbbing pain in the ball of the great toe, which the slightest pressure greatly intensifies.

The affected joint becomes red, swollen, hot and shining ; the veins are distended, and it resembles a joint about to suppurate. There is some febrile movement which varies in intensity with the number of joints affected. The temperature may in a severe attack reach 105° F. The pulse is full and bounding but compressible. As the fever comes on the pain in the affected joint is so great that the patient cannot move it; he becomes restless, tossing for hours, until finally, in a profuse perspiration, he falls asleep. In a few hours he awakes refreshed and comparatively free from pain ; but the affected joint is swollen, tense, and vividly red. He continues comfortable during the day, but about the same hour the next night there is a recurrence of the local pain and the fever, which is followed by another remission the following morning.

These nocturnal exacerbations and morning remissions continue with about the same severity for two or three days, then the maximum of pain is reached. At the end of a week they have gradually subsided ; the affected joint remains tender and swollen for a week or two longer. This swelling is due to œdema, and pits on pressure, and as it disappears desquamation occurs. Following the attack, there is a feeling of well being which has led to the popular belief that an attack of gout is beneficial.

There is usually marked digestive derangement during an attack, with anorexia, a thickly furred tongue, and constipation. The urine is scanty, high colored, and contains less uric acid than normal, depositing on cooling a copious sediment. The bladder is irritable, and there is a scalding sensation on urination. Intense cramps in the muscles adjacent to the affected joint may occur. Occasionally during the first attack both great toe joints are involved ; and instead of disappearing at the end of a week, successive outbreaks occur in the other joints.

An individual may have only a single attack ; but usually a second supervenes within a year. Gradually the attacks approach each other, and are more prolonged though less severe, until a condition of chronic gout is reached. In the second and third attacks the joint formerly involved. or its fellow of the opposite side, presents the same phenomena as in the primary attack.

Although all gout is strictly speaking *chronic*, by *chronic gout* is understood those gouty manifestations which are developed as the paroxysms coalesce, and the patient is scarcely ever free from some gouty manifestation. Tophi form around the affected parts, and the joints become so distorted or crippled that walking becomes difficult. Gradually the health deteriorates, and feebleness and a gouty cachexia become marked and visceral derangements become prominent. When chronic gout follows acute, the articular phenomena are always prominent. But when, as not infrequently happens, gout is chronic *from the onset*, tophi form early without acute inflammatory symptoms, and the visceral affections are prominent. In chronic gout the urine is greater in amount, lighter in color, of lower specific gravity, and contains less uric acid than normal. In a few cases casts and albumen make their appearance. Misplaced gout—gout that has retroceded from a joint to an internal organ, also called visceral, masked, internal and metastatic gout—may attack any organ and result in a long series of functional disturbances.

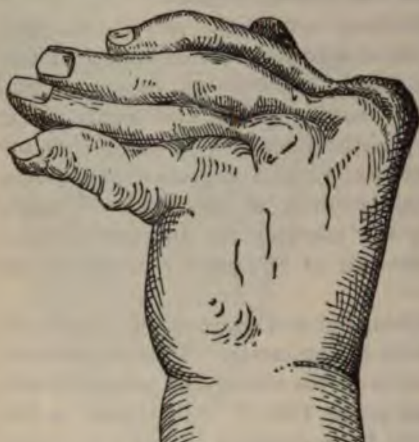

FIG. 187.
Deformity from Gout.

The sequelæ and complications of gout are numerous. Those referable to the nervous system are vertigo, neuralgia, headache, stupor, convulsions, delirium, apoplexy and lunacy.[1] Those referable to the vascular system are arterial degeneration, angina pectoris, cardiac palpitation, and valvular disease. Those referable to the lungs are asthma, which alternates very often with the articular phenomena of gout, and bronchitis, which some regard as the commonest manifestation of gout, *after arthritis*. Referable to the digestive tract is a long list of gastro-intestinal catarrhs, cirrhoses of the liver, jaundice, and cirrhotic kidney.

Differential Diagnosis.—Gout is generally easy of diagnosis. It may, however, be mistaken for *rheumatism*.

Gout attacks the small and *rheumatism* the large joints. A rheumatic attack is of longer duration than a gouty paroxysm, and has no periodicity. In gout the fever is slight, 102° to 103° F., and is in inverse ratio to the number and size of the joints involved ; in rheumatism there is usually a higher range of temperature. Cutaneous affections are common in gout and rare in rheumatism. The heart is frequently involved in acute rheumatism, and rarely in gout. The gouty attack coming on at night in the great toe joint is in marked contrast to the onset of rheumatic fever. Acute articular rheumatism is a disease of early adult life, while gout is rare before thirty-five. In gout there is a history of high living, or an hereditary predisposition; in rheumatism there will be a history of exposure or exhaustion. In gout there is an excess of uric acid in the blood; this is never the case in rheumatism. When we are enabled by the microscope to see uric acid crystals derived from serum of a patient with an arthritis, the diagnosis of *gout* is established. Tophi never form in rheumatism, but are always present late in gout.

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[1] These are regarded by some observers as evidences of cerebral gout.—Garrod, Todd, Trousseau.

The joint affection of *pyæmia* may be mistaken for gout; but the history, in connection with the constitutional signs of pyæmia, will remove all doubt.

Prognosis.—Gout rarely kills, but complete recovery from it is also rare. Death is generally the result of visceral complications or of the cachexia induced by blood changes. The prognosis is less favorable in hereditary gout, and in those who persist in high living and the use of alcoholic beverages, and when the larger articulations are affected. The appearance of albumen in the urine, and total absence of uric acid from the secretions are grave symptoms. The prognosis is exceedingly unfavorable when there is great crippling of the joints accompanied by an extensive cachectic condition. Concerning the doctrine of antagonisms, it has been proved that gout does not exclude cancerous or phthisical developments.

Treatment.—The treatment of gout will be considered under four heads: (1) General hygiene; (2) Dietetics; (3) External, and (4) Internal treatment.

I. Gouty subjects should take systematic exercise in the open air, especially horseback-riding. A country residence is to be preferred to the city; and a warm, dry climate at a moderately high elevation is preferable to a severely cold one. They should always be clad warmly in flannel, and should avoid sudden and violent physical exertion, severe mental strain, and all unnecessary exposure to vicissitudes of temperature. They should retire and rise early, sleeping in a large, well-ventilated apartment without drafts.

II. *Dietetics.*—Simple, nutritious food should be taken at stated intervals and in small quantities. Starving will not cure gout. As vegetables may be taken more freely than animal food, all pastry, eggs, tea and coffee, and alcohol should be avoided, and great care should be taken not to overload the stomach. Game and highly seasoned food, cheese, dried meats, tomatoes, and strawberries are to be avoided. Vegetables that contain the least starch, such as cabbage and the salads, are to be preferred. The principal articles of diet should be beef, mutton, and chicken, bread, milk, and fruits. Some patients, however, bear albuminoids badly, and find relief from their rheumatic symptoms on a farinaceous diet. Alkaline mineral waters, seltzer, vichy, lithia, etc., may be taken with and after meals. When stimulants must be given on account of the enfeebled digestion, light wines, whiskey, or gin will be found least objectionable.

III. *External Treatment.*—The affected limbs should be kept raised above the level of the body during an acute attack, and wrapped in flannel or cotton batting. Cold applications and leeches to the affected joints do harm. When the pain is intense opium may be applied to the joint, or morphine may be injected near it. Tepid alkaline washes and horse-chestnut oil are strongly advocated by many.[1] Vapor and Turkish baths are often of the greatest service and should be taken weekly during the intervals between the paroxysms.

IV. *Internal Treatment.*—Colchicum and the alkalies are our chief remedies during the paroxysm. For thirteen centuries colchicum has been used in this disease; it relieves the symptoms, but *how* we do not know. Its efficacy has been attributed at various times to the elimination of uric acid, its sedative action on the circulation, its purgative and narcotic powers. My rule is to give one of the following pills[2] every three hours until the specific purgative action of the colchicum is obtained; or five drops of the fluid acet. ext. of colchicum in alkaline water may be given every two hours. The maximum dose of the latter should be given at the end of the attack; small doses only are admissible at the commencement. When marked cerebral, circulatory, or gastro-enteric phenomena occur, colchicum is to be discontinued. Carbonate of potash, Rochelle salts, and the urate or citrate of lithia, are important adjuvants to the colchicum treatment.[3] Chloral, sulfonal, opium, and hyoscyamus may be given during the acute attack, to relieve the pain and restlessness of the patient. Common ash leaves, cinchona and gentian, the sulphate of quinine, and the iodide of potassium with tr. guaiac. ammo. have been extensively used in the treatment of gout, not only during the paroxysm, but during the interval. The benefit derived by gouty patients at the different springs which are so highly recommended seem to me to be due to the change of air and scene and to the dietetic restrictions more than to the bathing. A restricted diet, exercise in the open air, and a Turkish bath once or twice a week have succeeded with me as well as a residence at some spring.

In chronic gout, tonics (iron, arsenic, etc.) are usually demanded. The inhalation of oxygen has been advocated as a remedy for the impoverished blood condition. Chemically active remedies—ammonia phosphate and benzoic acid—have not proved as useful clinically as theoretically. It should be remarked that the excessive use of mineral waters is contraindicated in those who are advanced in years, in individuals whose kidneys no longer have the power of elimination, and in those with whom alkalies disagree on account of some peculiar idiosyncrasy.

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[1] Charcot speaks highly of atropine and blisters as topical remedies.

[2] ℞

Pulv. ipecac.	gr. i.
Ext. colchi. acet.	gr. i.
Hydrarg. protochlor. (calomel)	gr. i.
Ext. aloes fl.	gr. i.
Ext. nuc. vomic.	gr. ⅙.

[3] Stricker caused the tophi to disappear by giving 1⅙ grains of lithia carbonate and 3⅙ grains of soda bicarbonate in 16 ounces of carbonic acid water a day.—*Virchow's Archiv.*, vol. xxxv.

LITHÆMIA.

Lithæmia strictly implies an excess of uric acid in the blood—an excess which at a certain point becomes gout. The term is often employed, however, to cover a general diathesis in which disturbances in the retrograde changes produce a persistent increase of uric acid without marked tendency to its accumulation, the renal excretion being proportionately increased. The condition being purely a functional one, it has of itself no morbid anatomy. Its pathology is also somewhat obscure, although it is now generally accepted that the primary defect is in the liver. It is impossible to make any exact distinction between this arrest of the retrograde elements at the uric acid stage and the excessive formation of both urea and uric acid in gout. The former, however, appears to be the condition present in cases of an inherited lithæmic diathesis, while the latter is developed either by excess of consumption of nitrogenized element or lack of oxygenating power. The one is inherited and the other acquired. In the one the lithæmic condition and its consequences are present, despite the most careful dieting; in the other they are avoided or removed by proper diet and exercise. For the lithæmic diathesis we must assume, therefore, an inability on the part of the cell to produce oxidation, even when the necessary elements are furnished. This diathesis is so closely connected with the fibroid diathesis as to make it impossible to say in just what degree the arterial and other scleroses, so invariably present, are the result of the lithæmia.

Whatever the etiological relation, arterial changes are the more frequent and serious accompaniments of the lithæmic state, even in those chronic forms which do not pass into distinct gout. Derangements of the gastro-intestinal functions, cutaneous eruptions, and innumerable evidences of nervous disturbance, as asthma, cardiac palpitation, neuralgias, headache, etc., are also present in all distinct cases.

Etiology.—An inherited defect of constitution is the primary and principal cause in patients who have never been given to excesses in eating or drinking. The acquired form depends upon a decrease in the ratio between the amount of food consumed and available oxygen for its oxidation. This may be induced by excesses in eating and drinking, or by a sedentary life, causing deficient respiration. Indulgence in stimulants and narcotics, sexual excess, or any form of nervous strain, may so affect the vital functions through the nervous system as to develop a latent lithæmic diathesis, or to exaggerate to serious proportions a moderate lithæmic condition.

Symptoms.—The primary disturbances in the lithæmic diathesis are with the gastro-intestinal tract, and include all the forms of acute and chronic indigestion. Acidity of the stomach with gaseous eructations are characteristic symptoms. The evidences of intestinal indigestion are less marked. The flatus, which such patients pass rather abundantly, even when there is neither diarrhœa nor constipation, gives evidence, by its odor, of the condition of the intestinal contents. Hæmorrhoids are usually present. Diarrhœa may alternate with constipation, but the bowels are often perfectly regular. The tongue is often free from coating, but the breath is quite constantly offensive. In extreme conditions the skin becomes dry and scaly, or even covered with a persistent eczematous eruption, the hair is dry and wiry, the nails brittle, and the perspiration strong and offensive. Irregular or weak cardiac action is one of the earliest evidences of the effects upon the nervous system. Palpitation is prominent, but appears to depend upon no fixed cause. Frequent after eating, it also appears without cause, while the patient is quiet or even in bed. It is equally perverse in its continuance and disappearance as in its advent, lasting sometimes five minutes, or, again, several hours. Such hearts are also often easily excited to a rapid but regular action by slight exertion. Respiration is not affected by these turns of cardiac palpitation. The other nervous symptoms include all forms of neuralgias, headache, nausea, dizziness, vertigo, somnolence when erect, and wakefulness when recumbent, with all the mental irritability, anxiety, and depression which can be crowded into a sane man. This melancholia appears to depend more upon the gastro-intestinal condition than the lithæmia, although it is not absent when digestion is apparently perfect. On physical examination the liver may be found congested and slightly enlarged. The urine is high-colored and contains an excess of uric acid in combination, and often an excess of urea and phosphates. Oxalate of lime crystals are frequently abundant. On listening to the heart early, little is learned; but as the condition progresses, evidences of increased arterial tension will be found long before the radial pulse gives any sign of arterial changes.

These patients remain in this condition for years at times, but sooner or later the arterial or renal changes become prominent, and they pass on to chronic gout, contracted kidney, or arterial fibrosis, with their attendant dangers.

Treatment.—The indications for treatment are: first, a reduction of the nitrogenized elements of the food to a minimum; second, the stimulation of oxygenation by a full supply of oxygen and an increase of the circulation; third, an increase in hepatic function; and, fourth, the removal of the excess of uric acid. It is generally stated that such patients should be put upon a meat diet, and all starches and fats withheld, because these latter undergo fermentation in the stomach. When gastric digestion is slow or imperfect, the presence of *both albuminoid and starchy* foods will be followed by fermentation, when either class alone will be fully digested. The general rule, therefore, is to give to such patients *only one class* of foods. If albuminoids are best borne, let them be used alone, and *vice versa*. When gastric digestion is weak, the starches are better borne; but when intestinal digestion is most at fault, the albuminoids are to be preferred. Physiologically a diet free from flesh meat is to be advised, and clinically it will be found to be the better in the larger proportion of cases.

Oxidation is best improved by regular but persistent exercise in the open air. When this is impossible, patients must come as near to it as possible. Muscular exercise is the best stimulant to nutritive changes in the tissues, but passive exercise, cold and salt baths, frictions, etc., are not to be neglected.

As stimulants to hepatic activity, nitric or nitrohydrochloric acid is at times of value. Mercurials, podophyllin, and other cholagogues, may also be employed. Other medication is directed to the indigestion, and has already been considered. This indication will have been largely fulfilled by the active exercise already advised.

For the removal of the excess of uric acid in the blood no remedy has yet been suggested which excels the salts of lithia. The carbonate or citrate should be taken constantly in moderate amounts. They may be made palatable if given in cold carbonic water, or the lithia waters may be used. Large amounts of water not only dissolve the uric acid, but assist the kidneys in its removal, rendering it less irritating.

DIABETES MELLITUS.

The term diabetes mellitus is applied to a constitutional disorder arising from malassimilation, in which the first appreciable change is the presence of sugar in the blood. When the proportion of saccharine matter reaches three parts in one thousand, it appears in the urine, producing the symptom which has given the name to the disease.[1] It has at various times been regarded as a disease of the kidney, alimentary canal, liver, and nervous system, but its exact pathogeny has never been determined.

Physiological experiments and clinical facts, however, tend to show that the abnormal condition may be the result of either of these pathological processes or of their united action :

First.—Excessive activity in the glycogenic function of the liver or in the primary assimilative processes may so overload the blood with sugar as to cause it to appear in the urine. Whether this hyperactivity is the result of active hyperæmia simply, or arises from disturbed nervous supply is uncertain. The *occasional* appearance of diabetes following the use of stimulants, and its very general occurrence as a result of irritation of the vaso-motor areas in the floor of the fourth ventricle, would seem to indicate that hyperæmia, either from local or central irritation, is an important factor, but what nervous influence is primarily at fault in producing this mysteriously perverted functional activity is undetermined.

Second.—The secondary assimilation may fail to dispose of the sugar produced, and the kidneys are again called upon to eliminate it from the blood. Until physiology can speak with more definiteness than to say that sugar is disposed of in the system "either by oxidation or, *as seems* more probable, in other ways,"[2] it is useless to speculate as to what organ or functions are at fault in this form of diabetes.

Morbid Anatomy.—The only characteristic lesion is the presence of sugar in the blood in varying proportions, up to nine or ten parts in a thousand, and secondarily in all the organs, secretions and excretions. It is most abundant in the urine. Glycogen, acetone and kreatin are generally present in the organs and fluids, and the blood contains more fat than normally. The parenchymatous changes in the viscera are principally degenerative, the result of the blood change. The liver is usually hyperæmic, with possibly some fatty degeneration. The lungs are nearly always tuberculous, with points of catarrhal pneumonia, or possibly of gangrene, attended by pleurisy. The heart is soft and flabby, and, like the other muscles, pale and dry. The spleen is hyperæmic, hypertrophied and firm. Aside from hyperæmia, the kidneys often present the usual changes of chronic parenchymatous nephritis (large white kidney), possibly the result of the excessive work thrown upon them. Softening, cirrhosis, and tumors may be present in the brain, and when they involve the fourth ventricle become of interest from an etiological standpoint. Emaciation becomes marked early, and in protracted cases may be extreme. The skin, which is harsh and dry, is generally the seat of furuncles, carbuncles, bed-sores, and gangrene.

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[1] Synonyms : Glycosuria, Glucosuria, Mellituria, Glycohæmia.

[2] Foster's Phys., 25 Am. Ed., 1881, p. 537.

Etiology.—Diabetes is a disease of early adult life, and is met with more frequently in males than in females. In some cases it appears to be hereditary. It is a well-established fact that mechanical irritation of a certain area of the medulla—an area corresponding very closely with the vaso-motor area in the fourth ventricle—invariably produces glycosuria, and clinical facts prove with a great degree of certainty that diabetes is frequently the result of lesions producing similar irritation. Such irritation may result from general shock or concussion, cerebral hemorrhage, softening, cirrhosis, abscess or tumors, also from excessive mental labor, shock, grief, and possibly from the excessive use of cerebral stimulants. Blows upon the epigastrium are included in its list of causes. Pregnancy, impaired digestion, and immoderate use of sugar, new wine, and alcohol have also been named as causes.

Symptoms.—Although diabetes may be acute, and result fatally within two or three weeks from the time the increased flow of urine is first noticed, it usually comes on insidiously. The patient notices that for some time he has been passing more urine than usual, and has been unusually thirsty. While his appetite has been good, and he has taken more food than he is accustomed to, he is losing flesh and strength ; and there is an abnormal dryness of the mouth, throat and skin, with intolerable itching, followed by desquamation. His sleep is disturbed by a frequent desire to empty the bladder.

As the disease advances he becomes listless and debilitated, and there is decrease or abolition of sexual desire ; in women the menses are often suppressed. The tongue is red or coated, and nearly always thicker than normal ; the gums are pale, retracted, and bleed easily, and the teeth become carious. There are nausea, vomiting, and well-marked dyspeptic symptoms, with constipation, and most patients complain of a constant sinking feeling at the epigastrium. In many instances the breath has a heavy, sweet odor, and the taste is perverted. Attacks of profuse diarrhœa, lasting for a day, occur in advanced cases, and may precede an unexpected fatal issue. Headache, often amounting to intense hemicrania, is common. There is derangement of the special senses, especially of sight (soft cataract and amblyopia); dulness of mind, irritability and restlessness, melancholia and hypochondria. The temperature, pulse-rate, and respirations are below the normal.

In some cases the classical course of the disease will be varied from ; there will be little thirst or loss of appetite and no emaciation ; the patient may even gain in flesh.[1] In diabetes from "over-production" the quantity of urine passed is greater than in the other form, the skin affections are more severe and frequent, the patient does not emaciate, but loss of sexual desire comes earlier. In the variety due to defective assimilation, the emaciation, anæmia, loss of strength and flesh, and palpitation, vertigo, and dyspnœa are early and marked signs, while the nervous phenomena are not prominent.

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[1] Frank describes a "diabetes decipiens" in which the amount of urine passed is not above the normal while a large amount of sugar is present. Quincke relates some very interesting cases of diabetes where delirium, stupor and coma have appeared before death.—*Berlin. Klin. Woch.* No. 1, 1880.

The Urine.—Very rarely the amount of urine passed is but little increased; generally, however, it very rapidly rises to twenty, thirty, fifty or more pints in twenty-four hours. The calls to urinate become very frequent both by day and night, and the genitals are inflamed and excoriated. The urine is acid, of a light straw color, with possibly a faint green tint, clear, without sediment, and of a slightly aromatic odor and a sweet taste. The specific gravity varies from 1.030 to 1.070 with an average of 1.040, and the proportion of sugar from a trace to 50 to 100 or more parts in a thousand. In rare cases, a low specific gravity of 1.008 or 1.010 is recorded, but a specific gravity of 1.030, when the quantity of urine passed is normal or increased, should always lead to an examination for sugar. Urea is always present in increased amount, and uric and hippuric acid, kreatinin, sulphuric and phosphoric acids, acetone, alcohol, and albumen are frequently found. In certain cases where the patient continues to fail, although the quantity of sugar lessens, *inosite* appears in the urine, and continues to increase as the sugar lessens. It may amount to two or three hundred grains in twenty-four hours.

In those cases where sugar and the starchy element of the food are the sole sources of the diabetic sugar, diet may reduce the quantity and specific gravity of the urine to the normal and remove all traces of sugar from the urine and blood. In other and more advanced cases animal food is also converted into sugar, and in such conditions dieting can only modify the urinary symptoms. In a third class of cases the tissues of the body also contribute to its formation, and the quantity of sugar eliminated is but little, if at all, affected even by starvation.[1] Urea is excreted in abnormally large quantities by diabetic patients; some claim that there is increased decomposition of albumen into *urea* and *sugar*. Albumen does not necessarily indicate that there are grave kidney changes in diabetic subjects. The amount of urine passed in twenty-four hours varies from fifty to one hundred pints.

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[1] The following are the best tests to determine the presence of sugar in the urine:—

Trommer's Test.—To the suspected liquid add a few drops of a slightly alkaline solution of tartrate of copper. Boil. Sugar precipitates copper as a yellowish red oxide.

Fehling's Test is founded on the fact that glucose has the property of precipitating the red cupreous oxide from an alkaline solution of sulphate of copper.

Dissolve 36.64 grammes cupric sulphate in 200 c. c. distilled water; dissolve 80 grm. sodium hydrate in 600 c. c. water, and add 173 grm. Rochelle salts. Mix the two solutions and add water to make one litre. Keep in small, carefully sealed bottles. The copper in one c. c. is entirely precipitated by 5 milligrammes of grape sugar.

Pour 5 c. c. of the above test-solution into a test tube and heat to boiling; add the suspected urine, *guttatim:* If grape sugar is present the blue changes to green and the red oxide of copper is precipitated. When the blue is discharged the copper is precipitated.—*Draper.*

Warren's Test.—In a test tube containing three drams of urine add two drops of a solution of sulphate of copper and one-half as much liq. potass. as the amount of urine; boil, *and if the urine contains sugar*, the red sub-oxide of copper will be thrown down.

Maumené's Test consists in heating saccharine urine in the presence of bichloride of tin, which causes it to throw down a black-brown "caramel" looking deposit.

Moore's Test consists in boiling liquor potassæ with the suspected urine: if sugar is present a bistre brown appears.

The *Fermentation Test* consists in putting German yeast in a test tube filled with the urine, and standing it inverted, in a warm place: alcohol and carbonic acid are formed and the bubbles of the latter, tested with lime water, give evidence of its character. Non-saccharine urine does not ferment.

Torulæ form during the fermentation as a scum, and, microscopically, are easily recognizable.

The large amount of urine secreted distends the bladder; and the large amount passed, and its saccharinity, cause a constant itching, burning and uneasy sensation at the prepuce, along the urethra, and at the neck of the bladder; in females, it may cause redness, irritation, excoriation, or an eczematous condition of the vulva. Incontinence of urine is especially frequent in diabetic children.

Differential Diagnosis.—Diabetes mellitus may be mistaken for *glycosuria*, for the *atrophic* form of *Bright's* kidney, and for *diabetes insipidus* or polyuria.

Diabetes occurs at all ages, and often from undiscoverable causes; simple *glycosuria* is very common in *the aged*, in the insane, in fits of ague, after sudden excitement, blows on the head, the taking of chloral, etc. In diabetes mellitus the amount of sugar seldom varies much from day to day; while in non-diabetic glycosuria it varies greatly.[1] Polyuria, polyphagia, and polydipsia are *marked* symptoms in diabetic glycosuria. The symptoms referable to the nervous system and skin are prominent in diabetes mellitus; they are absent in simple glycosuria. Volumetric analysis by Fehling's method is easy in diabetes mellitus; while in simple glycosuria it gives obscure results, owing to the presence of kreatinin.

Diabetes mellitus is at once distinguished from *Bright's disease* or *diabetes insipidus* by the presence of sugar in the urine, a condition which does not occur in either of the other diseases.

Prognosis.—Although diabetes is a progressive disease, it has no regular course, and the prognosis depends very largely upon the form which the disease assumes. In that class of cases where diet reduces the amount of sugar, or possibly at first removes it entirely from the urine, the fatal termination may be long delayed, but those cases which appear to depend upon faulty assimilation are more rapidly fatal. Between these extremes the disease may last from a few weeks to ten or twelve years. Pulmonary tuberculosis (twenty-five to thirty per cent.) and uræmia (ten to twenty per cent.) are the most frequent causes of death. Asthenic inflammation with suppuration is frequent in all the tissues, and boils in successive crops, and carbuncles, are frequent complications which influence the prognosis. Pulmonary gangrene may occur with an odorless breath. Cataract, amblyopia, retinitis, and retinal hemorrhage are often present even in cases not attended by albuminuria.

The prognosis is more unfavorable the younger the subject, the less amenable to treatment the case proves to be, and the severer the gastro-intestinal symptoms. It is always bad in those who emaciate rapidly. Death may occur from marasmus, gangrene, dysentery, anæmia, or not infrequently in *diabetic coma*.

Treatment.—In spite of the fact that there are no "specifics" for diabetes, the greater number of cases can for a time be brought under control; and to this end *dieting* is of first importance.

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[1] *Gerin Rozes.*

All saccharine form of food, or any article that can be converted into sugar, should be withheld. Hence, starchy foods, bread, arrow-root, tapioca, sago—such vegetables as potatoes, parsnips, turnips, carrots, beans, and peas are to be absolutely avoided or partaken of sparingly. Salads, greens, acid fruits, all kinds of flesh and fowl, eggs, cheese and butter, unsweetened tea and coffee can be taken. Alcohol in any form is harmful, but should exhaustion demand stimulation a light sherry or claret may be permitted.[1] Koumyss is sometimes given as a substitute for mild stimulants. To allay the intense thirst acidulated drinks, cracked ice, or alkaline waters may be used in as moderate quantities as possible; while water increases the amount of sugar passed, it is not certain that it increases the amount formed, and the patient should use water in moderation rather than attempt distressing self-denial. A *meat diet* is therefore to be enjoined.

In the above bill of fare the patient will not find much that is unpleasant or distasteful, except deprivation of bread. Gluten breads, bran cakes, and biscuits and buns made from almond flour have been devised as substitutes Some patients cannot eat bread thus made, and in such cases, if bread must be taken, it should be well toasted. Moderate exercise must always be advised, and the skin should be kept thoroughly active by means of baths; in the feeble, warm baths, and in the more robust, sea or cold baths. But as pulmonary complications are so common, the body must always be warmly clothed. The success of dietetic measures depends upon the patients rigidly following them.

The drugs which exert a most beneficial influence are the extract of opium, morphia, and codeia. They must be used sparingly, and usually only when the meat diet is given up for a time. Small doses should always be administered at first. When arthritic muscular and neuralgic pains are severe, narcotics are especially beneficial. Cures are reported by high and trustworthy authorities from their use.

On theoretic grounds, *lactic acid* has been proposed as a substitute for sugar. It has been given until arthritic (rheumatic) symptoms have appeared.[2] Alkalies, bicarbonates, acetates, and citrates are highly recommended. And since they are always most beneficial in the form of natural thermal mineral waters at the *springs*, half their benefit may be ascribed to change of air, mode of life, and the surroundings that attend a visit to watering places. Carbolic acid and creosote have been used as antiseptics, and salicylic acid has been proposed as an anti-fermentative; sulphide of calcium is of benefit where there is much suppuration (*e. g.*, boils, carbuncles, etc.). Ergot and jaborandi have apparently been beneficial in some cases; the constant galvanic current has been productive of good results. The anæmia which attends it demands iron, cod-liver oil, strychnia, quinine and a change of air and scene. Surgical operations should on no account be undertaken on diabetic patients.

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[1] Donkin recommends the continuous administration of skimmed milk, three or four quarts a day; when the patients are able to pursue this plan it is followed by good results.

[2] Two Neopolitan professors, Primavera and Caucani, claim that a meat diet with ℈ ij-iv of lactic acid and ℥ ss. of alcohol in 12 oz. of water at a meal, will furnish no materials for the formation of sugar, yet will be a substitute for the saccharine and farinaceous elements of the food.

DIABETES INSIPIDUS.

Diabetes insipidus, or *polyuria*, is characterized by extreme thirst and the secretion of a large quantity of colorless urine, of low specific gravity, free from sugar and albumen. It is also called hyperuresis or *polydipsia*, the latter term having reference solely to the *intense thirst* which attends it.

Morbid Anatomy.—Polyuria has been produced both by mechanical and pathological lesions of the brain just above the floor of the fourth ventricle. Disease of the pineal gland, and cerebral disease extending into the medulla have also been found associated with it.[1]

Etiology.—Diabetes insipidus may occur at any age and in either sex. Some consider its immediate cause to be a dilatation of the capillary vessels of the kidney, which has its origin in a disturbance of the sympathetic ganglia. Blows on the head, injuries to the medulla or region of the fourth ventricle, injuries to the spinal cord, violent emotions, have all apparently caused its development. Drinking large quantities of ice-water when overheated, and exposure to cold and wet are among its supposed causes. Diabetes insipidus temporarily disappears during the course of acute febrile disease.

Symptoms.—This affection may come on insidiously or suddenly. Its chief symptom is the passage of a large quantity of limpid urine; the quantity varies from thirty to sixty pints *per diem*. Its specific gravity ranges from 1.003 to 1.008; it is remarkably *clear*, faintly acid, and of a greenish opalescent hue. Urea, uric acid and kreatin are secreted in larger quantities than normal. It contains no sugar or other abnormal ingredients. Intense thirst accompanies this increased flow of urine; so great is it that patients who have had all fluid withheld from them have drunk with avidity their own urine. The quantity of urine equals the amount of fluid taken. The skin becomes harsh and dry; and the temperature becomes subnormal. The appetite, gastric and intestinal symptoms are all very variable. A strong corroborative proof of its nervous origin is the occasional *increased salivation* that is clearly due to nerve influence.[2] The other symptoms which attend this disease are variable. In some cases the patients are well in all other respects: in others there is vomiting, rapid emaciation, and the general signs of acute phthisis.

Differential Diagnosis.—A careful examination of the urine for one month will distinguish this condition from all other diseased conditions which are attended by the secretion of abnormally large quantities of urine.

Prognosis.—Recovery from diabetes insipidus is rare, although it may last many years without any disturbance of the general health. Pleurisy and acute rheumatism occurring during its course have been followed by complete recovery.[1] In most instances death is caused by intercurrent disease.

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[1] Dickenson found degenerative changes in the solar plexus. The blood is said to contain an abnormally large amount of solid constituents.

[2] See discussion of "*submaxillary gland and chorda tympani nerve*" in Foster's Physiology.

Treatment.—When a cause can be reached, it should be removed. At all times the body should be warmly clothed, and the skin kept active. The food should be highly nutritious and easily digestible. Great attention must be paid to the surroundings and general hygiene of the patient. Narcotics have been advocated, but they are not so efficacious as in diabetes mellitus.[2] Nitrate of potash, iron, alum, lime-water, tannic and gallic acid, creosote, bromide of potassium, acetate of lead, jaborandi and belladonna have all been recommended, either for *narcotic*, *astringent* or *vaso-motorial* action; the chief idea in all being to constringe the renal capillaries. The constant galvanic current passed between the loins and epigastrium is advocated, and deserves more extensive trial.[3]

ANÆMIA.

Simple anæmia is a condition in which the number of the red corpuscles is markedly diminished; when local it is called ischæmia. If the *blood-mass* is diminished it is called *oligæmia*. *Spanæmia* and *hydræmia* are synonyms of anæmia.

Morbid Anatomy.—The density of the blood is diminished. There is diminution in the number and size, and change in the form and color of the red blood discs; one hundred corpuscles may occupy no more space than is normally taken by seventy-five, and the number may fall below one-half the normal. Besides quantitative there are qualitative changes in the blood; the amount of hæmoglobin in the red discs may be diminished twenty-five per cent. When corpuscular abnormalities are due to imperfect *development* and formation, the condition is called *anæmatosis;*—but when they have been perfect and have *subsequently degenerated*, the name *hæmophthisis* is applied.

The heart in one who has died in a state of extreme anæmia is flabby and pale; the blood is of a lighter color than normal and more fluid; if coagula exist they are pale and crumbly. There may be a diminution in the fibrinogen and fibrinoplastin. There is usually a small amount of fluid in the serous cavities. Ecchymoses are common; and minute hemorrhages may be found at various points.

Etiology.—Simple anæmia may be caused by anything that decreases the number of red corpuscles or that interferes with their production.

Acute anæmia is the result of sudden and excessive loss of blood; febrile anæmia is acute.

Chronic anæmia may be the result of numerous small bleedings, or of exhausting discharges other than blood, which attend many forms of chronic diseases. It is a constant accompaniment of many forms of chronic visceral diseases, of which Bright's disease is the best example. Chronic blood poisons cause what is called toxic anæmia. Interference with nutrition, from insufficient or improper quality of food, anti-hygienic surroundings, etc., are prolific causes of simple anæmia. Women are more liable to anæmia than men; and the condition is much more frequent at the two extremes of life, than during the period between twenty and sixty. A tendency to an anæmic condition is not infrequently congenital. Structural changes in the cytogenic tissues, and disease of the lymphatics, induce anæmia. Malignant growths and chronic tuberculosis are attended by conditions of extreme anæmia.

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[1] Dickenson and Desgranges.

[2] Rayer and Trousseau strongly advocate valerian; and Sidney Ringer regards ergot and ergotin as efficacious.

[3] Laycock; *The London Lancet*, vol. ii., No. 7, 1875.

Symptoms.—The symptoms of acute anæmia—such as results from profuse hemorrhages—are extreme pallor, pinched features, and cold sweats; the pulse is feeble, rapid, is quickly accelerated by slight mental excitement or physical exertion; a blowing cardiac murmur and even a "bruit de diable" is present in severe cases. A condition of syncope is of frequent occurrence. Vomiting, delirium, tinnitus aurium, and other nervous phenomena are common. The thirst is intense, and the urinary secretion is scanty.

In *chronic anæmia* there is a pale, waxy, or sallow hue of the skin, and a pale, bloodless condition of the mucous surfaces. The skin becomes œdematous and the muscles flabby. The hands and feet are always cold. A cachectic or marasmic condition is developed; the skin becomes harsh, and often desquamates in patches. As a result of long-continued anæmia a hemorrhagic diathesis may be established. The urine is pale, contains less urea and less pigment than normal. Dropsies are liable to occur when anæmia has persisted for a long time. The temperature is frequently subnormal. Extreme exhaustion and muscular feebleness are among its earliest and most prominent signs. Anæmic patients are irritable, excitable, usually hyperæsthetic and suffer from neuralgias. Anæmic females complain of a pain in the left side and a burning sensation on the top of the head. They are often hysterical. Temporary *aphasia* may result from anæmia. Anorexia and atonic dyspepsia result from deficiency either in quantity or quality of the gastric juice. A morbid, craving appetite sometimes exists.

The constant and important signs of anæmia are hæmic murmurs, which may be *cardiac*, *arterial*, or *venous*. The cardiac murmurs are systolic in rhythm, blowing or bellows-like in character, and have their point of maximum intensity at the base of the heart. Arterial murmurs are heard over the large arteries, and they *may* be accompanied by a thrill perceptible in the radial vessels. Over the jugulars, particularly the right, there is heard a continuous venous hum. A deep inspiration intensifies, while coughing diminishes the intensity of the venous hum. It is also diminished by the horizontal posture. It may sometimes be felt as a thrill on palpation. The heart's impulse is always feeble; the heart sounds are muffled, and the radial pulse is compressible and small. Severe attacks of cardiac palpitation are common.

Differential Diagnosis.—Acute anæmia from either internal or external hemorrhage is not likely to be confounded with any other condition. The diagnosis of chronic anæmia is readily made from the history and general appearance of the patient, and by the presence of hæmic anæmic murmurs.

Prognosis.—The prognosis in anæmia is determined by the conditions under which it occurs. The earlier its cause is discovered, and the more readily removed, the better the prognosis. Its duration varies from days to years; some individuals, especially women, are anæmic during their entire lives. When it is associated with, or dependent upon, organic disease, the prognosis is unfavorable. Death, in *acute* cases, results from annulling the function of the medulla, or cardiac paralysis; in chronic cases, inanition and exhaustion, or some complication, induce the fatal issue. Death may occur in syncope, convulsions and coma.

Treatment.—The treatment of anæmia is always restorative, and must be especially directed to improving the blood-making power. Acute anæmia, the result of profuse hemorrhage from wounds, accidents, during labor, etc., must be treated surgically rather than medicinally. The preventive treatment of chronic anæmia when it depends upon exhausting discharges, prolonged lactation, and anti-hygienic conditions, is the removal of its causes. The diet should be most nutritious, embracing a large proportion of nitrogenous elements. If the digestive organs are feeble, food must be taken in small quantities and at short intervals. Alcohol is food to anæmic patients. Burgundy, Madeira, and rich wines are to be preferred; but in anæmic females the malt liquors are often more beneficial. Daily exercise in the open air and exposure to the direct rays of the sun are essential, and should be taken regularly without producing excessive fatigue. The clothing should be carefully regulated; in winter warm flannels should always be worn, and in the spring and fall great care should be exercised not to allow the surface to become chilled.

Iron is the one drug that best combats anæmia. There are many preparations, but the chloride, Vallet's mass, Blaud's pills, and, in children, reduced iron or the citrate are the forms that have given me the best results; it should be given *after* meals. The combination of iron with quinia, strychnia and phosphorus or arsenic is efficacious in many cases when iron alone fails to improve. Emulsions of cod-liver oil are valuable adjuvants when they can be borne by the stomach. It should not be given after it produces headache or dyspeptic symptoms.[1] Recently I have found malt extracts combined with iron, pepsin and pancreatic preparations efficacious when there is deficiency of stomach digestion. The operation for transfusion of blood or milk in extensive anæmia has never proved successful in my experience, although there is good authority for resorting to it. If the bowels have a tendency to constipation, aloes should be given with vegetable tonics. Travel and a change of climate, often act beneficially when all other means have failed.

CHLOROSIS.

Chlorosis is a special form of anæmia, which occurs almost exclusively in *young females* about the age of puberty, without any assignable cause.

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[1] Goodhart, Fothergill, and others maintain that anæmia predisposes to cardiac dilatation, and hence propose that *digitalis* should be combined with *iron*.

Morbid Anatomy.—The body is well nourished, the organs are abnormally pale, the serous cavities contain fluid, and there is more or less œdema of the lower extremities, the red blood corpuscles are diminished in number, and the hæmoglobin is less in amount than normal. The amount of albumen in the blood-serum is often increased and the mass of the blood is increased.

Virchow states that a constant and characteristic lesion of chlorosis is imperfect development of the *vascular system*. The aorta and arteries are generally smaller in chlorosis than normal, and thin walled; the aorta, throughout its entire extent, may only reach the normal size of the carotids. Fatty degeneration of the tunica intima is very common, and this coat may exhibit spots of superficial erosion. The intima exhibits fatty change in little spots or streaks, not in large connected masses. The middle coat is seldom involved. The heart cavities are usually somewhat dilated, and hypertrophy of their walls is not infrequent. Spots of extravasation and ecchymoses may be found on the mucous surfaces and in the serous cavities. The ovaries and uterus are usually abnormally small.

Etiology.—Chlorosis is regarded by some as a neurosis, the blood changes being secondary to the neurosis. The unaltered state of the cytogenic organs—spleen, lymph glands, and osseous marrow—shows that it is not, strictly speaking, a disease of the hæmatopöetic system.[1] There is always more or less anæmia, and there is nearly always some functional derangement of the sexual organs. All of these causes, however, are not sufficient to account for its development in the majority of cases. In very many instances its cause cannot be reached. It is met with most frequently in young girls.[2] There is a form called amenorrhœal, and another menorrhagic chlorosis. Self-pollution is claimed as an exciting cause of chlorosis.

Symptoms.—With or without derangement of the menstrual function, chlorosis comes on insidiously in precisely the same manner as simple anæmia, with which, in its early stage it is so readily confounded. As it develops, the mental condition changes, the individual becoming morose or despondent. The countenance assumes a peculiar waxy, yellow, or yellow-green pallor. The face is puffy, the eyes are surrounded by deep, blackish circles, the sclerotic is pearly, and the mucous membranes are pale almost to the verge of bloodlessness. The puffy look of the face is soon shared by the rest of the body; but it is not œdematous.[3] Sometimes the cheek will retain a slight degree of color; and on excitement, mental or physical, the face is suffused.

In some cases the onset is sudden, and the above-named signs appear soon after some menstrual derangement. In all cases lassitude, muscular weakness, dyspnœa, and fits of cardiac palpitation are common. Muscular pains follow violent physical exercise. The appetite is capricious; the patient will crave the most indigestible substances, and will eat with avidity chalk, slate pencils, ashes, dirt, or strongly acid and spiced food. Sometimes there is *anorexia,* sometimes *bulimia.* Cardialgia is a common symptom; it is accompanied by a sense of weight over the stomach and by belching large quantities of inodorous gas. It is to be remembered that *gastric ulcer* is frequently met with in chlorotic females. Chlorotic girls are usually melancholy, abstracted and irritable. In some cases they have attacks of nervousness, so that the term *chlorotic hysteria* is used to denote a hysteroid attack in young females. The sexual desires are *diminished* rather than increased. The rapid breathing, dyspnœa, and palpitation are undoubtedly in most cases purely nervous. Dyspnœa is often a very prominent symptom and is accompanied by a short, *dry* cough; the respiratory murmur is feeble, and a full inspiration causes a fit of coughing, which may lead to the suspicion of phthisis. There is rarely fever; when fever occurs there is something more than chlorosis. Often late in the disease, œdema of the feet and ankles occurs.

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[1] Virchow regards the predisposition to it as dependent upon congenital abnormalities of the heart or aorta. Immermann regards chlorosis as due (in part) to *functional* derangement of the cytogenic organs.

[2] Niemeyer says that "obstinate chlorosis attacks all young girls, without exception, in whom the menses have appeared in the twelfth or thirteenth year, and before the development of the breast and pubes."

[3] Immermann states that a tendency to *obesity* exists in chlorosis,

The *urine* is pale and watery. The specific gravity is below normal. Urea and coloring matter are deficient in quantity. Leucorrhœa, and amenorrhœa or menorrhagia are common attendants of chlorosis. The heart is feeble and excitable. A systolic hæmic murmur is present which is heard in the carotids as in anæmia, but the venous hum in the neck, though not infrequent, is still not so common as in anæmia.

Differential Diagnosis.—Chlorosis may be mistaken for *progressive pernicious anæmia,* for simple *anæmia, Bright's* and *cardiac disease.*

In *anæmia* emaciation is marked; in chlorosis there is no loss of flesh. The peculiar greenish color and the mental state are important points in its differental diagnosis. The age, sex, and presence of uterine complications are all important in distinguishing chlorosis from anæmia.

An examination of the *urine* in the one case and a physical exploration of the *chest* in the other, will soon decide as to the existence of *kidney* or *cardiac* disease.

Prognosis.—The course of chlorosis is influenced by the hygienic and social surroundings of the patient, and by the treatment employed. It is not a self-limiting disease, and shows no tendency to spontaneous cure. Its duration is very uncertain. As a rule the prognosis is unfavorable, on account of the liability to serious complications, as phthisis, valvular endocarditis, ulcer of the stomach and thrombotic formations. Rheumatism, septicæmia, typhoid fever, and pneumonia are nearly always of a malignant type and fatal in one who is chlorotic. Hysteria, chorea, paralysis, and epilepsy sometimes complicate, and sometimes are sequelæ of chlorosis.[1] It is to be remembered, in giving a prognosis, that *relapses* are common after intervals of seeming health.

Treatment.—When a cause can be reached, such as self-abuse, masturbation, or disease of the functions of the uterus or its appendages, the removal of such cause will be the first step in the treatment. Chlorotic patients should have an out-of-door life with cheerful companions and surroundings;

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[1] Friedreich and others state that *Basedow's Disease* is unmistakably connected with chlorosis.

they should eat regularly of a diet of which meat and vegetables form the chief part. Late hours and bad air and gaslight should not be allowed. Mental is as important as physical hygiene. The patient should not be idle; something pleasant must always occupy the mind. I have found that iron does not act as well here as in anæmia ; indeed, in many instances it is not well borne.[1] When there is a tendency to fulness about the head it does harm ; some authorities state that only small quantities of iron are absorbed by this class of patients, others say that large doses are of much more service than small.

Arsenic I regard as the most valuable medicinal agent, either alone or combined with iron. Constipation is to be overcome by the careful but persistent use of small doses of aloes and nux vomica. Quinine, calumba and quassia may be given alone, or in combination with ferric preparations. Zinc and the mineral acids will often prove valuable adjuvants to the iron plan of treatment. In cases of long standing the rest treatment combined with massage are followed by good results when all other means have failed.

PROGRESSIVE PERNICIOUS ANÆMIA.

This form of anæmia has received many names ;[2] but progressive pernicious anæmia is the term now most generally accepted.[3] It may be defined as that form of anæmia which occurs without discoverable cause, resists all treatment, and steadily progresses, with greater or less rapidity, to a fatal termination. The degree of oligocythæmia is greater than in simple anæmia.

Morbid Anatomy.—The *blood* is scanty and pale with a specific gravity in some cases as low as 1.030, and shows but slight tendency to coagulate. The number of red corpuscles is greatly diminished and their size and outline are altered. There is no increase in the white corpuscles, but they seem to be in excess, solely from the great loss in the red. It is stated that the amount of hæmoglobin in each red disc is diminished ; and that the white globules contain traces of it. Myelogenic pseudo-leukæmia was the name proposed by those who found that in this disease the adult marrow became fœtal, red and adenoid.[4] But the change in the medullary structure of the bones is a secondary, not a primary change. Large nucleated embryonic corpuscles, lymphoma, and multiple sarcomatous growths have been found in the marrow.[5] Secondary to the anæmia the heart undergoes circumscribed or diffuse fatty degeneration. When this is partial the papillary muscles and inner part of the heart seem to suffer most. In many cases there is more or less dilatation, but no change occurs in the valvular apparatus. The inner coat of the larger arteries and certain capillary tracts also exhibits fatty degeneration.

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[1] Immermann and Niemeyer both regard all else as subservient to *iron*. The former says a couple of boxes of iron pills do more good than anything else. They both regard iron as of more benefit than in any other (allied) malady.

[2] Addison called it *idiopathic* anæmia ; Lebert, *essential* anæmia and *puerperal chlorosis* ; and Ponfick the *anæmia of fatty heart*. Anæmatosis and pseudo-leucocythemia, have also been proposed for it.

[3] Andral, Wilks and Wagner describe it under other names.

[4] Cohnheim, Gardner, Litten, Fede, Pepper and Eichhorst all found the bone-changes prominent in this disease ; and the last named found *microcytes* in the marrow.

[5] See a case described clinically by Ehrlich, and pathologically by Growitz, in *Charité-Annalen*, 1880 Berlin.

The liver, spleen, kidney and stomach are anæmic,—even *bloodless* in severe cases—and their epithelial elements present similar fatty changes. In the kidney multiple sarcomatous formations have been found.[1] The liver and spleen may be enlarged; although some observers maintain that anæmia attended by splenic enlargement, disease or swelling of the lymphatics, or any change in the medulla of the bones is *to be excluded from* the class called *progressive pernicious anæmia.* The body is commonly covered with small—rarely large—spots of ecchymosis, and internal hemorrhages are not uncommon.

Retinal hemorrhage, demonstrable during life, is so common as to have been regarded as affording, when taken in connection with the symptoms of anæmia, almost positive proof of its pernicious character. These hemorrhages are due to fatty degeneration of capillary aneurisms. The serous sacs of the body contain more or less blood-stained fluid, and may also exhibit ecchymotic spots. There is not much emaciation, though the skin, membranes, and internal viscera are much paler than in simple anæmia; and rigor mortis is late and slight.[2]

Etiology.—The essential nature of progressive pernicious anæmia is unknown. It has been suggested that at times an endemic and specific cause may be at work, since the disease has been found to occur with comparative frequency in certain localities.[3] Women suffer more than men, and are liable to it especially during pregnancy.[4] The period between twenty and forty is the age when most cases occur; the disease being rarely seen in early youth or extreme age. When no cause can be found for extreme anæmia, and when no treatment retards its progress, the case is probably one of *progressive pernicious anæmia.*

Symptoms.—It comes on insidiously. Whether the patient has been ill or in perfect health, the course is the same, and it begins with a sense of languor, which increases from day to day. The skin and mucous membranes become very pale and assume a dusky yellow color quite distinct from the green of chlorosis. The muscles are soft and relaxed, and muscular weakness may be so extreme as to force the patient to take to his bed; and yet there is often little or no emaciation. Cardiac palpitation, dyspnœa, and attacks of syncope are the results of the oligocythæmia and attendant exhaustion. More or less œdema and puffiness appear about the legs and ankles, associated with hemorrhages from the nose, gums, bronchi, and female genitals. At the same time the body exhibits ecchymotic and petechial spots and vibices, due to subcutaneous or external hemorrhage.[5] Retinal apoplexy is not uncommon, and an ophthalmoscopic examination should never be omitted in any suspected case. Persistent diarrhœa is often present quite early, and the urine is darker than normal.

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[1] Quincke found an abnormal quantity of iron in the liver, and regarded that as a proof of great destruction of red discs.

[2] Brigidi found that the cœliac ganglia showed fatty and pigmentary degeneration of the nerve cells. Lo Sperimentale. May, 1878.

[3] Cf. *Biermer* in the Zuch Canton.

[4] Gusserow proposes the name "extreme anæmia of pregnant women." Lebert also calls it "puerperal chlorosis."

[5] Immermann says the bleedings are due to the intense oligocythæmia.

There are early signs of atonic dyspepsia; and anorexia, nausea, and vomiting evidence the great irritability of the gastro-intestinal tract. Toward the end there are periods of pyrexia during which the temperature is remittent and may reach 104° F. The rise in temperature is attended by other febrile phenomena, such as thirst, furred tongue, etc., etc. Still later there is a time of absolute apyrexia, and toward the very last moments of life the temperature runs far below normal. Cardiac, venous, and arterial murmurs are present and are far more intense than in simple anæmia. The cardiac systolic murmur is associated with the *fremissement cataire*. The heart is always feeble, and in severe cases intermittent; sometimes it is slightly dilated from fatty degeneration.

Differential Diagnosis.—Progressive pernicious anæmia may be mistaken for *simple anæmia, leucocythæmia, pseudo-leukæmia,* and *chlorosis.*

Ordinary *anæmia* presents a discoverable cause, is attended by *marked* emaciation, has no febrile symptoms and no evidence of retinal hemorrhages or purpuric spots. In progressive pernicious anæmia the case is *just the reverse.* Finally, simple anæmia is amenable to treatment, while pernicious anæmia is not.

In *leucocythæmia* the spleen and lymphatics are enlarged, often enormously; and the bones, especially the sternum, may be tender and somewhat swollen, and the blood will contain an actual as well as relative—1-20 excess of white globules. None of these conditions are present in progressive pernicious anæmia.

In *pseudo-leukæmia* the spleen and lymphatics undergo the same enlargements as in leucocythæmia, but there are no blood lesions, while in pernicious anæmia there is no enlargement of lymph-structures, and the diminution of red discs is greater than in any other disease.

Chlorosis is a disease of young females; it is uncommon except at the time of puberty. Pernicious anæmia is common among women in the puerperal state, and occurs between twenty-five and forty as a rule. Chlorosis is unattended by dropsy, while dropsical symptoms are common in pernicious anæmia. Hemorrhages, spots of extravasation, and febrile phenomena form no part of the natural history of chlorosis; while they are constant symptoms in pernicious anæmia. Chlorosis is curable and does not progress beyond a certain point; pernicious anæmia is incurable and progressive. In chlorosis the face has a yellow-green hue; in progressive pernicious anæmia it is of a dusky yellow.

Prognosis.—Death occurs in 90 per cent. of all cases. Of course those who make *death* a necessary factor in their acceptation of the definition, justly hold that it is a fatal malady. The duration varies from six weeks to six months. Whether it may supervene on a benign anæmia or a chlorosis is uncertain. In very rapid cases hemorrhage and diarrhœa will be excessive. Death may occur from inanition, exhausting hemorrhages or apoplexy.[1]

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[1] Bramwell states that the end is often ushered in by diarrhœa, coma, or delirium.

Treatment.—The treatment is purely symptomatic and supporting. Iron, quinine, strychnia and arsenic, in combination with a highly nutritious, mostly fluid diet, will be found the most available means in such a line of treatment. Change of air and, when the strength allows, sea-bathing are highly recommended. Transfusion of blood has been tried, and has failed.[1] One case was reported as cured by transfusion of two ounces of human blood by the Dieulafoy aspirator.[2] Though the outlook is discouraging from the onset, yet vigorous supporting treatment should be the rule from first to last.

LEUCOCYTHÆMIA.

Leucocythæmia is an abnormal blood condition marked by increase in the number of the colorless corpuscles, and associated with new formation of cytogenic tissue in different parts of the body.[3] It may assume either a splenic or lymphatic form, associated, in some cases, with a myelogenous leukæmia, in which the medulla of the bones is involved. When two of the adenoid structures are simultaneously involved, it is spoken of as medullo-splenic or lymphatico-splenic leucocythæmia.

A *temporary* increase in the colorless corpuscles (leucocytosis) is not infrequent during and immediately after full digestion, in pregnancy and fevers. It is not leucocythæmia.

Morbid Anatomy.—The blood in leucocythæmia is pale, its specific gravity is diminished, and it does not readily coagulate, although the amount of fibrin is said to be increased. These changes are due to a very marked increase in the number of white corpuscles, which sometimes equal the red. In *splenic* leukæmia the white discs are larger and more granular than normal; while in the lymphatic form they are smaller. In some instances corpuscles containing several nuclei preponderate; in others the white discs are found with only one nucleus. Fat is sometimes found in the white discs. The red blood globules are diminished in number, and they lose their normal outline. When leukæmic blood is defibrinated the corpuscles sink and form two distinct layers—an upper *white* layer, and a subjacent *red*. Some state

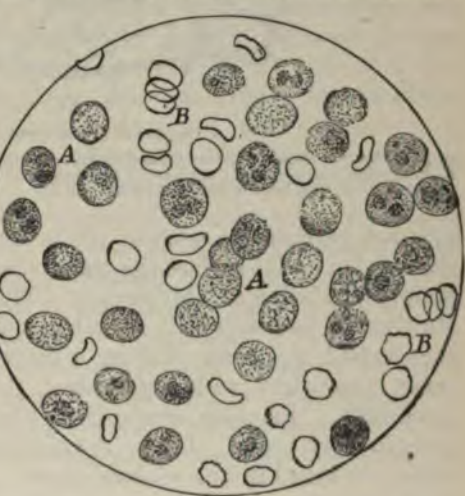

FIG. 188.
Blood from a case of Leucocythæmia.
A, A. White Blood Corpuscles.
B, B. Red Blood Corpuscles. × 300.

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[1] Dr. Weldon states that he has cured four cases by the intra-venous injection of milk.

[2] Dr. Cary, in the *Buff. Med. and Surg. Jour.*, Jan., 1881.

[3] Prof. J. Hughes Bennett, in 1845, described this condition as suppuration of the blood. Virchow, the same year, called it *leukæmia*. Six years later, Prof. Bennett used the name leucocythæmia.

that the reaction of leukæmic blood is *acid*, others that it is *alkaline*. Elongated, octahedral, colorless crystals of albuminoid material are often found in the blood; chemical analyses have resulted in the discovery of multifarious ingredients, as lactic, uric, formic, acetic, and glycerin-phosphoric acids, leucin, tyrosin, etc., etc., some of which are present (normally) in the spleen.[1] It is, however, only when the blood *persistently* shows a proportion of white to red of one to twenty that leukæmia can be diagnosticated.

The *spleen* usually enlarges uniformly in all cases, and it may reach

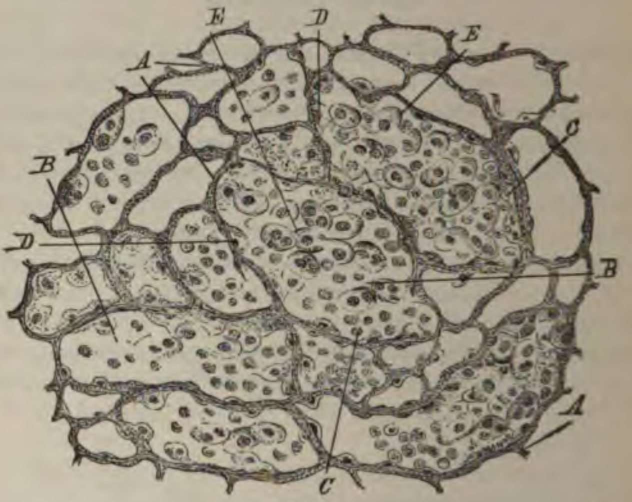

FIG. 189.

Section of a Leucocythæmic Spleen.

A, A. Fibrous trabeculæ of the vascular sinuses—slightly thickened.
B, B. Dilated venous sinuses containing lymphoid cells C, C. swollen and proliferating endothelial
D, D, and large multinucleated cells E, E.

eighteen pounds in weight. It is usually *firmer* than normal; *never* softer. On its cut surface the trabeculæ stand out as whitish striæ, and the Malpighian bodies as whitish dots. These last are lymphadenomata of the Malpighian bodies, which resemble hypertrophies, the tumors having an encephaloid look, and yielding a cancer-like juice.[2] The organ is sometimes "mottled" with *infarctions*, the vessels being plugged by white blood-cells. In these cheese-like islands the vessels are changed into granulo-fatty tracts. Large multinuclear cells and many lymphoid elements are found in the venous sinuses. The *capsule* is somewhat thicker than normal, and inflammatory adhesions may bind it to surrounding parts. In the *hilus* of the organ lymphomata the size of an egg are sometimes found.

Virchow thus sums up the splenic changes: "Hyperplasia of this lymphatic organ induces chemical and morphological changes in the blood." In the lymphatic variety the splenic enlargement is slight, and the principal change is hyperplasia of the lymphatics. The inguinal, axillary, and cervical glands are enlarged and soft, and present a red, pulpy appearance. The meshes of their connective-tissue are crowded with lymphoid elements.

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[1] *Physiology*, M. Foster. "Metabolic Phenomena," etc.

[2] Cornil and Ranvier say that the diagnosis of lymphadenomata (which may affect other glands than the splenic Malpighian bodies) is to be made from cancer, by the fact that the capillaries in *leukæmia are full of white discs which carmine stains.*

In *myelogenic leukæmia* the marrow in the long bones and the spongy tissue of the sternum, ribs, and vertebræ is changed to a creamy-white puruloid mass. In some cases this hyperplasia produces, or is attended by, a red, fleshy appearance, like fœtal marrow. The larger vessels in these cases have their walls infiltrated with lymph-cells, and the small vessels that remain in the changed marrow are chiefly filled with red cells.[1]

Between the liver cells are found circumscribed or diffused patches of white blood cells that have escaped from the overfull capillaries—these are called "apoplexies of the white blood corpuscles"—and in connection with connective-tissue hyperplasia form the whitish masses found in the organ. The liver is enlarged from hyperæmia and from inter-lobular and inter-cellular infiltration.

In the *kidneys* the infiltration of lymphoid elements appears in lines parallel to the tubules. Here also lymphadenomata start from the connective-tissue. Similar new growths are common in the stomach and intestines, often measuring from 1 to 1½ inches in diameter, and presenting many of the characteristics of cancer.

The lungs, brain, skin, testicle, and retina are sometimes the seat of the leukæmic developments. The heart may suffer fatty degeneration, and there may be effusions into the pericardium. Indeed, effusions are frequent in all the serous cavities. Cerebral embolism occurs in about five per cent. of all cases. Hemorrhage from mucous surfaces or into serous sacs is a very common pathological accompaniment of leucocythæmia.

Etiology.—Leucocythæmia occurs at all ages and conditions, but it is most frequent in early adult life. It is twice as frequent in men as in women. In women there is a notable connection between the generative functions and leucocythæmia. Cold, wet, and all anti-hygienic conditions predispose to it. When the Peyerian patches are lymphadenomatous, it is possible that previous intestinal catarrh was the etiological factor. In the majority of instances the etiology cannot be determined.

Symptoms.—The early symptoms of leucocythæmia are almost identical with those of simple *anæmia*, accompanied by swelling of the abdomen with a sense of fulness in the left hypochondrium and wandering pains in the splenic region. The splenic enlargement may or may not be attended by fever.

In the lymphatic variety, enlargement of the glands in the groin, neck, and axillary regions may be the first symptoms which will attract attention, and may exist for months before the blood changes can be detected.

In the myelogenic variety, the bones, especially the ribs and sternum, become tender upon pressure, and as the disease advances the patient becomes pale and assumes a waxy appearance. There will be more or less pyrexia with evening exacerbations, the fever being usually in proportion to the rapidity of the leukæmic development. The pulse is accelerated, feeble, and marked by a peculiar throb. The appetite is capricious, and the tongue and pharynx may be the seat of ulcerations. Early in the disease the bowels are constipated; but its late stages are attended by exhausting diarrhœa. Dyspnœa, arising from the blood condition and from the splenic enlargement, is associated with a chronic bronchitis. As a result of the blood changes, a hemorrhagic tendency is developed, which may cause hemorrhages from any mucous surface, or apoplexy. Retinal hemorrhages and whitish patches due to apoplexy of the white blood corpuscles can be detected by the ophthalmoscope. Ecchymotic spots often appear over the body, and the fatal termination is hastened by this hemorrhagic tendency.

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[1] Neuman regards the myelogenous changes as primary, and states that hyperplasia of the marrow causes immature corpuscles to enter the blood. *Ueber Myelogene Leukæmie*, Berlin, Klin. Woch., 1878 (6–10).

If a hemorrhagic diathesis is not developed the disease runs a tedious course. In such cases the enlargement of the spleen and lymphatics is very great and leads to symptoms of pressure. General anarsarca is of frequent occurrence toward the end of the disease, and under these circumstances the dyspnœa becomes extreme. The urinary symptoms are not important, though the abnormal constituents present the greatest variety. The amount passed diminishes with the progress of the disease.[1] As leukæmic patients emaciate the dyspnœa increases, and the fever, which was at first intermittent, becomes continuous. If no complication occur death results from exhaustion, preceded by delirium, stupor, and coma.

Differential Diagnosis.—Leucocythæmia may be mistaken for *progressive pernicious anæmia*, for *chlorosis*, and in its *early* stages for *inflammatory* and *cancerous enlargements*, and for pseudo-leukæmia, or *Hodgkin's disease*.

In all cases the differential diagnosis rests upon the result of a microscopic examination of the blood. When the ratio between the white and red globules reaches one to twenty the case must be regarded as one of leucocythæmia.

Prognosis.—No case of recovery from leucocythæmia has yet been recorded. It varies, however, in its duration. If it comes on abruptly with active symptoms it may prove fatal in three or four months; but when insidious in its approach three or four *years* may elapse before the fatal result is reached. The average duration of the cases under my observation has been about fourteen months. Sometimes the disease does not progress steadily, but advances by stages, progressing rapidly for a few months, and then remaining stationary for as long a period.

The more rapidly the patient emaciates, the more frequent and profuse the hemorrhages, and the higher the temperature, the more unfavorable the prognosis. Dropsy is always an unfavorable symptom. The most frequent complications are effusions into the serous cavities and hypostasis in the lungs. Pneumonia, pleurisy, and intestinal catarrh stand next; then follow a long list of visceral diseases which appear as coincidences, rather than complications. Death may occur from profuse hemorrhage, from cerebral apoplexy, from pulmonary and serous inflammation, or from exhaustion.

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[1] A number of cases are recorded where priapism, venereal excess, and seminal emissions have marked the onset and course of the disease, but the value of such symptoms is yet to be determined.

Treatment.—None of the many measures proposed and tried have as yet produced a cure or proved successful in arresting its progress. *Quinine* in large doses is, however, advocated by all; it is tonic, and also has a direct influence upon the spleen. Arsenic and iron should also be steadily given, either alone or in combination with quinine. Faradization of the spleen, extirpation of the spleen and transfusion of blood have been tried, the latter *with*, the two former *without*, success. Nitric and nitro-muriatic acid, internally and in the form of baths (locally) are recommended. Iodine preparations do not seem to have any efficacy, and the water-cure plan has been abandoned. The malady is delayed by cheerful surroundings and simple and nutritious diet. Cod-liver oil and phosphorus are valuable adjuvants to the dietetic plan. But the chief indications for treatment are found in the accidental complications.

HODGKIN'S DISEASE.

(*Pseudo-Leukæmia.*)

Pseudo-leukæmia is a disease resembling leucocythæmia in all its anatomical characteristics except the blood changes. It consists in enlargement of the lymphatic structures and development of *lymphomata*; it so invaribly terminates fatally that the term *malignant lymphoma* has been suggested for it.[1]

Morbid Anatomy.—Histologically the lesions of this disease in no wise differ from *lymphomata*. They are divided into *hard* and *soft* tumors. The soft lymphomata are of encephaloid consistency. Their color is a reddish gray, studded with spots of extravasation. The glands undergo simple hyperplasia, and as they enlarge become confluent, forming a large, soft, fluctuating, lobulated tumor. The capsule is in some cases attacked ("periadenitis"), and then the process is not confined to the lymphatics, but extends to, and infiltrates the adjoining tissues. The fluid expressed from a cut surface resembles cancer-juice. Hard tumors have a fibrous feel, and on section exhibit a shining, waxy appearance. The capsule and medullary structure are indistinguishable and no fluid can be expressed. Many regard the extensive hyperplasia of the *soft* as the initial stage of the *hard*; while others consider the greater proportion of cellular elements as the cause of the difference. These tumors are rarely larger than a hen's egg. Unlike scrofulous glandular enlargement they undergo no caseation, suppuration, or retrogressive changes.

As a sequela of this disease the number of red globules in the blood is diminished. The disease first attacks one group of lymphatics and later invades those of the whole body. The glands at the angle of the jaw, in the axilla and groin are usually first affected.

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[1] Hodgkin, whose name the disease bears, was the first to describe these peculiar lymphatic changes (1832). Trousseau called it "Adene." Anæmia lymphatica, Lympho-sarcoma, Progressive glandular hypertrophy, and Lymphadenosis are terms also applied to it.

The *spleen* is enlarged, but rarely to such a degree as in leucocythæmia. On section, it is seen studded with grayish nodules, the Malpighian bodies having undergone hyperplasia. The lymphatic enlargements may be followed by similar developments in the liver, kidney, stomach, lungs, ovaries, testicles, brain, retina, muscles, subcutaneous tissue, and in the serous membranes. These are called metastatic or secondary deposits; and develop especially in and along vascular walls.

Etiology.—Lympho-sarcomata were formerly thought to be carcinomatous or a malignant form of tuberculosis. There is no connection between pseudo-leukæmia and scrofulosis, or between it and syphilitic affections of the lymphatics. The disease has been found in *men* oftener than in women; but no etiological relations have been established.

Symptoms.—When pseudo-leukæmia runs its regular course, and begins by swelling of the glands in the neck, armpit, or groin, it can be recognized without difficulty, and the time of its commencement can be fixed with considerable certainty. But when the enlargement commences in the tonsils, the retroperitoneal, mediastinal, or other deep-seated and impalpable glands, its diagnosis is difficult. In all forms, however, emaciation and anæmia are marked and progressive. In the majority of cases the spleen will be more or less enlarged; and dull pains, with a sense of fulness and distention, will be felt in the left hypochondrium. Attacks of cardiac palpitation are common. The pulse is small and rapid. In the majority of cases there is a steady pyrexia (100° to 101° F.) towards the end, attended by slight evening exacerbations. With the increasing anæmia there will be dropsy, and when the disease is well advanced the muscular weakness is marked. When nausea, vomiting, and diarrhœa are prominent symptoms, lymphadenomata of the stomach or intestines may be suspected. When dyspnœa, cough, impeded venous return, and all the signs of a bronchitis exist, the bronchial or mediastinal glands are probably so enlarged as to diminish the calibre of the bronchial tubes or venous trunks near them. The symptoms of laryngeal paralysis may be induced by a lymphadenoma pressing upon the recurrent laryngeal. Pressure on sensory nerves will cause more or less *pain* in the regions supplied.

The impoverished blood condition is evinced by hemorrhages from the various mucous surfaces and by the appearance of petechiæ over the surface of the body; or in children by the occurrence of convulsions or coma. Jaundice has occurred in a few cases from pressure on the common bile-duct. A large inguinal tumor may exert such pressure on the femoral vein as to cause œdema of the legs.[1]

Differential Diagnosis.—Pseudo-leukæmia may be confounded with *leucocythæmia*; the points of differential diagnosis have been considered in leucocythæmia. It is to be remembered that pseudo-leukæmia has *all* the symptoms of leucocythæmia except blood changes mentioned; and *in*

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[1] A rare case is reported where a lymphadenoma about the Eustachian tube caused deafness. Another is mentioned where paraplegia resulted from tumors pressing on the cord.

addition the cervical, axillary and inguinal glands are early and notably enlarged.

Prognosis.—No case of recovery is reported where the diagnosis has been certain. One year is its average duration; two months and three years are its extremes. As in leukæmia, so in pseudo-leukæmia the course may be rapid and attended by pyrexia and well marked constitutional symptoms; or it may be slow and essentially chronic. Pseudo-leukæmia is frequently complicated by pleurisy, pneumonia, so-called diphtheritic sore throat, nephritis, lardaceous degeneration of the lymphatic glands, and pulmonary tuberculosis. Death may occur from asthenia, exhaustion, anæmia, or from complications.

Treatment.—The general treatment of this disease has been mainly surgical, the theory being that if the glands are excised the disease will be checked. It is true that the removal of the enlarged glands relieves the pressure symptoms, which are often exceedingly painful, but it is of no permanent service. Electrolysis, galvano-puncture, the injection of caustics, iodine, arsenic, and phenic acid have been tried without avail. If excision is practised, it should be when there is little or no anæmia and *no fever*. All forms of topical remedies have been tried, but to no purpose. Internally, *arsenic* seems to have some controlling effect.[1] Iron and cod-liver oil should be given in all cases, and sea-bathing sometimes arrests for a time the progress of the disease.

ADDISON'S DISEASE.

Addison's disease, or melasma suprarenalis, is a cachexia accompanied or caused by degenerative changes in the suprarenal capsules, that resemble tubercular infiltration. Many believe that any of the three conditions, VIZ.: tuberculosis, chronic interstitial inflammation, or fibro-caseous metamorphosis, or the three combined, may be present in this disease. But its real pathogenesis is still in obscurity, for the reason that the physiology of the suprarenal capsules is still unsettled. Histologically these capsules closely resemble a lymphatic structure.[2]

Morbid Anatomy.—In the early stages of this disease the capsules are *enlarged*. In the centre of the medullary portion of the glands are found small, gray, tubercle-like granules. As the development of these masses progresses towards the cortex, those at the centre become fused and suffer caseous metamorphosis. This change may be so uniform that the *whole* capsule becomes a uniform caseous mass. At other times the process, beginning in several foci at the same time, causes the gland to assume a more or less lobulated appearance. The line of demarcation between the cortical and medullary portions of the gland is lost, and either the whole cut section may be of a yellow color, or yellow masses may be entwined by gray tuberculoid fibrous tissue. The gray or yellow color predominates, according as the condition has existed for a *short* or a *long* time. As the process extends to the cortex the centre begins to soften. In some cases the gland is a shell of hard, cheesy material, with diffluent creamy contents. Instead of this liquefaction it is not uncommon to find the centre of the capsule in a state of calcification. In either case no sign of normal gland structure remains.[1] In the stage where gray matter predominates the capsules are enlarged; where yellow material is in excess, especially if associated with chalky, degenerative changes, the gland is hard and *indurated*.

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[1] Warfynge reports improvement in four cases from its use.—*London Medical Record*, March 15, 1881.

[2] Kölliker describes the renal capsules as both vascular glands and as appendages to the nervous system. Brown-Séquard's physiological experiments accord with these views of Kölliker.

In some cases the suprarenal glands may become a mass of cicatricial tissue, the central portion of which is in a state of caseous degeneration. The capsules are usually bound to all the surrounding organs or parts by firm connective-tissue.

The *skin* is discolored. In the lower layers of the *rete Malpighii* there are granules of pigment matter which vary in color from a very light to a dark brown. The coloration is deepest in those parts in which the pigment is normally most abundant. In some instances granular pigment is found in the papillæ and in the cuticular connective-tissue cells. It may be distributed along the line of the nerves and blood-vessels.

The spleen may be enlarged and softened. The adjacent mesenteric and retroperitoneal glands are often tuberculous or in a state of cheesy metamorphosis. Peyer's patches and the solitary glands of the intestine are enlarged, and the testicles and the prostate are in some cases tuberculous. The same changes occur in the liver as in the spleen. Scattered tuberculous nodules are often found in the brain and apices of the lungs. The heart may undergo fatty change and the kidneys are occasionally the seat of parenchymatous degeneration. Connective-tissue hyperplasia is marked about the nerve-sheaths in the sympathetic nerves near the capsules. There is hyperæmia of the nerve trunks and ganglia of the large adjoining plexuses.[2] The blood is anæmic, fibrin is diminished, the red discs are altered in size and form, and do not run together in rouleaux as normal discs do. The white blood globules are increased.

Etiology.—There is an undoubted connection between tuberculosis and *morbus Addisonii*. In over 80 per cent. of reported cases, tuberculosis existed in the capsules or in other organs.[3] The latest and by far the most reasonable theory is that the highly nervous capsule contains *gland-cells* which have a close connection with the vascular and hæmatopōetic system.[4] Buhl thinks that not only is it a blood disease, but that it is infectious. It has been regarded as analogous to leukæmia and pernicious anæmia.

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[1] G. Merkel states that there is cell proliferation attended by the formation of a delicate reticulated structure containing many fusiform lymphoid and giant cells. In opposition to most authorities, Cornil and Ranvier affirm that the process *first* invades the *cortex*, and the medullary portion suffers secondarily. Upon microscopical examination the yellow and gray matter present the same pathological aspects as tubercle.

[2] Cornil and Ranvier say that "the lesions of the nerve centres of the suprarenal capsules and of the great sympathetic in part account for the phenomenon of pigmentation."

[3] Letulle points to its association with tuberculous disease of the spinal column. Wilks and Moxon found *pulmonary* tuberculosis in 80 per cent. of their cases. Riesel claims that there is a paralytic state of the vaso-motor fibres of the sympathetic, and as a consequence the blood is imperfectly and unequally distributed. La France Médicale, No. 40, 1880. Also see Zür Pathologie des morbus Addisonii. *Deut. Arch. für Klin. Med.* 1870, Be. 7.

[4] Henle and Von Brunn

der and enlargement of the prostate gland, comes on very insidiously. The complexion becomes sallow, then of a dingy brown hue. There is progressive emaciation and restlessness, headache and insomnia. Irregular chills occur at infrequent intervals; the temperature is constantly above normal, and the pulse is accelerated. *Vomiting* is persistent. The mucous membranes assume a dry, glazed, shining appearance; the skin becomes dry and harsh; the breath and perspiration are distinctly ammoniacal. But it should be remembered that the amount of perspiration in ammonæmia is probably *less* than in any other disease. When the disease is far advanced convulsions may occur. At this time the complexion may become quite dark, and emaciation is marked. Diarrhœa alternates with constipation.

As a cachexia develops, insomnia and restlessness give place to somnolence, delirium, lethargy, stupor, or coma, and the patient passes into a *typhoid state.* Old men with enlarged prostates pass into a condition of stupor; and then low muttering delirium, rapid, feeble, and irregular pulse and subsultus tendinum terminate in a fatal coma.

The *urinary* symptoms are important. The urine is ammoniacal and strongly alkaline *when passed.* It often contains pus, and there is a deposit of amorphous phosphate of lime with crystals of ammonio-magnesian phosphate. The odor is offensive and pungent.

Differential Diagnosis.—Ammonæmia may be mistaken for *typhoid fever, sub-acute gastritis, pyæmia,* or *septicæmia.* In *gastritis* the urinary symptoms are negative, while in ammonæmia they are diagnostic. Nausea and vomiting are persistent in ammonæmia, and intermittent in gastritis. Catheterization, or a rectal examination, will rarely fail to discover some organic genito-urinary obstructive cause for urinary retention; while a physical exploration in gastritis gives negative results.

In *pyæmia* there will be an initiatory chill, profuse, exhausting, and recurring *sweats,* a high temperature (103° to 104° F.), a sweet, sickly odor to the breath, and the evidence of thrombi, infarctions, and multiple abscesses in some central organ or organs. All these symptoms are *absent* in ammonæmia; and the ammoniacal breath and urine, and the *parched,* dry skin are in direct contrast to the symptoms of pyæmia.

In *septicæmia* the temperature is very high—105° to 107° F. Besides there is no ammoniacal odor to the breath or urine; the skin is not dry, and genito-urinary obstructive conditions will not be found. The history of the case will also greatly aid in making a diagnosis.

Prognosis.—The prognosis in ammonæmia is determined to a great extent by the condition which causes it; when it is possible to remove the cause the prognosis is good, but when the cause cannot be removed the prognosis is very bad. In all cases there is slow but progressive impairment of the general health. Aged patients rapidly become exhausted, and sink into a typhoid state if the causative condition cannot be speedily removed.

Treatment.—The most important thing to be accomplished in the treatment of this condition is the removal of its cause. And, since atony of the bladder and enlarged prostate are its most common causes, the first years. Death may occur from asthenia, diarrhœa, convulsions or coma, or from complications.

Treatment.—All remedial measures have thus far proved unavailing.[1] Faradization and galvanism of the sympathetic have been proposed. Quinine combined with iron, and iodide of potassium have been strongly recommended. In all cases perfect rest, quiet and good hygienic surroundings are important. It must be remembered that sudden and unexpected death at any period of the disease may occur when treatment is apparently arresting its progress. I have never obtained any positive beneficial results from any plan of treatment.

AMMONÆMIA.

Ammonæmia is a condition in which an excess of carbonate of ammonia is found in the blood, the result of the decomposition of urine retained in the urinary tract.

Morbid Anatomy.—The intestines are the seat of chronic catarrh and contain a greenish yellow alkaline fluid. Sometimes ulcers are found in the intestines similar to dysenteric ulcers. When ammonæmia occurs *with anæmia*, it is possible for the urea excreted by the intestines to change into carbonate of ammonia.[2] A *ferment* has been obtained from cystitic urine that is said to be capable of changing urea into carbonate of ammonia.[3]

Etiology.—The one essential condition necessary for the development of ammonæmia is *the retention of urine* in the body sufficiently long for the urea to undergo decomposition. The conditions under which it most frequently occurs are, stricture of the urethra, enlargement of the prostate gland, atony and paralysis of the bladder, pyelitis, pyonephrosis, hydronephrosis, sacculated kidney and chronic cystitis.

Symptoms.—There are, *clinically*, two forms of ammonæmia; in one the conditions which give rise to it come on suddenly; in the other the causative condition comes on slowly but continuously.

In the *first*, or, as it is called, *acute ammonæmia*, there are nausea and *vomiting*, intermittent and irregular chills, acceleration of the pulse, and rapid rise of temperature. Diarrhœa and vomiting, if excessive, lead to exhaustion and a typhoid condition. The complexion assumes a dingy bronzed hue; there is great muscular weakness with a tendency to lethargy. Œdema of the face and ankles is of very rare occurrence. Delirium may occur. The tongue is dry, brown and shining—the beefy tongue—and the mucous membranes that are exposed to the air, that of the throat especially, are remarkably dry. The breath and the perspiration are ammoniacal.

Chronic ammonæmia which accompanies atony and paralysis of the bladder and enlargement of the prostate gland, comes on very insidiously. The complexion becomes sallow, then of a dingy brown hue. There is progressive emaciation and restlessness, headache and insomnia. Irregular chills occur at infrequent intervals; the temperature is constantly above normal, and the pulse is accelerated. *Vomiting* is persistent. The mucous membranes assume a dry, glazed, shining appearance; the skin becomes dry and harsh; the breath and perspiration are distinctly ammoniacal. But it should be remembered that the amount of perspiration in ammonæmia is probably *less* than in any other disease. When the disease is far advanced convulsions may occur. At this time the complexion may become quite dark, and emaciation is marked. Diarrhœa alternates with constipation.

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[1] Greenhow asserts that glycerine ʒ ij. combined with spts. chloroform. et tr. ferri chloridi, āā ♏xv., is highly advantageous.

[2] Rosenstein denies any connection between ammonæmia and carbonate of ammonia; but the weight opinion is in favor of it. Rosenstein, "*ueber Ammonæmia*," Deutsche Zeitschrift f. pr. Med. 20: 1874.

[3] Pasteur only succeeded in finding a ferment when bacteria were present; but those who follow P teur's teachings claim that a specific ferment is the cause of ammonæmia.

As a cachexia develops, insomnia and restlessness give place to somnolence, delirium, lethargy, stupor, or coma, and the patient passes into a *typhoid state.* Old men with enlarged prostates pass into a condition of stupor; and then low muttering delirium, rapid, feeble, and irregular pulse and subsultus tendinum terminate in a fatal coma.

The *urinary* symptoms are important. The urine is ammoniacal and strongly alkaline *when passed.* It often contains pus, and there is a deposit of amorphous phosphate of lime with crystals of ammonio-magnesian phosphate. The odor is offensive and pungent.

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Prognosis.—The prognosis in ammonæmia is determined to a great extent by the condition which causes it; when it is possible to remove the cause the prognosis is good, but when the cause cannot be removed the prognosis is very bad. In all cases there is slow but progressive impairment of the general health. Aged patients rapidly become exhausted, and sink into a typhoid state if the causative condition cannot be speedily removed.

Treatment.—The most important thing to be accomplished in the treatment of this condition is the removal of its cause. And, since atony of the bladder and enlarged prostate are its most common causes, the first step is to empty and wash out the bladder. Very often when the patient seems to be sinking into a typhoid state, when there has been nothing to direct attention to the bladder, on the introduction of a catheter a large quantity of stinking urine will be evacuated; and after having thoroughly washed out the bladder, rapid improvement takes place, and the gastric symptoms subside. The diet should always be supporting and stimulating. If tepid water is used to wash out the bladder, carbolic acid, bicarbonate of soda, borax, or glycerine may be added; and the washing should be continued till the withdrawn fluid is perfectly clear and free from odor. Iron and vegetable tonics are indicated.

HÆMOPHILIA.

The *hemorrhagic diathesis* is an hereditary disease marked by a tendency to immoderate bleedings—spontaneous or traumatic—and to obstinate swelling of the joints.

Morbid Anatomy.—No changes in the blood or vessels have been found in the few post-mortems that have been made. The swellings of the joints are probably due to blood extravasations within the articulation.

Etiology.—Its most marked cause is hereditary predisposition. It attacks men far oftener than women.[1] The tendency descends to sons through the mother, who herself may give no evidence of the disease. Fathers do not transmit the tendency to their sons. Pregnancy is said to be a developing cause. There is often nothing to indicate the existence of this diathesis prior to the occurrence of the hemorrhage. Whether the *hæmophilia of the new-born* is a distinct disease is an unsettled question. Their blood has been found to contain fungoid organisms which afford an apparent explanation of the hemorrhages. The hemorrhagic diathesis has been regarded as allied to scrofula, chlorosis, gout, and similar dyscrasiæ, and again as a pronounced manifestation of a rheumatic diathesis, as both these conditions occur under the same influences. One of the most recent theories is that it is dependent on deficient capillary innervation with resulting dilatation. This neurotic theory is favored by the frequency of nervous disorders in bleeders and the fact that neurotic remedies exert the greatest control over it.[2] In its transmission from parent to offspring it often skips one generation.

Symptoms.—The symptoms appear as a rule during the first or second year of life. There is nothing about the appearance that indicates the existence of any diathesis. The disease remains latent until a cut, a fall on the nose, or the pulling of a tooth starts a hemorrhage, which at times is uncontrollable. The blood will ooze for days, and death from acute anæmia may result. Usually the bleeding slowly ceases and after a long time the patient recovers. The bleedings may come on spontaneously. Then there may be *prodromata:* signs of plethora, of cerebral congestion, stupor, cardiac palpitation, and painful swellings of the joints. In childhood bleeding from the nose is the commonest form of hemorrhage. The joints affected are the larger; the knee is most frequently attacked.

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[1] Legg (in *Quain's Dictionary*, p. 569) gives the proportion as high as 11 to 1.

[2] Mosler states that over fifty per cent. of his cases of leukæmia were complicated by it, and in one hundred and fifty cases collected by Gowers, eighty were bleeders.

In bleeders a slight bruise will be followed by extensive blood extravasations into the connective-tissue. Hemorrhages may take place into the stomach, intestines, lungs, bronchi, kidney and brain. Extensive blood tumors may form in any part of the body.

Differential Diagnosis.—There is no condition which is liable to be mistaken for hæmophilia, if the history of the patient is accurately taken.

Prognosis.—Complete recovery is rare, but life may be prolonged by judicious management; an example of which can be cited in the case of Prince Leopold, who was thirty-two when he died of hæmophilia.

Treatment.—For the traumatic forms styptics and mechanical surgical measures should be promptly made use of. When hemorrhage arises spontaneously little can be done. The diet of bleeders should consist largely of animal food. Chalybeate tonics should be constantly administered, and the patient should lead a quiet life in a warm climate. Niemeyer recommends cathartic doses of Glauber's salts and ergot. Harkin, of Belfast, recommends the chlorate of potash,—one ounce of the saturated solution three times a-day—combined with the muriated tincture of iron, and claims to have had excellent results. He states that this plan will eradicate the constitutional tendency.

SCURVY.

Scurvy or *scorbutus* is a chronic blood disease, which may be regarded as a peculiar form of anæmia arising from deficiency of vegetable diet. Until recently it prevailed very extensively in armies and among crews of sailing vessels. Improved means for the preservation of supplies have rendered it of much less frequent occurrence, and greatly mitigated its severity even during long campaigns, and at the present time it is seen but infrequently among sailors.

Morbid Anatomy.—The red blood corpuscles are diminished and the albumen and fibrin-factors are increased, although the albumen does not coagulate readily, and there is a peculiar viscidity to the blood. There is said to be a deficiency of potash salts.[1] The capillaries have been found choked with red corpuscles and their endothelial cells altered. Some describe the blood as thicker, others as thinner than normal—at one time lighter, at another darker. Ecchymoses are very characteristic of scurvy, and occur in and beneath the skin, in the muscles, between the periosteum and the bone, and within the joints. In these situations they may be very extensive. They are also found on all the mucous and serous membranes and may partially fill the pleura or pericardium.[2]

The heart, kidney, and liver often undergo fatty or parenchymatous degeneration. The spleen is enlarged, softened, and exceedingly friable. Ulcers occasionally form on the mucous surface of the large intestine, resembling those of dysentery. The changes in the gums are even more characteristic lesions. In nearly all cases they become soft, spongy, and œdematous, and ulcerated masses overhang the teeth and bleed upon the slightest provocation.

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[1] Cornil and Ranvier.

[2] Dr. Ralfe regards disproportion between the various acids and bases of the blood as the cause of the disorganization of the blood corpuscles and subsequent mucous ecchymoses.

The bodies of those who have died of scurvy are emaciated, the skin is ashen gray, and there is more or less œdema, especially of the lower extremities.

Etiology.—Deprivation of fresh vegetable food for a long time will very surely induce scurvy, independent of climate, latitude, race, or sex.[1] It is rarely met with from any other cause, although an unvaried diet of poor quality may induce it. Sudden atmospheric changes, mental disturbance, severe and prolonged physical labor with insufficient food, and bad hygienic surroundings may predispose to scurvy, but seldom develop it so long as fresh vegetables in moderate amounts are eaten.

The theory that scurvy is due to a specific infection, while improbable, cannot absolutely be rejected.[2]

Symptoms.—The earliest noticeable changes are in the skin of the face and eyelids, which changes color and appears bruised and swollen. The pulse is soft and the temperature lower than normal. The patient rapidly becomes less and less capable of mental or physical labor, the face grows pale and bloated, there is great despondency and a sense of weight in the lower limbs. The skin is dry, rough, and of a muddy pallor; later it becomes sallow and leaden. The conjunctivæ are pearly white, the tongue is clean and pale, the teeth loosen and are surrounded by bright red ulcerated or fungous-looking gums that present a purple line where they join the teeth, and contrast strongly with the pale or livid lips. The breath is exceedingly offensive, frequently from necrosis of the jaws. The eyes are sunken and surrounded by a dark blue circle.

Ecchymoses and petechial spots cover the body and extend over a large surface on the slightest blow or injury. Severe darting pains which simulate rheumatism are felt in the limbs, about the calf of the leg and the popliteal space. The legs may become fixed, owing to the hardness of the muscles of the calf and thigh. Node-like swellings occur over the tibia from subperiosteal ecchymoses. The pulse is slow except upon excitement, when palpitation and dyspnœa are also marked. Slight exertion may occasion syncope in those in whom the disease is advanced. Anæmic murmurs are heard upon auscultation. The bowels are constipated, unless there be scorbutic dysentery. The urine is high colored, sometimes albuminous, and there is a diminution in its normal ingredients except potash salts and phosphoric acid. The chlorides are abundant. Insomnia and disordered vision are common.

Differential Diagnosis.—The history of a case and a close inspection of the gums will enable one to distinguish scurvy from *mercurial poisoning.*

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[1] In the Crimean war more died from scurvy than from any other cause. It was the cause of death in a large proportion of those who died during the potato famine in Ireland.

[2] Fabre regards scorbutus as a miasmatic affection which especially affects the nervous system.

Scurvy is distinguished from *purpura* by the spongy gums, painful swellings, and more profuse though less numerous hemorrhages. Purpura frequently occurs in those whose health has not been impaired by faulty nutrition; scurvy very rarely Purpura is not affected by lime juice or change in diet, while either will at once produce marked improvement in scurvy. Purpura occurs in isolated cases; the vital powers are not as depressed as in scurvy, and muscular swellings are absent.

Prognosis.—Scurvy is not a fatal disease; appropriate treatment in uncomplicated cases always effects a cure. It may be complicated by dysentery, syphilis, the various forms of malaria, typhus, typhoid, and chronic alcoholismus. The former diseases assume a scorbutic character. Death may occur from complications, exhaustion, general dropsy, hemorrhage, diarrhœa, dysentery, pleurisy, pericarditis, or pulmonary œdema. It is said that meningeal hemorrhage is sometimes a cause of death. Hemeralopia often occurs as a sequela.

Treatment.—In long voyages or campaigns lemon or lime juice or citric acid should be taken daily when fresh or preserved vegetables cannot be obtained. By their use in the English navy scurvy has been diminished nearly ninety per cent. One who is seriously ill of scurvy should be kept in bed, and the diet at once be made to consist largely of fresh vegetables and acid fruits with fresh meats in such proportion as the patient can easily masticate and digest. Mustard, radishes, cabbage, and water-cresses are anti-scorbutics. Three or four ounces of lime or lemon juice, largely diluted with cold water, should be taken daily. If stimulants are required malt liquors are to be preferred. A wash of chlorate of potash will afford relief to the oral symptoms, and potash may be given internally; quinine, iron and strychnia act both as tonics and appetizers.

PURPURA.

Purpura is a general disease, characterized by sanguineous effusions into the upper layers of the cutis and beneath the epidermis.[1]

Morbid Anatomy.—Either from changes in the walls of the vessels or in the blood itself (excess of salts, or water, etc.), or quite probably from both combined, extravasations occur into the connective-tissue spaces of the rete mucosum and papillary layer of the cutis or in the spaces between the ducts and hair follicles. The serum is soon absorbed and the more solid elements may gradually undergo complete absorption or result in permanent pigmentation of the parts. Similar lesions are found in the mucous membranes, attended by hemorrhage from the free surfaces. Such hemorrhages are more common in the nares and along the alimentary canal. Serous membranes are less frequently affected, but extravasations have been found in the pleural, pericardial and peritoneal cavities and in the meshes of the pia mater.

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[1] It may be *simple*, *rheumatic*, *hemorrhagic* or *symptomatic*. Purpura hemorrhagica is also called "*Morbus Maculosus Werlhofii.*"

Rarely are the muscles, periosteum, bones, conjunctiva, and retina the seat of extensive blood effusions.

Etiology.—Age appears to have no bearing upon the development of purpura, but it is found more frequently in women than in men. It appears in some cases without any discoverable cause in the healthy and robust; sometimes its causes seem almost identical with those of scurvy. Rheumatic purpura may complicate acute polyarticular rheumatism or occur in those of a rheumatic diathesis. Purpuric spots are not infrequent with valvular disease of the heart, Bright's disease, phthisis, cirrhosis of the liver, and various forms of malarial fever.

Its occurrence with leucocythæmia is interesting on account of Penzoldt's discovery of the peculiar form of the blood discs in Werlhof's disease.[1] Purpuric spots have followed large doses of chloral and iodide of potash. Distinct exciting causes, if such exist, are obscure; fright, severe coughing fits, and epileptic attacks are said to have induced it. There is no doubt but that the enfeebled condition of the vessels often depends upon a state of general debility either hereditary or acquired.

Embolism and thrombosis have been suggested as causes, while disordered vaso-motor innervation, which might possibly account for its occurrence after exhausting diseases, has also been considered the primary lesion.[2]

Symptoms.—In many cases for days or weeks before the eruption occurs there will be a general feeling of malaise accompanied by digestive derangement. In all varieties of purpura the eruption has the same general characters. The spots appear upon the extremities and trunk as a rule, but in severe cases they cover the head and face as well. They vary in color from a bright red to a livid or purple; they are round or irregular with serrated edges, and vary in size from a pin's head to a large pea, or a spot may measure an inch or more in circumference. They do not disappear upon pressure. The smaller extravasations are spoken of as petechiæ, and the larger as ecchymoses, and when they occur in lines or stripes they are called vibices.

If the hemorrhage is so extensive or of such a form as to cause the spots to be elevated above the level of the skin the disease receives the name *purpura papulosa*, or *lichen lividus* when they are conical and located around a hair follicle. The elevated wheal-like nodules are designated as *purpura urticans*, and if they form bullæ containing serum and blood the name *purpura bullosa* is given.

While the primary spots are undergoing absorption, as indicated by the gradual change of color from the dark blue through the green to yellow, another livid red crop is appearing. In ordinary cases a crop lasts from a week to ten days. Desquamation never follows, and once formed a spot does not increase in size, except by fresh hemorrhage in its vicinity.

Sometimes there are no constitutional symptoms whatever in *purpura simplex*; but in *purpura rheumatica* slight fever and rheumatic pains in the knees and ankles are accompanied by red and swollen joints, gastric and intestinal disturbances, colicky pains, etc., in addition to the usual eruption. In purpura hemorrhagica, preceding and accompanying the eruption, there is great constitutional disturbance; the spots are large and numerous, and invade the whole body; there are free hemorrhages from all the mucous tracts and from the lungs. So extensive may these hemorrhages be that acute anæmia is rapidly followed by typhoid symptoms and death. The amount of hemorrhage does not depend upon the extent of the eruption. Cerebral symptoms may occur from ventricular or meningeal hemorrhage.

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[1] *Blutbefund bei der Werlhof'schen Krankheit*, 1878, Erlanger.

[2] Cavalie reports a case associated with organic disease of the brain.

When purpuric spots accompany the exanthems and contagious fevers the usual symptoms of those diseases and the eruption are purely symptomatic of the extensive degenerative changes engendered by the primary infection.

Differential Diagnosis.—The points of diagnosis between purpura and scurvy have already been given. The fact that there is no itching, no desquamation, no suppuration or discharge, and no change in purpuric spots upon pressure suffices to distinguish them from the eruption of any form of *skin disease.*

Prognosis.—In uncomplicated purpura the prognosis is good; but when venous thrombosis, scurvy, diarrhœa, or an incurable organic disease exists, life is endangered by the liability to hemorrhage from mucous surfaces, and the occurrence of extravasations into the serous cavities or brain. Anæmia and dropsy are often causes of death.

Treatment.—At one time the treatment consisted in administering quinine and sulphuric acid. At the present day rest, a highly nutritious concentrated diet, and moderate stimulation with a nutritive wine are the principal measures employed. Tinctura ferri perchloridi,—15 to 20 minims three times a day,—is very efficacious, and should be given in connection with some one of the mineral acids, preferably sulphuric. Ergot, turpentine, gallic acid, and other hæmostatics are all highly recommended when the hemorrhages become dangerous. When hemorrhage from the lungs occurs, the treatment is the same as in other forms of bronchial hemorrhage. Recently, small doses of mercury have been given, and apparently effected a cure. Shand has obtained excellent results from Faradization (*Lond. Lancet*, July 9, 1879).

MYXŒDEMA.

Myxœdema is a name given by Prof. Ord to a progressive disease where the tissues of the body are invaded by a jelly-like, mucus-yielding dropsy.

Morbid Anatomy.—All over the body the connective-tissue is found abnormally abundant, the fibrillar element being especially increased and unusually well defined. In Prof. Ord's cases the corpuscular elements were enlarged and multiplied, and the interstitial element greatly augmented. The skin in myxœdema yields many hundred times as much mucin as normal or ordinarily dropsical skin. Besides the overgrowth of connective-tissue, it seems to have undergone a retrograde degeneration. In the skin the mucous infiltration causes swelling, translucency and defective secretion. The connective-tissue stroma of the mucous membranes, of the outer coat of the arteries, the glands, muscles, and the central nervous ganglia is similarly infiltrated and degenerated.

Prof. Ord states that the structure of the thyroid glands may be entirely destroyed by the material; and he thinks its inroads on the Malpighian bodies and tubules of the kidney cause the albuminuria which occurs late in the disease. Dr. Mahomed, on the other hand, argues strongly in favor of the identity of myxœdema and Bright's disease. Recently it has been almost conclusively proven that the central nervous system is affected, and in two cases marked bulbar paralysis has been found.

Etiology.—Myxœdema, in the few cases first described, only occurred in adult females, and of these more married than single women were affected. Recently, however, Dr. Andrew Clark states that in his experience males suffer oftener than females, in the proportion of seven to three. The number of recorded cases is, however, too small to admit of positive statements.[1]

Symptoms.—The face is swollen as in real dropsy; but the skin has a waxy anæmic look, and the œdema involves not only the dependent portions, but *every feature of the face.* Both lips are equally enlarged; the nose is thickened, and the rounded cheeks have a pinkish hue, contrasting peculiarly with the rest of the waxy white skin. There is no pitting on pressure; on the contrary, the skin is rather elastic. The shape and form of the hands is lost.[2] The dry, rough, translucent skin seldom or never perspires. The thyroid body disappears or diminishes, while there is elastic tumefaction of the skin in the lower triangle of the neck above the clavicle. The expression of the face is stolid and sad; the speech is monotonous, slow, and leathery; the limbs move slowly and lazily; a fixed attitude cannot be maintained, and consequently the patient is apt to suddenly fall. The intellect becomes dull, sensation is slow but finally sure, and the muscles are so relaxed at rest that a long contraction occurs before a proper equilibrium can be maintained; hence a quiver often runs through the body as one foot is raised from the ground and the body is balanced on the other. The muscles of the neck are so lax that the head droops on the chest. Sometimes the patella is fractured by the forward bending of the body. There is no real loss of muscular power, no wasting, and no loss of sensation.

Thoughts and expressions are tardy and deliberate, but correct. The bodily temperature ranges between 98° and 94° F. These patients are constantly chilly. Late in the disease patients grow morose and irritable, and are subject to delusions, hallucinations, loss of memory, and finally complete mental failure.

Differential Diagnosis.—There is no disease with which myxœdema can be confounded when the mucoid œdema is well marked.

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[1] In the sixteen cases on which Ord based his descriptions, pregnancy in one or two cases preceded the myxœdema.

[2] Sir William Gull calls them spade-like.

Prognosis.—This is always unfavorable; its duration varies from six to eight years. Death may occur from coma, uræmia, or inanition.

Treatment.—Besides warm clothing, tonics, and good food little can be done. Prof. Ord found that ten to sixty minims of the fluid extract of jaborandi, administered four times a day, was followed by marked relief. He says the signs of myxœdema almost entirely disappeared under this treatment. Nitro-glycerine benefited one case. Vapor baths are advocated. Dr. Andrew Clark says that baths, assiduous friction, a careful diet, and arsenic and iron as tonics, may sometimes cure the disease.

SCROFULA.

Scrofula is a term applied to many different physical conditions depending upon a diathesis which is regarded as identical with the tuberculous.

Morbid Anatomy.—The characteristic lesions of scrofula are to be found in the lymphatic glands, although the skin, mucous membranes, bones, joints, and organs of special sense may be involved.[1] Inflammation antedates the scrofulous change, and whether occurring in the glands, skin, mucous membrane, subcutaneous connective-tissue, bones, joints, kidney or testicle, the inflammatory product is the same. When fresh it is rich in cells, consisting of a dim, glistening protoplasm with a large single or double nucleus. The exudation is either nodular or diffused. It may undergo resolution, suppuration or organization; all taking place slowly and imperfectly, on account of poor vascularity. Anæmic necrosis sometimes occurs in the glands.

On the skin the lesions appear as eruptions. Impetigo of the eyelashes and external otitis are common strumous diseases. In one who has this diathesis any skin disease takes on a scrofulous character. Scrofulous inflammation of mucous membranes is marked by a thick, sticky exudation, with a tendency to form scabs. The bones most frequently involved are those of the ankle, lower part of the femur, the vertebræ, and rarely the fingers and toes. The scrofulous development may assume the form of synovitis, osteitis, periosteitis, or general arthritis.

Etiology.—The scrofulous diathesis is very largely an inherited condition whose exact nature is unknown, and whose etiology is perhaps equally obscure. It usually has been considered a functional disturbance of impaired vitality, but some recent observations afford ground for the suspicion that it may possibly possess an anatomical basis.

The children of intemperate, phthisical, syphilitic, very old or very young parents develop early all the characteristic features of the scrofulous diathesis. It is also very apt to appear in the children of parents closely related by blood. Heredity is by no means always present, however, for a marked scrofulous diathesis is acquired in early infancy by healthy children from improper food, over-crowding, and anti-hygienic surroundings.

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[1] Virchow taught that the primitive strumous lesion is a simple hyperplasia of the gland tissue, but Schüppel has proven that *a scrofulous gland is a tuberculous gland*. Tubercles stud the glands, which soon become enlarged and soft. When cut they either resemble a normal gland or contain a white, soft cheesy mass mixed with thick pus. Abscess or ulceration may ensue and leave an unsightly scar. Simple chronic hypertrophy results in the formation of knotty groups of glands. Of all the tissues the lymphatic is the most embryonic, the most plastic or potential.

Lack of fresh air, exercise, and sunlight exerts an equally powerful influence in reducing the vitality and the reactive power of the system under irritation.[1] Scrofula and the tuberculous diathesis, if not identical, are so closely related as to be interchangeable.[2]

Symptoms.—Scrofula presents no lesions that may not occur in other diseases, and the scrofulous inflammation has no characteristics, beyond a tendency to extreme *chronicity* and to undergo caseous changes. It is principally a disease of childhood; rarely, however, appearing before the second year.

Children with a scrofulous habit are markedly different in appearance from their healthier mates. Most of them have a transparent, white skin, with delicate blue veins; large, lustrous eyes; bright red lips, and altogether look more like wax figures than healthy children. They are apt to show abnormal mental development, with an irritable nervous system. On the other hand, they may have a large head with coarse features, a thick skin, which has a flabby, spongy feel, an enlarged abdomen and cervical glands. About the upper lip and the nose there is frequently an over-production of fat.[3] In their development no two cases present the same characteristics. Chronic inflammations of the skin, especially about the face and scalp and at the junction of skin and mucous membranes, are frequent, either alone or associated with persistent chronic catarrh of the adjacent mucous surface. Coryza, conjunctivitis, ulceration of the cornea, and otorrhœa often follow an eczema of the face and neck or alternate with it. Laryngitis and bronchitis are obstinately persistent, and may extend to the alveoli and eventuate in phthisis. Pyelitis, cystitis, and vaginal or vulvar catarrh are rarer indications of the depraved condition.

The articular manifestations may appear as a simple synovitis or *tumor albus*, or some slight injury may be the starting-point of caries and necrosis, with suppuration, burrowing of pus, and complete destruction of the joint.

Glandular enlargements so invariably develop sooner or later in scrofulous patients as to be accepted as the most characteristic lesion. This enlargement, which is non-inflammatory and due to cellular hyperplasia, is very gradual, and forms a smooth, firm tumor, which, with similar adjacent glands, may unite in an irregular, shapeless mass. Occasionally these hypertrophied glands subside, but more frequently they finally excite inflammation with suppuration or caseous changes.

The disease progresses slowly with periods of apparent well-being, but toward puberty pulmonary disease is apt to be established; or, if there has been much suppuration, waxy degeneration may occur in the viscera or intestinal tract. Such a condition will not long continue without the development of extreme anæmia and a characteristic cachexia.

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[1] Buhl favors a specific virus theory, but the parasitic origin rests on no certain anatomical facts.

[2] Birch-Hirschfeld found tubercles in nine out of ten lymphatic glands removed from the necks of scrofulous patients.

[3] Canstatt calls this latter the *torpid*, and the former the *erethitic* form of scrofulosis.

Differential Diagnosis.—Scrofulous developments *per se* can hardly be mistaken for any other disease, and a question of diagnosis can only arise as to the nature of chronic degenerative changes other than glandular. Such a diagnosis can be made from the obstinacy of the disease and coincident evidence of the peculiar diathesis.

Prognosis.—The prognosis is good when the patient is seen early, and means exist for a change of diet and surroundings. Scrofulous children may die from tuberculous intestinal disease, acute hydrocephalus, or croup.

Treatment.—The prophylactic treatment embraces a consideration of all the laws of health. Until unhealthy, old and closely related individuals cease to marry, until children receive the proper amount and kind of food for the first two or three years of life, scrofula will exist.

The diet of scrofulous children should be the same as that advised in the treatment of chronic phthisis (*q. v.*). Cod-liver oil will be the chief agent for arresting its progress and development, and should be given daily during the greater portion of infantile and adult life. Iodine is no longer regarded as a specific. Chloride of calcium and the sulphites have been recently highly recommended. Sea or brine baths or even ordinary cold water baths are frequently of the greatest benefit. The treatment of the skin, joint, and eye complications, and the question of extirpation of scrofulous glands, belong to the domain of surgery.

RICKETS.

Rickets or *rachitis* is a disease of general malnutrition with characteristic lesions in the osseous structures.

Morbid Anatomy.—Deficient ossification is the essential pathological change; bones already ossified are softened, and ossification in parts still cartilaginous is prevented or delayed. Growth of the bone is retarded or advances in an irregular manner, and while the medullary cavity increases the osseous shell becomes deficient, owing to proliferation of unossified matter at its circumference. There is an undue development of the cartilaginous epiphyses and fibrous periosteum,[1] causing the clumsy appearance of rachitic bones. The flat bones are greatly thickened at their circumference, from proliferation of the periosteum, but thinned at their centres,—a condition called craniotabes:—this is especially marked in the occipital and other cranial bones. In the lower jaw the anterior wall of the alveolus is sometimes perforated by the milk teeth.

The liver, kidneys, spleen and lymphatic glands are often enlarged from irregular hyperplasia of their fibroid and epithelial elements, conjoined with a deficiency in earthy salts. The brain enlarges from increase in its neuroglia. The muscles are small, pale, flabby, and soft, and their striæ are very indistinct. The ligaments are also wasted. The fontanelles close very late in rachitic children, and, on this account, chronic hydrocephalus may be suspected.

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[1] Virchow thus describes the changes in the diaphyses:—(1) Increasing density of periosteal proliferation and progressive rarefaction of the substance in the areolæ and cancellated tissue. (2) Deficient ossification of the cancellated tissue and continuance of the deep layers of compact exterior substance. (3) Partial formation of cartilage in the areolæ.

Etiology.—Our knowledge of the primary blood changes which result in deficient ossification is largely theoretical. It has been supposed to be: (1) the presence of lactic acid holding the salts in solution; (2) deficiency of lime salts; (3) an inflammation of the epiphyseal cartilages and periosteum;[1] (4) some irritant in the blood.[2] Clinically, rickets is caused by anti-hygienic surroundings. Poor or deficient food and foul air are the most potent factors. Acute disease and troublesome dentition predispose to it. It is more apt to occur in children of rachitic, syphilitic, or phthisical parents.

The disease usually develops during the first year of life, and is rare before the seventh month or after the seventh year of life.[3] Fœtal and congenital forms occur, and in many cases no cause can be ascertained.

Symptoms.—Usually gastro-intestinal disturbances are the earlier symptoms of rickets. There may be vomiting, and the motions are frequent, pasty and offensive. The child, when awake, is listless and drowsy, and when asleep is restless and sweats profusely, mainly about the head and upper parts of the body, regardless of the temperature of the room. He dislikes to be disturbed and frets when any one approaches his cot. There is an intolerance of the bedclothes, which the child is constantly throwing off.

The final distinct evidence of the osteal changes is the enlargement of the lower extremity of the radius and tibia and of the corresponding portion of the ulna and fibula. The softened bones yield readily to pressure, and if the child is allowed to stand or walk, the legs become bent and twisted, and the gait unsteady and swaying. The limbs may remain perfectly straight, though stunted, thin and flabby, when the disease occurs very early in life. The head is large and elongated antero-posteriorly, the fontanelles are wide and the sutures thick. The forehead is very prominent, while the face is small and wizened, with the skin wrinkled as in old age. The lower jaw is shortened, so that the upper teeth overlap the lower. The teeth appear late, the incisors may not appear until the end of the first year, and dentition proceeds very irregularly. The spine is curved, and distortions of the ribs induce an unsymmetrical or oblique thorax.

Rachitic children are usually pigeon-breasted, and there is often marked deformity of the pelvis. The joints are large, loose, and lax. The child is short for his age, and the limbs are short in proportion to the trunk and head. The abdomen is prominent, and the liver and spleen will usually be enlarged; sometimes their enlargement gives the first indication of rachitis. The large cranium, thin face, and distorted limbs cause a rachitic child to present the appearance of a monstrous deformity, when intellectually it is bright and mature beyond its years. Rachitic children are anæmic and very sensitive to changes of temperature.[1] The nervous system is very impressionable, and general convulsions or spasms of the larynx are frequent. All rickety children do not emaciate, and some only suffer pain when they attempt an exertion.

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[1] Niemeyer.

[2] Wagner.

[3] Rehn states that he never saw it develop after the third year.—*Centralb. f. Kindrh.*, 1877 to '78.

Persons who were rachitic in infancy not infrequently become very strong as they reach adult life. They remain of short stature and the deformities persist. In fœtal rickets the body is large and plump, the abdomen protrudes, all the abdominal organs being large, the skin is thick, and the extremities are short and thick. In these cases the chicken-breast is not present.

Differential Diagnosis.—The nocturnal sweats about the head, the osseous changes, the enlargement of the spleen and liver, the weakness of the legs, the rims around the cranial bones, the large, lax joints, and the gastro-intestinal disturbances form a train of symptoms that prevent rickets from being confounded with any other disease.

Prognosis.—As a rule, when the cause is removed the disease will disappear. The greater the thoracic deformity and the longer the disease has existed the worse the outlook. Bronchitis, pneumonia, enteritis, laryngismus stridulus, convulsions, difficult dentition, diarrhœa and chronic hydrocephalus are not infrequent complications. Death may occur from the wasting and anæmia, from the complications, or from asphyxia due to thoracic deformity.

Treatment.—Cleanliness, fresh air, and nutritious food suitable to the age of the patients are of the utmost importance. Children kept too long at the breast often become rickety; they should be weaned at once and have liquor calcis saccharatus added to their food. Cod-liver oil should be taken as early and in as large doses as the child can digest. Scraped raw beef, with a small amount of wine, often produces marked improvement. The intestinal derangements are best corrected by castor oil or rhubarb and soda. In older children quinine, iron, and lime preparations may be administered.[2] The hydrate of chloral is to be used for any nervous derangements. Rickety children should not sleep on feather beds or high pillows, and must not be allowed to run about or exert pressure on any part that may become deformed. Orthopædic measures are treated of in works on Surgery.

ALCOHOLISM.—DELIRIUM TREMENS.

Alcoholismus may be acute or chronic. Acute alcoholismus often manifests itself as *delirium tremens*.

Morbid Anatomy.—In *acute* alcoholismus the mucous membrane of the stomach and duodenum is intensely injected. Patches of aphthæ are found upon it, and the mucous surface of the stomach is covered with ropy mucus slightly tinged with blood. The gastric juice is altered in quantity and quality. The brain, lungs and kidneys are the seat of active hyperæmia, and the pericardium and pleura are often filled with bloody serum. In *chronic* alcoholismus there is chronic gastritis, congestion or cirrhosis of the liver, emphysema and bronchitis, fatty degeneration and dilatation of the heart, atheroma of the vessels, and Bright's disease of the kidneys. Chronic meningitis and pachymeningitis are common. In long-standing cases cerebral softening occurs, and in such the viscera are fatty and the subcutaneous tissue and omentum are loaded with fat if the subjects are beer or wine drinkers; those who drink spirits are emaciated and grow prematurely old, on account of the increase in connective-tissue. Frequently the abnormal accumulation of fat in the abdomen is in striking contrast with the thin, wasted limbs.

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[1] Barthez regards a blowing sound audible over the cranial sutures, as diagnostic of the affection.

[2] Recently the phosphates have been more recommended than cod-liver oil. The fluorides and arsenic are esteemed highly by German physicians.

The blood in chronic alcoholismus contains more fat than normal; one of the first effects of alcohol is a true chemical combination with nerve-tissue, and as the ingestion of spirits is constant, the nerves progressively atrophy and harden. This is hastened by general interference with nutrition from poor blood. The face of the confirmed toper shows turgid and varicose veins,—especially about the nose, which becomes clubbed,—injected conjunctivæ, and pimples of *acne rosacea*. Puffiness under the eyes indicates the changes taking place in the kidneys.

Etiology.—Even more deleterious than alcohol itself are the adulterations of fusel oil, wormwood, and cocculus indicus. Delirium tremens comes on after a prolonged debauch in an old drinker, or when one unaccustomed to alcohol takes a comparatively large quantity of raw spirits. After exposure to cold, prolonged abstinence from food, or some exhausting disease, a small amount of alcohol may induce acute alcoholismus. Chronic alcoholismus is often met with in families where epilepsy, hysteria, insanity, and allied disorders show themselves. In such cases a peculiar constitutional condition which renders abstinence from alcohol especially difficult, is undoubtedly present.

Symptoms.—In acute alcoholism, after a period of exhilaration and semi-delirium, acute coma is very apt to supervene; in this condition the breathing is stertorous, the face is pale, and the pupils as a rule are dilated. The skin is cold and clammy, and the temperature below normal. The urine may be albuminous, and always contains more or less alcohol. Sometimes control over the sphincters is lost. In rare cases delirium tremens occurs after the first debauch.

In *chronic alcoholismus* there is muscular tremor and pyrosis, or vomiting on waking, with entire loss of appetite, the sleep is disturbed, and there is headache and vertigo; the will-power and memory are progressively weakened until entirely lost, the gait becomes ataxic, the face is flabby and the eyes watery. The breath and sweat have a peculiar, offensive odor, the generative functions are enfeebled, muscular tremors become constant, and the patient is in a continued state of dread or anxiety.

Delirium tremens occurs most frequently in old topers after a severe drinking bout, or it follows the sudden withdrawal of stimulants.

The early stages are marked by great mental depression and anxiety—the condition known as "the horrors." There is complete anorexia, the muscular tremor becomes prominent, and the patient's sufferings are increased by a persistent insomnia. At this stage the pulse is weak and feeble, though often rapid, and the surface cool and often covered with a free perspiration.

For several days this condition continues, the patient's mind remaining clear notwithstanding the extreme depression. The advent of the second stage is marked by temporary mental aberration. Gradually these wanderings become more prolonged, and the victim no longer appreciates, as he did at first, the unreality of sights and sounds which torment him. These delusions and hallucinations may begin very simply as imaginary questions, voices, or visions of persons. More frequently they begin, and in any case quickly become, extremely horrible in character. Visions of snakes and toads and all manner of crawling vermin, of wild beasts and still more ferocious men, fill the sufferer with the most agonizing fear. He may cower in a corner paralyzed with fear, or attempt to grapple with his imaginary tormentors. Such patients may attack their attendants with an insane fury. During this delirium the patient talks incessantly; is constantly moving in a quick, nervous manner. His eyes assume a wild or vacant expression, the face is drawn, and gives evidence of the mental distress. The pupils are contracted, the pulse is still more rapid, and, as exhaustion supervenes, what has been a weak pulse becomes distinctly dicrotic. From the first the muscular tremor is persistent, and may even increase as the delirium becomes more violent. Insomnia is still the most dangerous symptom. Four or five days may pass before the patient can be made to sleep, or before he passes into a state of coma vigil. In the former case the delirium is lost in sleep, and usually does not reappear when the patient wakens, or, at most, is speedily ended by renewed sleep. When such a patient can fall asleep, he usually passes to a more or less speedy recovery. But when the delirium passes into coma vigil it is attended by marked typhoid symptoms, which are the precursors of death. Under such conditions a partial delirium continues until complete coma supervenes, shortly before the fatal termination. At times an attack of delirium tremens becomes distinct alcoholic mania or an acute melancholia, which persist for long periods. Other cases of chronic alcoholismus develop chronic dementia with suicidal tendencies, and may become permanently insane. Such cases are rare with acute alcoholismus.

Differential Diagnosis.—The *coma* of alcoholism may be confounded with *uræmic coma*, which has already been considered. Its diagnosis from apoplectic coma will be considered under apoplexy.

It can only be distinguished from *opium* poisoning by an examination of the contents of the stomach, and by an examination of the urine.

The *delirium* of *acute* diseases will not be confounded with delirium tremens if the history of the case and the patient's temperature be taken.

Meningitis is distinguished from alcoholismus by the firm, hard pulse, the pyrexia, the projectile vomit, the retracted abdomen, the photophobia (absent in alcoholismus) and the agonizing headache.

Chronic alcoholic tremor has been confounded with *shaking palsy* (*q.*

v.), with locomotor ataxy and softening of the brain; their differential diagnosis will be considered in connection with the history of these diseases.

Prognosis.—The prognosis is good if the patient is manageable. Death may occur in acute alcoholic coma, and from acute lobar pneumonia which so often complicates it. A patient in delirium tremens may suddenly pass into a comatose state, which will soon be followed by death. The degenerative changes which take place in the vessels and viscera in chronic alcoholism predispose to a long list of diseases, and tend to shorten life. Insanity, impotence, epilepsy, melancholia, and organic brain diseases, are its frequent sequelæ.

Treatment.—In acute alcoholismus the stomach may be washed out and the head douched with cold water, or the patient simply allowed to sleep off his "drunk." In acute mania the stomach should always be unloaded, and cold affusion applied freely. A patient with delirium tremens should be restrained in a large, quiet room, from which bright light is excluded, and should be watched carefully by a strong, trustworthy attendant. In severe cases it may be necessary for the patient's safety to employ a strait-jacket. His diet should consist solely of peptonized milk, and stimulants must be given as demanded by the pulse, and always in moderate quantities during the period of acute delirium. Alcohol is not to be continued after sleep is obtained, or during convalescence. The bowels must be kept open by mercurial and saline cathartics. The one great object is to secure sleep. This is best accomplished by bromide of potash and chloral in full doses, assisted, as may be necessary, by morphia. Morphia must be given with care, and its use is not to be prolonged. When the heart is feeble, strychnia not only increases its force, but augments the action of other remedies given to produce sleep. The bromide and chloral may be continued until the patient wakes from his sleep free from delirium. Large doses of digitalis have been advised, but they fail to sustain an enfeebled heart. During convalescence the vegetable bitters and iron are of service.

TRICHINOSIS.

Trichinosis is a parasitic disease, classed by some among the acute infectious diseases.

Morbid Anatomy.—Trichina spiralis, in the form of a minute worm, measuring about one thirty-fifth of an inch in length, enters the human system through the intestinal tract after the ingestion of trichinous flesh. The muscle larvæ mature two days after, and in six days the embryos are born. In about fourteen days the migrating progeny reach the muscles. Some believe that the blood-vessels are the channels of their conveyance. The most prevalent idea, however, is that they pass through the intestinal walls and peritoneal cavity and then enter the muscular system. Once in the muscles, ovoid protective capsules are thrown around the entozoa, each of which is curled up spirally like a hair spring. The muscular fibrillæ subsequently break down into a granular débris, interstitial connective-tissue forms in abundance, and in the neighborhood the muscles have an inflamed, gray-red appearance.

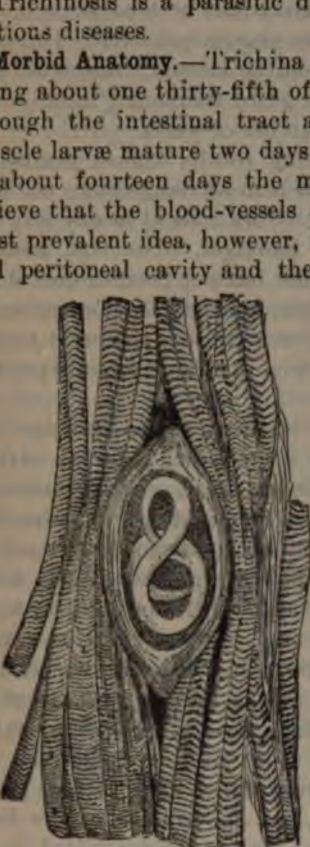

Fig. 190.
Encapsulated Trichinæ in voluntary muscle. × 300.

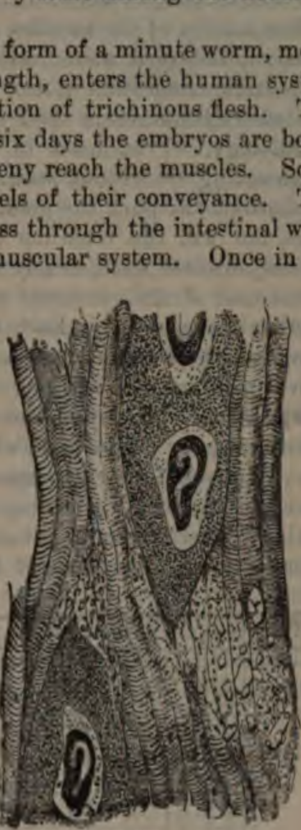

Fig. 191.
Trichinæ with calcareous deposits and degeneration of the capsules. × 300.

The voluntary muscles are those usually invaded. The ends of the muscle—where it becomes tendinous—exhibit the greatest number. The diaphragm, lumbar, intercostal, cervical, and laryngeal muscles, and those of the eye are the favorite sites. As a rule, the farther from the trunk the fewer the trichinæ. At times the heart has been infested with them. The number of the trichinæ in the muscles is greater the longer the disease has lasted.[1] Later the capsules become dense, fibrous, cheesy, and even chalky.

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[1] Cohnheim states that the muscles have no other changes except those met with in acute infectious diseases.

At the autopsy of one who has died of trichinosis during the first week, only the signs of more or less intense intestinal catarrh are found; after the fourth or fifth week, distinct signs of interstitial and parenchymatous inflammation of the muscles are found as fine grayish-red striæ. Intestinal catarrh, enlarged mesenteric glands, peritonitis, venous thrombosis, and hypostatic congestion of the lungs are also quite frequently found. Encysted trichinæ retain their vitality for a number of years.

Etiology.—Trichinosis in the human being results almost exclusively from eating trichinous pork. The raw flesh is most dangerous; the more underdone the pork the greater the danger. Pork cooked in any way that does not kill the trichinæ is dangerous. Sausages, ill-smoked ham, or quickly-broiled ham, or any form of pork that has not been subjected to a moist heat of 170°, is liable to induce it. Salting meat does not necessarily destroy the trichinæ. Each trichina may give birth to a thousand young; about one-half a pound of pork containing trichinæ could rapidly produce thirty millions of trichinæ.[1]

Symptoms.—The symptoms of trichinosis are first gastro-intestinal and then muscular; associated with these there is more or less fever. After a varying time following ingestion of trichinous meat, nausea, vomiting, vertigo, anorexia, a feeling of malaise, and a slight febrile movement occur. There is almost always diarrhœa, the passages being first brownish, then yellow; after a short time there are wandering pains in the limbs, which become stiff and painful to the touch, and the muscles are swollen and rigid. In from four to ten days œdema of the eyelids, perhaps of the entire face, occurs. The temperature ranges from 101° to 106° F., the pulse from 110 to 120; there is photophobia, and movements of the limbs or of the eyes are accompanied by excruciating pain. The pain in the limbs becomes so great that the patient cannot sleep. Œdema of the lower extremities is common; and there may be general anasarca. Copious perspiration with sudamina characterize the fever of trichinosis. The diarrhœa becomes exhaustive, the limbs are paralyzed and the patient lies in a state of utter helplessness.

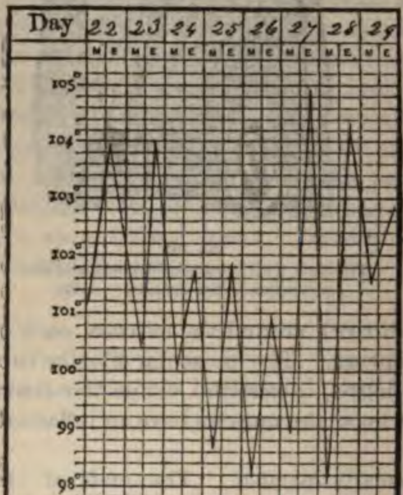

FIG. 192.
Temperature Record in the fourth week of a case of Trichinosis. Death on the 29th day.

Abdominal pains are sometimes present and the muscles of the extremities may become strongly flexed. Deafness and aphonia occur when trichinosis of the stapedius muscle or of the muscles of phonation respectively takes place.[1] When recovery is to occur the symptoms all gradually abate. This occurs in from four to five weeks after the first evidence of its commencement. When death occurs it is usually during the fourth week, and it may or may not be preceded by delirium.

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[1] Trichinæ have been found in rats. mice, dogs, cats, badgers, etc., and swine get them by eating the excrements of these animals or the dead animals themselves, rats and mice especially.

Differential Diagnosis.—Trichinosis may be confounded with *typhoid fever, myalgia, Asiatic cholera* and *inflammation* of the *muscles.* The points of diagnosis between trichinosis and typhoid fever have already been considered.

From *myalgia,* or *inflammation of muscles,* trichinosis is distinguished by the abdominal pains, the diarrhœa, febrile movement, and the history of the case.

Cholera is distinguished from it by the sub-normal temperature, absence of sudamina and copious perspiration, and by the presence of the characteristic rice-water discharges. It is said that the nematoid can be found in the fæces. In all cases a diagnosis can be reached by excising a piece of the deltoid muscle and examining it microscopically.

Prognosis.—There are no reliable statistics by which its rate of mortality can be determined. It may be complicated by hydrothorax, pneumonia, bronchitis, hæmoptysis, gastritis, enteritis, peritonitis and anasarca.

Treatment.—Preventive treatment consists in eating no pork that has not been so prepared as to kill any trichinæ that might exist. The first indication for treatment is to support the patient by a nutritious diet and moderate stimulus. We know of no means of destroying the trichinæ after they have once entered the muscles. Very early in the disease a prompt emetic or a brisk purge may remove the trichinæ from the intestinal tract. Calomel, jalap, scammony and colocynth are efficient for such purpose. To allay the fever and overcome the subsequent anæmia quinine and iron are of service. The treatment is mainly symptomatic.

SYPHILIS.

Syphilis is a specific infectious disease produced only by inoculation. It presents a characteristic acute, initial lesion, and multiform chronic manifestations, which follow a uniform order of development, and are of two distinct forms, called secondary and tertiary lesions.

Morbid Anatomy.—The pathological changes in the primary and secondary stages are essentially inflammatory. About the point of inoculation there is hyperæmia and cell infiltration, followed by necrosis or ulceration, and resulting at first in a papule, and later in a simple excoriation or a shallow, indolent ulcer, with characteristic induration and a dirty-gray base, which eventually leaves a discolored, retracted cicatrix. This ulcer is the typical Hunterian chancre. For its many deviations due to adventitious circumstances reference must be made to surgical works. In connection with the inflammatory changes of the secondary stage there is proliferation of connective-tissue with new formations which soon subside or merge with the tissue in which they occur, producing ostoses, or in vascular organs induration and atrophy. In the tertiary stage the process assumes the form of specific neoplastic formations termed gummata, which may be circumscribed and isolated, but more frequently are infiltrated through the affected tissue. They may appear as firm, gray, opaque nodules, or as soft, translucent masses. They consist histologically of a cell-growth, resembling granulation tissue, and, unlike the earlier manifestations, show little tendency to resolve, but evince a marked tendency to undergo caseous and calcareous changes and to produce necrotic processes in the infiltrated tissue.

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[1] Cohnheim states that the position of one suffering from trichinosis is that in which the various groups of muscles are least extended.

In the skin, mucous membranes, and smaller cartilages and bones, this degenerative process results in fatty degeneration, ulceration, and sloughing, and may result in widespread destruction of tissue. In the deeper organs it produces more or less circumscribed tumors, composed of caseous matter, granular detritus, calcareous deposits and fibroid induration. The distinction between secondary and tertiary lesions is often more distinct clinically than pathologically. The glandular changes which appear soon after the initial lesion are permanent, and are due to cellular infiltration and hyperplasia, but are not usually attended by suppuration.

Etiology.—There is no doubt as to the specific nature of syphilitic poison, or its transmission solely by inoculation, which may be mediate, immediate, or through the processes of conception. The poison is most frequently communicated during sexual intercourse, but inoculation may occur from deposition of the poison upon any abrasion of the surface or upon delicate membranes, as those covering the sexual organs, without any solution of continuity. Thus infection may take place in kissing, or from the use of pipes, drinking vessels, etc., upon which the poison has been deposited. Nursing children may infect, or receive the poison from their nurse. Physicians not infrequently receive it accidentally upon the fingers, or are the agents in its transmission by vaccination.

Even more unfortunate are the victims of syphilitic parents. The poison in the mother invariably manifests itself in the child, and when in the father, infects the offspring and, secondarily, the mother through the fœtal circulation. The syphilitic poison is most virulent in the primary sores and glandular affections, but is present in the blood in decreasing quantity through both the secondary and tertiary stages. Late in the disease it is found only in the discharges from those organs which are involved in the specific processes. One inoculation of syphilitic poison, with rare exceptions, confers protection from all subsequent poisoning.

Symptoms.—*Primary.* The period of incubation is variable, but is seldom less than ten days and averages about twenty-five. The first change is the appearance of a dark-red papule, which slowly enlarges, becomes indurated early, is not painful, and may even escape notice. Although it may run its full course without becoming moist, generally the apex becomes eroded, leaving a moist surface, or undergoes ulceration. The true chancre does not secrete pus unless it becomes inflamed, but remains a simple excoriation, either moist or scabbed, through its entire course. The induration may be thin and superficial, may simply underlie the excoriation, or may spread extensively into adjacent parts. In the course of six or eight weeks the sore begins to heal, the induration subsides, and finally there is no trace left, or if ulceration has been present, there remains a white or slightly pigmented cicatrix. Soon after the appearance of the primary sore, the nearest lymphatic glands indurate and enlarge but rarely suppurate. Nor do they resolve with the healing of the chancre, but remain enlarged for months and years, and are eventually joined by other glands throughout the body.

Secondary syphilis includes the earlier and generally lighter affections of the skin and mucous membranes, with some of the affections of the organs and nerves. The most prominent are those of the skin which usher in the eruptive stage of syphilis. They appear from six weeks to three months after inoculation, and in nearly one-half the cases before the initial lesion has healed. This stage is often attended, at its invasion, by some slight fever and constitutional disturbance, marked by weakness, emaciation, and wandering pains in the limbs and joints. The cutaneous syphilides assume nearly all the types of skin diseases, and present in this multiplicity of form a distinctive characteristic. The earlier eruptions are generally the simpler forms of erythema, and papules, and are diffused over the surface quite uniformly. Later, there appear vesicles, pustules, tubercles, and scaly eruptions which are more apt to be gathered in groups. Not infrequently several or all the forms may be present. In all syphilides there is a general roundness of form, an absence of pain and itching, and a peculiar livid coppery color which gradually changes in cicatrices to a glistening white.

Secondary syphilis most frequently affects the mucous membranes of the fauces and pharynx. In connection with the earlier symptoms there may be only a diffuse hyperæmia and a redness with or without ulceration ; but with the later secondary and earlier tertiary, there is a peculiar dusky red appearance, the result of chronic congestion, and more or less thickening and induration about ulcers and mucous patches. This condition seldom causes any pain or discomfort, and the ulcers may disappear spontaneously. Mucous patches appear most abundantly about mucous orifices, as the mouth and anus, but may appear on the skin. They are round or oval, slightly elevated spots of varying size, with a moist excoriated surface, which does not ulcerate unless irritated. They appear with the earliest eruption and continue with decreasing frequency into the tertiary stage.

The secondary affections of the eye assume the form of iritis with extensive exudation, and retinitis, which, appearing with but little pain or photophobia, is attended by extravasations and partial or complete abolition of function. In connection with the general tegumentary inflammation the hair-bulbs are involved, and the hair becomes thin or is lost entirely.

fumigation, baths and inunction; the latter being the most desirable for children with inherited syphilis. Iodine may be used in its combinations with potash, soda, ammonia, mercury, iron, etc. The doses of both mercury and iodine must be determined by trial for each case. With mercury they should fall short of salivation, but with iodine should increase to the limit of the patient's endurance, or until the lesions yield to treatment.

SECTION VI.

DISEASES OF THE NERVOUS SYSTEM.

(Including Diseases of the Brain, Spinal Cord, and Functional Nervous Diseases.)

GENERAL SYMPTOMATOLOGY.

THE symptomatology of nervous diseases presents many peculiarities which render their diagnosis especially difficult. Nearly every physical and rational symptom may be the result of so many different lesions that at best it is indicative of the seat and extent of the lesion only, and not of its nature, and often only determines the division of the nervous system which is affected. Symptoms are, therefore, often entirely negative when taken singly, and find their significance only in the order and manner of their development, or in their combinations with others equally valueless *per se.* All lesions of nervous tissues result in (1) impairment or abolition, (2) exaltation, and (3) perversion of function, and may manifest themselves through the motor, sensory, co-ordinating, or psychical systems, by symptoms which will vary with the location, nature, and extent of the lesion. I shall first consider some of the more important symptoms in their general relations to nerve lesions.

Motor Paralysis.—Loss of motor power, or voluntary nervous control of muscular movements, may exist in all degrees, from the slightest weakening or delaying of the nervous impulse to absolute abolition of the impulse, or its complete arrest in transit to the muscles. The lighter and intermediate grades are termed *paresis,* while *paralysis* is applied to extensive or entire loss of motor power. The nature and extent of the paralysis is evidenced in the muscles, so that the muscular condition becomes a matter of primary importance.

(a) Reflex Action.—In many instances, and more especially in spinal paralysis from circumscribed lesions, the muscular force, as indicated by reflex movements, is not diminished. Under suitable irritation the apparently powerless muscles execute violent movements and become powerfully contracted. These reflex movements are not always limited to the irritated limb, but may appear in other paralyzed muscles. For diagnostic purposes, then, reflex action indicates unimpaired nervous connection between the paralyzed muscles and the spinal centres, and can never be present when the paralysis is due to a disseminated destructive lesion of the nuclei of origin of the affected nerves or the nerve trunks, but is generally most distinct in disease interrupting the transmission of voluntary motor impulses. A very common and patent form of reflex action is known as *tendon reflex.*

If in health the tendon of any muscle be struck a sharp, quick blow, there will immediately follow distinct contraction of its attached muscles, most marked, of course, in the larger muscles, as those attached to the patella or tendo Achillis. Abolition or exaggeration of tendon reflex is an important point in diagnosis. Either may be associated with decrease or increase of reflex action from irritation of the skin. A third form of reflex action is called ankle clonus. This consists in a clonic tremor of the muscles, particularly of the leg, occurring whenever the muscle is stretched by flexion of the foot, and continues during flexion. In severe cases it may be excited by putting the toe to the floor, and then often involves the entire limb. Abolition of reflex action may be due to degenerative changes, either in the nerve trunk or spinal centre, but exaggeration is generally the result of central irritative lesions.

(b) Electrical Irritability.—Electrical contractility of paralyzed muscles may remain normal, be increased, or impaired. When not otherwise specified, it is understood that the muscular contraction is produced by a current applied to the motor nerves. When muscles atrophy from disease, or are the seat of degenerative changes, Faradic contractility is proportionately decreased. As dependent upon nerve changes it is not only generally retained in both cerebral and spinal paralysis due to interruption of nerve-currents, but is frequently, and especially in the latter form, increased. When disease involves central nuclei or nerve-trunks, Faradic contractility is often rapidly and extensively lost. Such muscles may still react, however, to the slowly interrupted galvanic current, even after they fail entirely to respond to the Faradic. Indeed, galvanic contractility may increase as Faradic decreases, and eventually become more marked than in healthy muscles. When nerve-trunks no longer respond to electricity, the muscle itself may, in some conditions, still show an increasing electrical irritability. This, in its various forms, is called reaction of degeneration.

(c) Muscular Nutrition.—Muscular nutrition and tonicity generally keep pace with contractility; and the muscles remain firm and are but slightly reduced in bulk, or they may become small and flabby, or in some cases contracted and rigid. The former condition prevails when disease lies above the origin of the implicated nerve, but sudden onset of the disease or implication of nuclei or nerve trunks results in flaccid and wasting muscles. Rigidity and contraction follow irritative lesions or complications and also occur in paralysis of long standing, in which case they are mostly due to secondary descending degeneration of the cord.

General Paralysis.—Lesions resulting in general paralysis of necessity involve such important parts as to be followed in most cases by immediate death. General paresis occurs in connection with insanity from diffuse disease of the cerebral cortex. It implicates all the voluntary muscles, not excepting those of deglutition and phonation, but is slight in degree, seldom extending beyond weakness and sluggishness of movement.

Bulbar paralysis is perhaps the nearest approach to general paralysis. Disease of the pons or medulla generally results in bilateral paralysis, and is the cause of the mixed and crossed paralyses occasionally met with, as paralysis of both arms, both legs, or one side of the face and the opposite side of the body. Motions of the eye, phonation, deglutition and respiration are especially liable to be interfered with, and death is seldom long delayed.

Hemiplegia is a motor paralysis limited to a lateral half of the body. It is generally the result of a lesion above the medulla, and most frequently of the corpus striatum, but may result from injury to a cerebral hemisphere or crus. It occurs on the side opposite to the disease or injury. Its most frequent cause is undoubtedly apoplexy, but it may be due to other cerebral injuries or disease, and not infrequently is functional. The causes of hemiplegia may be classified as follows :

Cerebral Causes....	Compression from bone, blood, pus, or inflammatory exudations. Tumors, especially carcinoma, sarcoma, gummata. Partial anæmia from thrombosis, embolism, softening, aneurism, apoplexy. Encephalitis,—abscess. Atrophy and sclerosis.
Spinal Causes......	As above, or any disease affecting a lateral half of the cord.
Functional Causes..	Hysteria, chorea, epilepsy, diphtheria, malaria, poisons, etc.

Although the paralysis is of central origin, the muscles are seldom affected uniformly, and it has been noted that those which suffer least are such as act in conjunction with their counterparts on the non-paralyzed side. The muscles of the arm and leg are chiefly affected, while those of the trunk and neck often escape entirely, so that the body and head remain erect and firm. The paralysis is generally descending in its onset, and ascending in its recovery, the leg being last involved and the first to regain its power. Occasionally, however, the leg escapes entirely, or it may suffer a more complete paralysis than the arm. Of the cranial nerves the third, fourth and sixth seldom suffer unless the lesion is in the crus, when the third will probably be involved. The fifth, also, as a rule, suffers but little, but may be paralyzed in either or both roots, a condition indicated by anæsthesia of the face and cornea and paralysis of the muscles of mastication on the affected side. The facial, on the other hand, seldom escapes entirely in lesions at the base. The face becomes blank and motionless, the mouth is drawn toward the healthy side, and the paralyzed cheek puffs on expiration. The muscles of the tongue may escape or suffer with the others, and the tongue will then be protruded with the tip pointed toward the affected side. When hemiplegia is uncomplicated its diagnosis is evident, but if associated with coma it may not be readily appreciated. The paralyzed limbs, however, will be more flaccid, and when raised and released will drop more heavily and limply than on the unaffected side. If the face is implicated the peculiar expression and retraction of one angle of the mouth will be readily appreciated. In the differential diagnosis of the causes of hemiplegia the location, nature, and extent of the paralysis will be of value, but the most important points will be found in the history of the case, the manner of invasion and the peculiar combination of other symptoms. It has been noticed that paralysis caused by lesions in the motor tract above the ganglionic cells in the anterior cornua of the cord is seldom followed by much muscular atrophy, or more than would be caused by inactivity, while disease affecting these cells or the peripheral nerve-trunks produces marked muscular atrophy as well as paralysis.

Paraplegia.—Bilateral paralysis, of whatever extent, is termed paraplegia, and, when of organic origin, affects only those parts of the body supplied by nerves leaving the spinal cord at or below the seat of the lesion. Its causes may be classified generally as follows:

Brain.......	Small clot in the pons.
Spinal	Compression of the cord from bone as in fracture, caries, dislocation, spina-bifida, from blood (traumatic), pus, exudations, tumors, all diseases of the cord, shock and concussion.
Peripheral ..	Inflammation or degeneration of nerve-trunks.
Functional..	Hysteria, catalepsy, rheumatism, syphilis, poisons.
Reflex	Diseases of genito-urinary organs, diseases of intestines.

Organic paraplegia, therefore, is commonly of spinal origin, and in extent varies with the seat of the lesion. If this is located in the dorsal region the lower extremities alone are affected; the paralysis becomes more extensive the higher in the cord it has its origin; when this is in the cervical region the entire body, including the diaphragm, may be paralyzed. In all forms, however, the sphincters are liable to be involved. Generally if the disease is high up there will be spasm and retention; if low down, paralysis and incontinence. Paralysis may be of all grades and varieties, according as more or less of the thickness of the cord is involved. There may be simply slight paresis, decided paralysis with sensation unimpaired, or complete paralysis of both motion and sensation. Disease of the cord has a special tendency to be unsymmetrical, and confined to particular tracts As a result, the effects are very varied. When the changes are confined to a lateral half, motor paralysis affects the parts below on that side; but owing to the immediate decussation of sensory fibres on entering the cord, and their consequent implication with motor fibres of the other side, anæsthesia is found on the opposite side of the body below the lesion, with possibly a distinct line of anæsthesia marking the upper boundary of motor paralysis. The limitation of spinal lesions to distinct tracts has given rise to such characteristic combinations of symptoms as to lead to their being considered as special diseases.[1]

Aside from the foregoing forms of paralysis for the most part due to interruption of the connection between nerve-nuclei or trunks and the higher centres, paralysis may result from direct injury to, or destruction of, these nuclei or trunks. In such cases the paralysis is confined to the distribution of the affected nerves, is generally more complete and permanent than in other forms, and is attended by rapid loss of Faradic irritability, with wasting of the muscular tissue and the reaction of degeneration.

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[1] See Locomotor Ataxia, Progressive *Muscular Atrophy*, etc.

Spasms, Convulsions.—In determining the seat and nature of the disease causing spasms the same anatomical facts are to be considered as in the diagnosis of paralysis. It is probable that irritative lesions of the same centres as are affected in paralysis result in motor disturbances; hence convulsions of a lateral half of the body may be ascribed to irritation of the opposite cerebral hemisphere, corpus striatum or crus. In a similar manner spasms confined to the lower portion of the body and bilateral are to be considered of spinal origin, while general convulsions may be the result of general cerebral disturbance or of a general affection of the cerebro-spinal system. Basing the diagnosis upon our knowledge of the motor areas of the cerebral cortex, it is possible in many cases to locate the lesion quite exactly by careful consideration of the location and extent of the convulsive movements. Spasms are even more varied in their distribution than paralysis, affecting single muscles, muscular groups, a single limb, half the body or all the muscles, not excepting those of respiration and deglutition, and they vary in degree from light fibrillary twitching to such violent cramps as to rupture muscles or to fracture bones. When the contractions are persistent they are termed tonic, but when rapidly alternating with relaxation are called clonic. Convulsions appear as symptomatic of both organic and functional disease. Tremulousness of the muscles often accompanies paresis, and paralysis is frequently followed by tonic contractions. Fibrillary twitchings are common in debilitated conditions, in general paresis, the typhoid state, paralysis agitans, etc.; while the severe forms are illustrated in inflammatory conditions of the brain and cord, epilepsy, tetanus, strychnia poisoning, hydrophobia, etc.

Sensory Paralysis, Anæsthesia.—Anæsthesia, like motor paralysis, may be located in any part of the body, may be of all degrees, and may be superficial or extend to deep parts. When slight it is only a sense of numbness which gives the impression of some soft substance covering and protecting the parts, and is generally attended by formication or burning prickly pains. In complete anæsthesia the patient is unconscious of the severest injury, and bed-sores may denude the bones without his being aware of their existence. In some cases sensations of heat and cold are still appreciated, while all other sensibility is lost. Among perverted sensations may be placed those conditions in which sensation is delayed, and the patient appreciates the impression only after the lapse of some seconds, or is unable to determine its nature. In many cases he suffers severe neuralgic pain in the anæsthetic parts, due to the central nervous irritation. Anæsthesia may be general with general paresis in insanity, but rarely so in other conditions. It more commonly appears as hemi-anæsthesia, from causes similar to those of hemiplegia, but is less frequent than the latter. It most frequently depends upon lesion of the external capsule or fibres of communication between the optic thalamus and hemisphere, and frequently implicates some of the nerves of special sense. Lesions of the tegmentum of the crus also result in opposite hemi-anæsthesia. Spinal anæsthesia is also far less frequent than paraplegia, but when present is almost always associated with paralysis. The condition of reflex action will indicate somewhat its nature.

When paraplegia and anæsthesia are the result of destruction of nerve nuclei in the cord, or of injury to the nerve-trunks supplying the paralyzed part, reflex activity will be abolished. When, however, the paraplegia is of parts below a spinal lesion, reflex action is normal or often increased. As noted before, a lesion of a lateral half of the cord may give a paraplegia compounded of motor paralysis on the side of the lesion and sensory paralysis on the opposite side.

Hyperæsthesia.—Hyperæsthesia, either general, partial, or of the nerves of special sense, is of common occurrence in nervous disease, since it may be the result of the most trivial disturbances. It is present in the congestive or early stages of inflammatory conditions of the brain and cord, and in functional disturbances. It is often a symptom in the earlier stages of febrile diseases and in inflammation of the skin. When normal sensation becomes painful, it is termed dysæsthesia, and appears as gastralgia, enteralgia, or, in the nerves of special sense, as sparks and flashes of light or even the appearance of distinct forms of men and animals, ringing or violent explosive sounds, and, in some cases, continuous conversation, or as disturbances of taste and smell.

Disorders of co-ordination are of rare occurrence except in connection with sclerosis of the posterior columns of the cord. Disease of the cerebellum is indicated by lack of co-ordination, a staggering gait, or entire inability to maintain the erect position.

Mental Disturbances.—All forms of cerebral disease are attended by more or less perversion of the mental powers, but such symptoms are suggestive only of the general nature of the cerebral changes, and but remotely of the character of the lesion. Hyperæmia and inflammatory conditions generally produce at first exaltation of mental processes which may vary from simple excitement to the wildest delirium. On the other hand, any lesion which causes sudden shock to nerve centres or interferes with nutrition, either by simple pressure or through destruction of the cerebral tissue, is generally indicated by depression or abolition of mental power. Patients evince the most varied forms of mental disturbance, and at different times suffer in their emotions, intelligence, or will. They may be happy, hilarious, angry, or sober, melancholy, sullen and distressed. In intelligence they may appear brilliant, vivacious, and the exaltation may extend to delusions and hallucinations, or they may lose all reasoning power and memory and become idiotic. Delirium of meningeal origin is generally active or even maniacal, but becomes low and muttering, as in the typhoid state, when the lesions implicate the cerebral ganglia and result in general nervous depression. Like all other symptoms of similar origin it commonly ends in coma, with abolition of sense, sensation and voluntary motion. Although *coma* is the usual termination of cerebral disease, it is dependent upon many other and diverse causes, and often demands a differential diagnosis as to its origin. Its causes are classed as cranial or extra-cranial, although they all act directly upon the cerebrum.

The more common causes are :

Cranial........	Hyperæmia, Anæmia, Œdema, Compression, Tumors, Thrombosis, Embolism, Apoplexy, Abscess, Softenings, Shock, and Concussion.
Extra Cranial..	Hysteria, Epilepsy, Uræmia, Ammonæmia, Cholæmia, Poisons of drugs, Narcotics and Anæsthetics, Antispasmodics, Alcohol, Poisons of fevers, Malaria, etc.

Trophic Changes.—Many forms of nervous disease are attended by peculiar and rapid trophic changes throughout the body. They appear in the skin, muscles, joints, bones, and viscera. The more common are bed-sores and inflammations of the urinary tract. It may suffice to say generally that trophic changes are associated only with inflammatory or irritative lesions, which implicate the nerve-trunks or their nuclei of origin in the case of motor nerves, but in the case of sensory nerves may be located in the gray matter of the posterior portion of the cord. Injury of motor nerves generally results in muscular or arthritic changes, while cutaneous changes are dependent upon lesions affecting sensory nerves.

DISEASES OF THE BRAIN

will be considered under the following heads :—

I. *Cerebral Hyperæmia—active or passive.*
II. *Cerebral Anæmia.*
III. *Meningitis.*
IV. *Cerebral Thrombosis and Embolism.*
V. *Cerebral Softening.*
VI. *Cerebral Apoplexy.*
VII. *Abscess of the Brain.*
VIII. *Cerebral Tumors.*
IX. *Sclerosis of the Brain.*
X. *Hypertrophy of the Brain.*

CEREBRAL HYPERÆMIA.

(Congestion of the Brain).

Cerebral hyperæmia is an increase in the quantity of the blood within the capillaries of the brain. It may be *active* or *passive*. In active hyperæmia there is increased current, and the blood is arterial, while in passive hyperæmia the current is retarded, and there is an excess of venous blood. It seems probable that both forms of hyperæmia may really produce anæmia, so far as the cerebral nutrition is concerned, by compression of the capillaries and lymph spaces.

Morbid Anatomy.—In *passive hyperæmia,* the veins and sinuses are engorged with blood, and, when long continued, the dura mater appears distended, and sometimes the cerebral convolutions are flattened, with a decided pinkish color in the gray substance. On microscopical examination, the perivascular lymph spaces are seen greatly diminished, or possibly obliterated. In the former case, large pigment granules are scattered outside the vessels along their line.

On section, the white substance is seen dotted with numerous blood points, and the cortex is grayish red. At the lower portions of the cerebellum there are dark red patches. In *active hyperæmia* the small arteries are enlarged, and the capillaries of the meninges are distended. This may be accompanied or followed by œdema of the pia mater and distention of the ventricular cavities.[1] The condition of the membrane is no guide, either to the existence or degree of hyperæmia, and transitory active or passive hyperæmia often leaves no trace discoverable at the autopsy.[2]

Etiology.—Active hyperæmia may be due to increase in the blood pressure, from excessive action of the heart, from contraction of the surface capillaries during a chill, from prolonged mental labor, intense emotion, digestive disturbances, acute blood poisoning, increased atmospheric pressure, and gravitation from a prolonged recumbent posture. Local arterial anæmia in other parts of the body, such as arises from sudden cold to the surface, intense muscular exertion, and pressure of tumors or dropsical fluids on the main branches of the aorta, may also induce active hyperæmia.[3]

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[1] Ecker states that the capillaries and small vessels are sometimes double their normal calibre. Niemeyer and Nothnagel state that atrophy of the brain may result from chronic passive hyperæmia.

[2] Many pathologists, while admitting the possibility of partial congestion, ascribe to post-mortem changes what others denominate local congestion.

[3] Watson states that men have been arrested as drunk on cold nights, when they were only suffering from active cerebral hyperæmia.

Paralysis of the vaso-motor nerves of unknown origin, or severe nervous shock, and poisons, alcohol and certain drugs, especially nitrite of amyl, will give rise to active cerebral hyperæmia. It occurs more frequently in hot climates than in cold, and is said to follow breathing exceedingly rarefied air. Insolation is probably more than intense active hyperæmia.[1]

Passive cerebral hyperæmia, when general, is the result of obstructed venous circulation, itself the result of pressure upon the jugular or vena cava descendens. Prolonged fits of coughing, playing on wind instruments, and prolonged straining at stool may induce it. Any cardiac valvular lesion that obstructs the blood in the pulmonary vessels, or any disease of the lungs which offers obstruction to the onward current, will lead to passive hyperæmia of the brain. Tricuspid regurgitation stands pre-eminent among these causes. Partial or complete stenosis of the larynx will induce it, as in croup and œdema glottidis. Thrombi in the cerebral sinuses may induce passive hyperæmia; and it sometimes occurs from feebleness of the incoming arterial flow.

Symptoms.—The symptoms of cerebral hyperæmia may be grouped in two classes:—those of excitement and those of depression. In all cases these symptoms are increased by a recumbent posture, by a forced inspiration and by stimulants. The symptoms of *excitement* are a diffuse pain and throbbing in the head, accompanied by dizziness, vertigo, flashes of light, ringing in the ears, restlessness, insomnia, and perhaps delirium and convulsions. Photophobia is present; and there may be nausea and vomiting. Sleep is usually broken and disturbed from the onset. The gait is unsteady, the mind confused, and sometimes the speech embarrassed. In active hyperæmia the pulse is accelerated, full, bounding, and hard, and the carotids and temporals pulsate forcibly. An ophthalmoscopic examination reveals injection of the retinal vessels, and the conjunctivæ are often suffused. In most cases there are both motor and sensory disturbances. As a rule, the mental state in active hyperæmia is one of exaltation. These patients are irritable, peevish and highly excitable. They are apt to talk a great deal. When coma occurs, the hyperæmia is described as *apoplectic;* when convulsions or spasms are present, it is called *epileptic;* and when there is delirium we have the *maniacal form.* The latter is *mania ephemera* or *impulsive insanity.*[2] Paralytic symptoms are rare in active hyperæmia.

The symptoms of *depression* are dull headache, vertigo, ringing in the ears, with confusion of mind and dulness passing into somnolence, stupor, or complete coma. Illusions and hallucinations are uncommon.[3] Convulsions may occur in children. When due to passive hyperæmia, as is not uncommon, there is a cyanotic hue to the face and neck, the jugulars and venous system are over-distended, and the arterial system is scantily filled. The pulse varies with the etiological (cardiac) lesion. In old people, after depression of spirits, or a long period of taciturnity, there follows wandering delirium, usually nocturnal, talkativeness, and a state often bordering on hysteria. The mind becomes more and more inactive, and sensation as well as motion is diminished. This condition is followed by coma, with stertorous breathing and relaxation of the sphincters. This coma, which is frequently interrupted by local or general convulsions, generally ends in death.

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[1] See the interesting experiments of S. Mayer and Pribram (*Sitz. der Wien. Akad.*, 1872), in which electrical or mechanical irritation of the walls of the stomach produced a reflex increase of the vascular pressure and considerable diminution in the frequency of the pulse.

[2] Trousseau and Nothnagel both claim that the epileptic and apoplectiform varieties are either true epilepsy or are due to actual cerebral hemorrhage.

[3] Griesinger describes a peculiar *fear of places* that seizes patients when in the midst of a crowd or while in a certain place or street.

Differential Diagnosis.—Cerebral hyperæmia may be mistaken for *apoplexy, embolism, uræmia, acute alcoholismus, epilepsy,* and *cerebral anæmia.*

In *cerebral hemorrhage* the onset is more sudden ; the coma is more complete and prolonged, and following the attack there is always hemiplegia.

In *cerebral embolism* the onset is sudden ; the face is pale, the head cool, the respirations and pulse-rate are rapid and *irregular,* there is usually evidence of cardiac valvular disease, aphasia follows the attack, and the symptoms are more permanent than in acute hyperæmia.

In *uræmia* the coma is deeper and generally preceded by convulsions. There is œdema of the eyelids or of the lower extremities, and the urine will be found to contain albumen and casts.

In *epilepsy* there is usually an *aura,* and the patient falls as if from a blow, uttering the epileptic cry. In the fit, which is of short duration, the convulsions are first tonic and then clonic, and there is a bloody froth about the mouth.

The diagnosis between stomachic and cerebral *vertigo* will be found under Diseases of the Stomach.

It is often impossible to distinguish between *acute meningitis* and cerebral hyperæmia in children, except from the results of treatment, until the disease is well advanced.

In cerebral hyperæmia the headache is generally diffused and the pupils are contracted, while in *cerebral anæmia* the headache is vertical and the pupils are dilated. In hyperæmia there are loss of memory and hallucinations, in anæmia we have simply incapacity for mental work. In anæmia the respirations are hurried, the pulse is quick, feeble, and irritable, there are murmurs at the base of the heart and in the vessels of the neck, and the face is pale and cold.

Prognosis.—The prognosis depends upon the cause ; as a rule, therefore, passive is less favorable than active hyperæmia. The maniacal or apoplectiform variety is the most, the convulsive the least, dangerous. The outlook for recovery is best in those whose habits are good, who can exercise mental control and avoid excitement, and in those between twenty-five and fifty. In youth and old age the prognosis is more unfavorable on account of the condition of the cerebral vessels. Accompanying cardiac and pulmonary disease, passive hyperæmia is a symptom of secondary importance.

Treatment.—During an attack of cerebral hyperæmia from whatever cause, the patient must be kept absolutely quiet in bed with the head *raised,* and the diet should be the simplest and most easily digested, and taken in small quantities at short intervals. In active hyperæmia cold to the head and heat to the feet, with the administration of a brisk purge, are to be the first measures. The bromide of potassium is beneficial in most cases. In severe active hyperæmia blood-letting is permissible in the form of leeches to the temples or nose. The constant current may be used to stimulate the sympathetic nerve, and thus contract the cerebral blood-vessels. Some advise zinc in combination with bromide of potassium. Ergot and antimony have been used with some success in active hyperæmia.

In passive hyperæmia stimulants may be given with bromide of potash or soda. Digitalis is usually indicated. Sulphuric ether, inhaled or given internally, often produces good results. In coma due to passive cerebral hyperæmia, all remedies will prove ineffectual, except *quinine*, which should be given in small doses at short intervals. When cessation of the menses or of an old hemorrhoidal flux is followed by cerebral congestion, leeches to the anus, sitz-baths, emmenagogues, etc., should be given in connection with the other remedies. A change of residence with rest from mental work is often of great benefit.

CEREBRAL ANÆMIA.

Cerebral anæmia is a condition in which there is a deficiency in the quantity or quality of the blood in the capillaries of the brain.

Morbid Anatomy.—The principal change in cerebral anæmia is pallor of the brain, which may be partial or general, accompanied by serous effusions into the meshes of the pia mater. The ventricles are often distended with fluid and the veins and sinuses are engorged.[1] In some cases hyperæmia of the meninges coexists with cerebral anæmia. *Partial* cerebral anæmia is not often demonstrable. It exists about neoplasia and adventitious products; and may be the result of local pressure, or partial occlusion of an artery.

Etiology.—It may be due to general systemic anæmia from excessive hemorrhages, or to sudden pulmonary hepatization and congestion in other organs. Exhausting discharges, prolonged lactation, etc., induce it. Spurious hydrocephalus following infantile diarrhœa is a condition of cerebral anæmia. It may result from defective blood nutrition, as in chlorosis, or cardiac weakness, mitral or aortic valvular disease, or cardiac insufficiency occurring in acute febrile diseases. Fatty heart is even more potent than valvular lesions in producing it. Any mechanical interference with the supply of blood to the head, as pressure on, or ligation of, the carotids, may induce cerebral anæmia. The arteries that form the circle of Willis are, in twenty per cent. of cases, so distributed that only imperfect communication exists between their two lateral halves. Mental influences through vaso-motor spasm may produce it; thus fright, joy, or anger will, in many, produce syncope due to cerebral anæmia.

Cerebral anæmia sometimes follows the application of strong electrical currents to the spinal region and irritation of the peripheral nerves. It is claimed that zinc oxide, the bromides, tobacco, calomel, and tartar emetic, if long continued, will cause cerebral anæmia. Partial cerebral anæmia is always due to local obstructions, which may result from narrowing of the vessels from disease of their walls, from spasm of their muscular coats, from embolism or thrombosis, and from pressure of tumors, blood, bone or inflammatory products.

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[1] Golgi claims that there is enlargement of the perivascular lymph-spaces.

Symptoms.—The symptoms of cerebral anæmia may appear suddenly or slowly. In the former case they are the ordinary phenomena of a *fainting fit;* the individual becomes dizzy, nauseated, and the sight is obscured; there is ringing in the ears, the pupils dilate, the gait is unsteady, cold perspiration covers the surface, the pulse becomes feeble, rapid, and thready, and the respirations hurried; and the patient may fall to the ground with slight spasmodic twitchings. Such a condition is common from reflex causes. If due to extensive hemorrhage the loss of sight in those that recover may be permanent.

When cerebral anæmia comes on slowly it is attended by headache, drowsiness, vertigo, muscæ volitantes, tinnitus aurium, sometimes attacks of total blindness, inability to perform work, insomnia, extreme *sensitiveness* to *light* and *noise,* and at times delirium and hallucinations. Notwithstanding the maniacal character of the delirium, melancholia is often its predominant feature. Though usually of brief duration, it may end in permanent insanity. In most cases the pupils are sluggish and dilated, the retina is anæmic, and the headache is confined to a small circumscribed spot. The recumbent posture induces insomnia, and the erect position often causes a sense of general muscular weakness and faintness. The face is pale and cold. In the cerebral anæmia of children—spurious hydrocephalus—restlessness, jactitation, grinding of the teeth, and muscular twitchings are followed by symptoms of collapse and coma.

Differential Diagnosis.—The points of differential diagnosis may be found under the heads of Tubercular Meningitis and Cerebral Hyperæmia.

Prognosis.—The prognosis in cerebral anæmia will be determined in most cases by its causes. Where acute anæmia results from hemorrhage, death may result, although the hemorrhage has been arrested. The prognosis is more favorable when there is no organic disease of the heart or vessels. The more speedily the cause can be removed the better the prognosis. In the so-called spurious hydrocephalus the prognosis is favorable, if met by prompt and suitable treatment.

Treatment.—In acute anæmia with syncope the head must be lowered at once and the patient may even be inverted, cold water may be dashed over the face, the vapor of ammonia inhaled, and bandages applied from the feet upwards. As soon as consciousness is regained, champagne, ether, ammonia, coffee, or other cardiac stimulants may be administered. If these measures fail transfusion should be resorted to. Alcohol is to be given very cautiously in cerebral anæmia; and exercise of either brain or body must be carefully undertaken. Bromides are never to be given.[1] For chronic anæmia the treatment can only be determined by *causal* indications.

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[1] Nothnagel advises small doses of morphine for the excitement that precedes anæmic delirium.

MENINGITIS.

The membranes covering the brain are the pia and dura mater; the *pia mater* is a delicate and exceedingly vascular membrane which is intimately adherent to the brain, sending prolongations into the sulci and fissures; its connective-tissue is very extensive. The external layer of the pia mater is the structure formerly called the arachnoid. The meshes of the pia mater were formerly denominated the subarachnoid space.[1] The *dura mater* acts as the periosteum of the cranial bones; it is firm, not very vascular, and encloses venous sinuses between its folds. Its inner surface is covered with a layer of endothelial cells.

Inflammation may have its seat in the dura mater, the pia mater, or both may be involved. Inflammation of the pia mater is usually called *meningitis*. The term *pachymeningitis* is applied to inflammation of the dura mater.

When inflammation of the pia mater is attended by rapid changes in the membrane and by the effusion of sero-fibrin and pus, with varying quantities of red blood globules, it is called acute meningitis.

So-called sub-acute meningitis is a mild type of acute, in which extensive serous effusions are the chief, and at times almost the sole, event, although small quantities of lymph, white and red blood cells, and fibrin are usually present.

Inflammation of the pia mater, when chronic, results in thickening, opacity, and adhesions. It might be called interstitial meningitis, while the acute and sub-acute belong to the class of exudative inflammations.

The dura mater has an external and an internal layer, the external is intimately connected with the inner surface of the calvarium. Inflammation of this layer is called *pachymeningitis externa*, and is marked by thickening, opacity and localized adhesions of the dura with the skull.

Pachymeningitis interna is an inflammation of the inner surface of the dura mater, and may be acute or chronic; it is usually localized. Chronic pachymeningitis interna often results in the formation of flat, oval, laminated sacs containing blood which lie between the dura and pia mater, and are called *hæmatoma* of the dura.

ACUTE MENINGITIS.

Acute meningitis is also called *simple meningitis of the convexity* (from its usual site), *cerebral fever*, and *arachnitis*. When the unqualified word meningitis is used, acute, non-specific inflammation of the pia mater is understood.

Morbid Anatomy.—The inflammatory process may be established in any portion of the pia mater; but it usually involves the hemispheres. Only in rare cases is it confined to the base of the brain. There is always more or less thickening and redness of the membrane, and it loses its glistening appearance. Around the vessels where the connective-tissue is most abundant, there is first a slight serous effusion, which is quickly followed by cellular infiltration. An opaque zone of exudation surrounds the vessels. In mild cases the exudation is limited to the perivascular lymph spaces.

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[1] *Handb. d. Hist.*—Stricker.

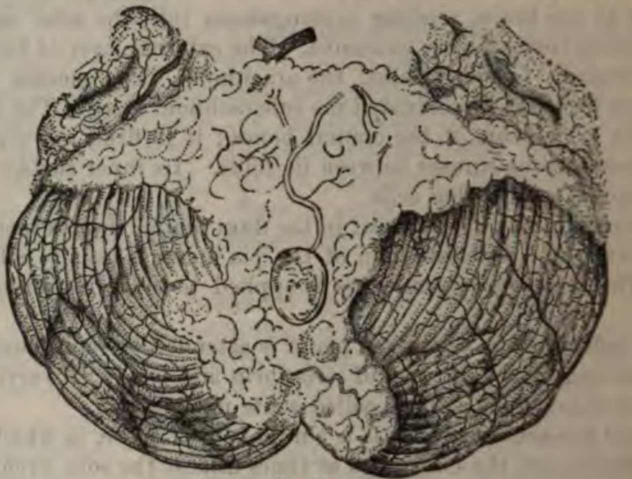

FIG. 193.
Acute Meningitis.
Portion of the under surface of the Brain, including the middle lobe of the Cerebrum and the Cerebellum.
The opaque exudation is seen upon the cerebral meninges completely covering the pons and surrounding the medulla. The basilar artery is dimly seen through the exudation.

The character of the exudation varies ; it may be serous or sero-purulent, and occupy the deeper meshes of the pia mater, or a thick puro-fibrinous exudation may cover the convexity of the brain as a firm membrane or a creamy diffluent mass.[1] This layer of fibro-pus or false membrane is thickest and most abundant in the fissure of Sylvius and the sulci and along the vessels, which are visible as red, inosculating lines in a yellow-green or distinctly green mass. On raising this layer the brain beneath is found studded with minute hemorrhages from the ruptured vessels connecting the pia and gray substance. The *white substance* may also show, on section, numerous puncta vasculosa. The convolutions are flattened, the sulci deepened, and the ventricular cavities will contain very little, if any, fluid.[2] When the cerebellar pia mater is involved, as in young subjects,[3] the roots of the cranial nerves about the medulla are sheathed in the exudation, and there is inflammatory infiltration of the neighboring plexuses.

Etiology.—Meningitis of the convexity is most frequent in early adult life and in young children. It is more common in males than in females. Acute alcoholismus and prolonged and intense mental anxiety and grief are among its predisposing causes. Injuries of the cranial bones, as fractures, severe blows, or punctured wounds are the most frequent exciting causes. Disease of the cranial bones and suppurative inflammation of the middle ear may induce meningitis, and inflammation of the dura mater is often complicated by simple meningitis. Diabetes, and irritation caused by cerebral *tumors*, or suppuration in the eyeball, and large carbuncles about the cranium, have caused it. In many cases of apparent idiopathic meningitis it is probable that an infection whose origin has been overlooked is the direct exciting cause.

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[1] Klob is of the opinion that the *pus is furnished in great measure from the arachnoidean epithelia.*

[2] Huguenin states that in meningitis of unknown origin the pia mater is often so adherent that it tears away portions of the brain substance when it is removed.

[3] Beduar.

Meningitis occurs as a complication in certain diseases—as measles, small-pox, scarlet fever, ulcerative endocarditis, Bright's disease, acute croupous pneumonia, typhus, typhoid fever, diphtheria, pyæmia and rheumatism. Acute alcoholism is said to induce meningitis in children.[1] Long-continued exposure to intense heat of the sun under conditions favorable to the development of miasmatic contagious diseases may result in meningitis. Finally, meningitis is sometimes idiopathic.

Symptoms.—The symptoms of meningitis may be divided into the three stages of *headache*, *delirium*, and *coma*. Premonitory symptoms, such as general malaise, wandering pains in the head and limbs, irritability, insomnia and a sense of impending trouble, are all indefinite "prodromata." It may be ushered in by a distinct *chill* or by repeated rigors.

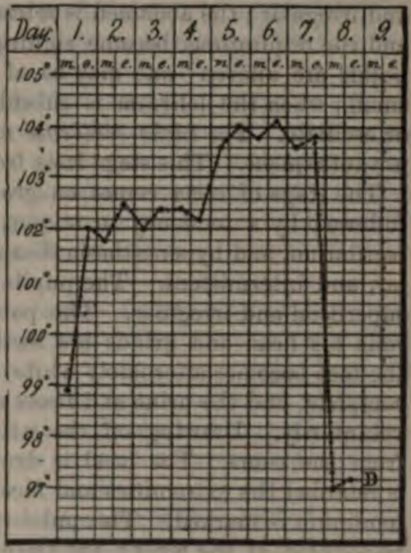

Fig. 194.
Temperature Record in a case of Acute Meningitis.

Usually the first prominent symptom is intense and persistent headache, localized in the frontal, temporal, or occipital region, which increases in severity from hour to hour. In severe cases the headache is diffuse, although it may extend obliquely across the head, or shoot from temple to temple. Accompanying the headache there is vertigo, intense photophobia, loud ringings in the ears, nausea, and *projectile vomiting*. There is cutaneous hyperæsthesia, and convulsive movements of certain groups of muscles. The upper and lower extremities, and the posterior cervical muscles suffer most. The face is rarely flushed, but has a pale, anxious expression. The conjunctivæ are injected, and the pupils are contracted and respond slowly to light. The degree of contraction of the pupil varies considerably in different patients during the twenty-four hours. It may only be contracted on the *same side* as the meningeal inflammation. Aphasia has been noticed as occurring in a few cases; loss of co-ordinating power is a marked symptom, so that the individual has an unsteady, tottering gait. The temperature, which rises rapidly, reaches 103° or 105° F., rarely higher; it may have a morning remission and evening exacerbation.

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[1] Ramskill states that in some instances when impetigo and eczema about the face and head suddenly disappear, the symptoms of acute meningitis of the convexity are developed.

In those cases which are rapidly fatal there is continuous high temperature. The pulse is firm, hard, wiry, and small, varying in frequency with the temperature range. The bowels are constipated and the abdomen is retracted. In old people the stage of headache may pass unnoticed, and the delirium first attract attention. In children general convulsions may be the initiatory symptom, with marked strabismus from its very onset. If constipation is not present the discharges are scanty and offensive. In this class of cases the first stage is always preceded by peevishness and irritability, which lasts from a few hours to two or three days.

In adults the stage of *delirium* is ushered in by an increase in restlessness, jactitation, irritability, and mental confusion. It is sometimes wild, simulating acute mania; at first it may be present only at night, coming on with the evening rise in temperature. In the aged the delirium is typhoid in character, or marked by incessant talking. Sometimes the aged are only lethargic and stupid. In adults muscular twitchings of the extremities and face are present in this stage; the eyeballs roll about vaguely, the flexor muscles are often powerfully contracted in one or both limbs, and there may be opisthotonos or even hemiplegia. Paresis of the thoracic and faucial muscles causes dyspnœa, irregular respiration, and dysphagia. In metastatic meningitis delirium may be the first symptom, and simulate delirium tremens, or the patient may be absolutely *mute*. The result, if death does not occur, is sometimes permanent insanity.[1] As this stage advances the temperature rises to 104° F., the pulse becomes more frequent and irregular; the abdomen is retracted, the vomiting continues *projectile*, and the respirations become sighing in character. During this stage the pupils are uneven—one may be of normal size while the other is quite small; when the delirium is subsiding they dilate and contract by turns, or oscillate. The *tâche cérébrale* may appear, and herpetic eruptions are not infrequent. This stage lasts from one to three days.

The stage of *coma* comes on gradually. The delirium subsides, and is followed by a tendency to stupor, lethargy, and deep sleep. Headache, jactitation, and hyperæsthesia disappear. The pulse becomes slow, irregular, and intermittent. The pupils are markedly dilated, the breathing is superficial and irregular. The patient is insensible to all impressions; he rolls his head and grinds his teeth, picks stupidly at the bedclothes, and the face becomes alternately white and red. Gradually the coma becomes complete, and the urine and fæces are retained, or the latter are passed involuntarily. Drawing off the urine may temporarily rouse the patient from the coma. The head is drawn to one side, and as the circulation is retarded the extremities and face are often of a purple hue. Subsultus tendinum is marked. The pulse runs up to 160 to 170, or until it cannot be counted at the wrist; the *Cheyne-Stokes*' respiration of ascending and descending rhythm becomes established. The expirations are puffing. The body is bathed in cold sweat, and death results from central paralysis, causing asphyxia, or heart-failure and pulmonary œdema. The temperature may rise very high or fall to a subnormal just before death.

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[1] Vigla, *Actes de la Soc. Méd. des Hôp. de Paris.*

Differential Diagnosis.—Acute meningitis may be confounded with *acute uræmia, typhus fever, variola,* and *delirium tremens.*

In *uræmia* the face will be turgid, and there will be puffiness about the eyelids; in meningitis the face is pale and anxious, and there is no œdema. In uræmia the urine will contain albumen *and* blood or exudative casts; in meningitis only a small amount of albumen is present and no casts. Convulsions, preceding the coma, are far more common in uræmia than in meningitis. The pulse, temperature, and the subjective symptoms of meningitis are absent in acute uræmia. In some cases only a microscopic examination of the urine will enable one to make a differential diagnosis.

In *typhus fever*, although the cerebral symptoms closely resemble those of meningitis, the temperature range is higher, often reaching 106° to 107° F. The pulse is more rapid and compressible in typhus than in meningitis. In typhus the countenance has a dull, leaden, or mahogany hue; in meningitis it is pale and anxious. Surface sensibility is blunted in typhus, and is exaggerated in meningitis. Vomiting is infrequent in typhus, in meningitis it is persistent and projectile in character. The characteristic typhus eruption appears on or about the fifth day; there is no characteristic eruption in meningitis. In typhus the pupils are equal, in meningitis they are unequal.

In *small-pox* the face is flushed, the pulse is full and bounding, there is intense pain in the back and loins, the vomiting is retching in character, and at the end of the third day the characteristic eruption occurs along the roots of the hair. These symptoms are all absent in meningitis. In many cases, however, it is necessary to await the appearance of the *eruption* before a diagnosis can be made.

In *delirium tremens* there is a busy delirium, the patient imagines persons and animals about him, and is wild in his gestures and utterances; in meningitis the delirium is incoherent and milder, but marked by a desire to get out of bed. The surface is bathed in a profuse, clammy sweat in delirium tremens; it is hot and dry in meningitis. In delirium tremens the temperature, pulse-rate and pupils are normal; there is no headache.

Prognosis.—The prognosis in acute meningitis is very unfavorable; severe cases terminate fatally, mild cases may recover. The duration varies from two days to four weeks; fatal cases rarely last more than eight days. If recovery takes place convalescence may not be fully established before the third week. The average duration of the disease is about eight days. Strabismus, hiccough, and local paralyses are very unfavorable symptoms. The prognosis is better in children than in adults.

Treatment.—The most important local measures at the onset of the disease are local blood-letting and the application of cold to the head. Leeches may be applied to the nuchal region or temples. The best method of applying cold is by means of the ice-bag or coiled tube, and that it may be thoroughly applied the head should be shaved. The patient must be placed in a large, quiet, darkened room, with the head elevated and all obstruction to the return circulation removed. The bowels must be freely acted upon by croton oil, and some drastic purge and saline diuretic are indicated if the urinary secretion is scanty.

In the stage of *coma* blisters to the back of the neck are of service. To relieve the great restlessness which often precedes the stage of delirium, hydrate of chloral and opium may be given. Iodide of potash and mercury are strongly recommended in all stages of this disease, but I have never obtained any positive beneficial effects from their use. Strong broths and alcoholic stimulants, if indicated, may be administered throughout the whole course of the disease. The condition of the bladder and the bowels must be carefully looked after during the stage of coma, and especially in the meningitis of old age.

SUBACUTE MENINGITIS.

Subacute meningitis differs from acute in that it is always secondary, is of longer duration, and is attended by less active symptoms.

Morbid Anatomy.—Upon the convexity of the brain there is found a sero-fibrinous exudation, containing few pus globules, and fibrin only in small quantities. The effusion will occupy the meshes of the pia and the ventricular cavities. As a result of the effusion the convolutions are flattened and the sulci are deepened. Flocculi of lymph will be found most abundantly along the line of the vessels of the convexity, and the vessels of the pia mater will be more or less distended. The pia mater will be lifted from the surface of the brain, will lose its glistening appearance, and in old cases will be slightly opaque.

Etiology.—This form of meningitis may occur during the course of any exhausting disease, as chronic diarrhœa, cancer, chronic Bright's disease, typhoid fever, etc.

Symptoms.—Being subacute and always secondary, its early symptoms are obscure. The stage of *headache* is wanting or lasts only a few hours, and is never severe. The delirium, which is often preceded by extreme jactitation, comes on gradually and is always quiet in character, and is characterized by an attempt on the part of the patient to get out of bed and walk around the room; the patient in walking staggers and is apt to fall forward. The stage of delirium may last only a few hours. In most cases headache and delirium are rapidly followed by coma. Thus, if a patient with chronic Bright's disease, after a short delirium, passes slowly into a state of coma, there is reason to suspect the development of this form of meningitis.

When the active delirium of typhus fever becomes muttering in character, when the pupils dilate and the pulse becomes slow and irregular, subacute meningitis may be suspected. As the coma begins the respirations become sighing and puffing in character, and the urine collects in the bladder. As it deepens, the pulse, which has been slow, is accelerated and intermits; the expirations are short puffs, and the interval between expiration and inspiration becomes lengthened. The typhoid state is rapidly developed, deglutition becomes difficult, there is blueness of the finger-nails with cyanosis, and the coma ends in death, or is slowly recovered from and followed by a long convalescence.

Differential Diagnosis.—The history of the case will aid very much in its diagnosis. *Coma* being its prominent symptom, the differential diagnosis is from other forms of coma.

From coma of acute meningitis, of tumors, compression, concussion, or cerebral softening, the previous history is sufficient to distinguish it.

From coma of poison and narcotics it is differentiated by examination of the secretions and excretions and by the state of the pupil; thus, in lead poisoning the line on the gums, in alcoholic coma an examination of the urine, and in opium coma the *pin-head pupils* will at once decide.

Hysteria, epilepsy, and catalepsy are attended with such distinctive signs that they cannot be mistaken for sub-acute meningitis.

Prognosis.—The prognosis of sub-acute meningitis is determined by the disease with which it occurs. When it occurs with Bright's disease it is usually fatal, but when complicating typhus and other fevers it is frequently recovered from.

Treatment.—The chief indication is to remove the cause. In Bright's disease, elimination of urea, which is causing the meningitis, should be attempted. In typhus fever free ventilation, stimulants, and concentrated nutrition will remove the cause so far as possible. In most instances blisters applied to the neck will be followed by marked improvement, and not infrequently by recovery.

CHRONIC MENINGITIS.

Chronic meningitis is an interstitial inflammation of the pia mater, which causes thickening and opacity of the membrane. It may be limited to the convexity.

Morbid Anatomy.—The pia mater is thickened, callous, and opaque, and may be infiltrated with serum, pus, and new connective-tissue cells.[1] There is thickening of the walls of the blood-vessels of the pia, and of their branches which enter the brain substance. These last adhere to the surrounding brain substance, so that it is torn when the pia mater is lifted from the brain.[2] Attendant upon all forms of chronic meningitis there is more or less *interstitial* inflammation of a very low grade of the *brain-substance* (diffuse interstitial encephalitis), although meningitis chronica is not ordinarily diffuse, but limited in extent. When the pia and dura mater are bound together, pachymeningitis has preceded the development of adhesions. The ventricles usually contain more or less fluid. The development along the falx of *glandulæ pacchionii* is due as much to senility as to chronic meningitis.[3] Atrophy of the cortex results from long-continued chronic meningitis.

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[1] Ramskill states that osseous plates have been found embedded in the thickened membrane.

[2] Some authors describe a degenerative change in the vessels resulting from previous peri- or endarteritis, which is said to resemble colloid metamorphosis. This change is rare.

[3] Foster states that he has seen cheesy degeneration of the exudation lying in the sulci, and that a cyst-wall of connective-tissue has formed.

Etiology.—Chronic meningitis is a disease of adult life, especially after fifty years of age. It is often idiopathic, and is met with among the poor and badly nourished. It is very frequently a complication of chronic alcoholismus, syphilis,[1] rheumatism, gout, and chronic Bright's disease (small granular kidney.) It is sometimes of traumatic origin, and is frequently found in the general paralysis of the insane.[2]

Symptoms.—The symptoms of chronic meningitis are always obscure. In some cases there are no symptoms; patients die of other diseases and meningitis is found at the autopsy. In others there are well-marked symptoms. The patient grows stupid, dull and apathetic, the mental faculties are blunted, and there is headache and a constant desire to sleep, or the patient may become morose and fretful. In the old the headache comes in paroxysms, and is attended by marked flushing of the face, frequent pulse and high arterial tension, occasionally attended by delirium. In nearly all cases vomiting becomes a prominent symptom, producing great exhaustion. Muscular weakness attends the decline in mental powers; the legs tremble in walking, control of the sphincters is lost, and the urine and fæces may be passed involuntarily. In many cases, chiefly the aged, there is paralysis of the bladder. The appetite is good, but digestion is slow and the bowels are constipated. The speech is thick, or the power to articulate certain words is lost. Vertigo, tinnitus aurium, and muscæ volitantes are present, with localized numbness or hyperæsthesia. The very aged often lie in a stupor, exhibiting no mental or physical signs of life except to breathe and take food.

There is no typical temperature range. Indeed, in many cases there is no pyrexia during the whole period of the disease, in others there will be a chill and fever resembling in its variations that of malaria. In some forms of chronic meningitis hemiplegia occurs, both with and without facial paralysis. When the trigeminus is involved the eyeball may slough.[3] Ptosis, strabismus, and variations in the size of the pupils indicate that the third nerve is involved. These irregular symptoms may continue with increasing severity for months or years. Toward the end convulsions may occur, and death takes place in deep coma.[4]

Differential Diagnosis.—The diagnosis of chronic meningitis is always difficult. It may be mistaken for *cerebral tumors* or *softening.*

With *tumors,* the headache is more intense and circumscribed, paralysis of nerves of special sense or of certain sets of nerves is common, and the signs of general decline in mental and physical power are less marked. Chronic meningitis appears in its whole symptomatology to be of a more *general* nature, while tumors produce *local* symptoms; speech and intellect are usually unimpaired. When neoplasiæ excite chronic meningitis in their vicinity the diagnosis is often impossible.

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[1] Virchow states that when constitutional syphilis exists in the human body, its localization depends greatly upon previous states of the system.

[2] Calmeil.

[3] See Effect of cutting fifth cranial nerve, in M. Foster's *Physiology,* p. 381 to 382.

[4] Many attribute the symptoms of chronic meningitis occurring in the insane, epileptic, and imbeciles to a cortical encephalitis frequently found at the autopsy, with the changes of meningitis.

Softening of the brain is also often associated with chronic meningitis. In softening there may be a history of a previous apoplectic seizure; muscular contractions are common, and the headache is not so severe as in chronic meningitis. In chronic meningitis there is more mental excitement than in cerebral softening, and the hemiplegia is more complete after apoplectic seizures.

Prognosis.—The prognosis is bad; the disease is a progressive one. Only in cases of a syphilitic origin can we offer any hope of recovery. Many become permanently insane, and some die from inanition. Death may also result from exhaustion, from cerebral pressure, and from complicating diseases, as pneumonia, etc.

Treatment.—The patient is to be kept quiet mentally, and the diet must be fluid and nutritious; stimulants being only administered to sustain vitality. The bichloride of mercury and iodide of potassium, in small doses, are the remedial agents most frequently employed. Blisters may be applied to the back of the neck, or a seton introduced there. The bowels need careful attention. The urine, if not passed freely, should be drawn and the bladder washed out.

TUBERCULAR MENINGITIS.

(*Acute Hydrocephalus.*)

Tubercular meningitis[1] is an inflammation of the basal pia mater, caused by an irruption of gray, miliary tubercles, and occurring most frequently in children.

Morbid Anatomy.—The dura mater is rarely involved. The pia mater in some cases is congested, in others pale, and it may be infiltrated with serum, fibrin, and pus. The vessels along the Sylvian fissure and the anterior peduncles of the cerebellum are studded, especially at their bifurcations, with *miliary tubercles*, which lie in the perivascular lymph-spaces. The lymph-spaces may become filled, and a covering be thus formed over the vessel; or the miliary granules dot the vessel like a string of beads. As they develop they compress and finally occlude the vessels. These granules are sometimes met with in small numbers on the convexity and along the longitudinal fissure, but are always more abundant at, if not confined to, the base, from which they may extend to the pia mater of the cervical cord. Even at the base they may be few and limited, or so abundant and extensive as to obstruct the circulation and impair the walls of the vessels, causing multiple hemorrhages and red softening.

The inflammatory exudation at the base is a turbid, serous effusion, or more commonly a thick, yellow, semi-plastic layer which extends from the fossa Sylvii, where it is most abundant, to the inferior surface of the cerebellum. When the pia mater of the *convexity* is involved it may show no evidences of exudative inflammation, but present a bright, rosy hue. The ventricles are distended by a serous fluid, usually turbid from admixture of cellular elements, which flattens the convolutions and causes oedematous softening. The membrane of the ventricles is thick and opaque. In cases running an acute course, the only lesion except the tubercles may be a large serous effusion into the ventricles. In most cases tubercular meningitis is associated with general tuberculosis.

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[1] Is also called by the Germans *basilar meningitis*; and by the English *acute hydrocephalus*. The disease was properly named tubercular meningitis by the French.

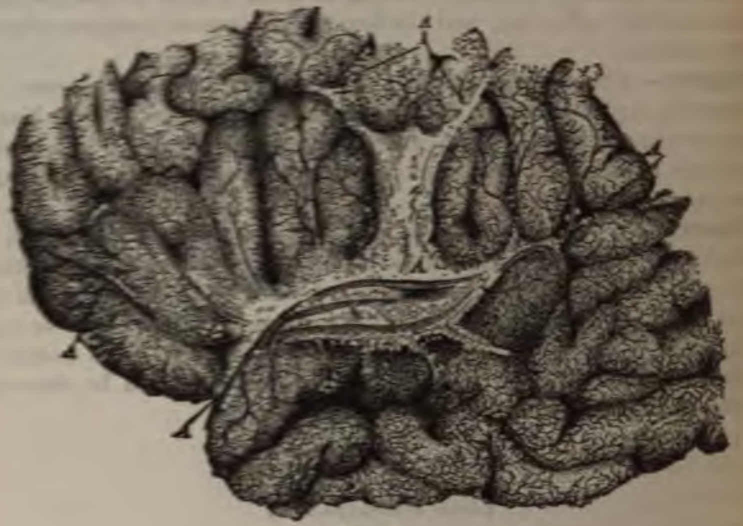

FIG. 195.
Tubercular Meningitis.
Region of the Sylvian fissure showing at A, A, miliary tubercles along the line of the blood-vessels.

Etiology.—Tubercular meningitis is rare before the first and after the fifth year. It occurs almost exclusively in children of a scrofulous diathesis, either inherited or acquired. In such children the tuberculous process is latent, and any debilitating disease may excite it, such as diarrhœa, the exanthemata, especially measles, whooping-cough, otorrhœa, and skin and scalp diseases of a chronic nature, dentition, insufficient or improper food, and injuries to the head, more particularly at the base of the brain. The immediate cause is the same as in all forms of tuberculosis. The bacilli may be carried to the brain from any tubercular focus or from sites of non-tubercular suppuration.

Symptoms.—The symptoms of tubercular meningitis are due not so much to the tubercular developments, as to the exudative products of the inflammatory process, as the pia may be studded with tubercles both at the convexity and base without the slightest symptomatic indications of their presence. Its advent is generally very insidious; if convulsions usher it in, its course is rapid.

The premonitory stage is marked by changes in the nutritive and digestive processes; the appetite is diminished and capricious; the breath is offensive, the tongue coated, constipation and diarrhœa alternate. The child becomes dull, languid, apathetic, and desires quiet; its sleep is restless and troubled. The face assumes a dull, anxious appearance, and

; play the child will stop suddenly and rest its head on its hands or r. During the progress of these symptoms a cachexia is developed re is progressive emaciation, which in connection with the etiology se to the suspicion that the disease is developing. These prodro- ay last from a few days to a month, and will often attract the n of strangers before they are observed by the parents. In most es there is a slight evening rise in the temperature and a short ; cough at intervals. The symptoms may be divided into three

first is the *irritative stage* or *period of invasion.* In this period the tients dislike to be disturbed; light and noise annoy them; they questions intelligently, but unwillingly and slowly. The expression ountenance is anxious. The cheeks will be alternately flushed and Headache, which is often severe, is paroxysmal and seldom constant. ually frontal and may be accompanied by dizziness. In some cases rmissions are short and the pain is diffused. The sufferer will moan sp the head with his hands. In young children pressure on the fon- increases the pain. There is mild delirium alternating with dis- and fitful sleep, from which the child starts with a piercing *hydro- cry.* They grind their teeth, roll their eyeballs, and the facial are contorted. The hands will be clenched and the thumbs flexed palms. Muscular tremors pass over the face and body in quick suc- The abdomen is retracted and hard, due to contraction of the es from central irritation. Projectile vomiting occurs at varying ls, independent of the ingestion of nd resists all treatment. The bowels stipated. The tongue is dry, covered white coating, and the tip is red. pils are contracted, there is photo- and possibly strabismus; the con- æ are injected and the brows con- . On drawing the finger firmly over n, a red line, after a moment, will the removal of the pressure. The ; temperature is a degree or two than that of the morning, but rarely bove 103° F. Sometimes, in cases convulsions usher in the disease, the ature will reach 104° or 105° F. The ations and remissions are always ar. The pulse is at first full, com- le and regular, or it may be normal in er. After the first twenty-four hours s a marked tendency to become accelerated and slightly irregular uscular exertion or excitement. The respirations at times are rapid egular, at others slow and regular, during sleep perfectly natural. At wsiness alternates with periods of excitement, but gradually becomes ersistent, and the child is disposed to sleep constantly unless aroused.

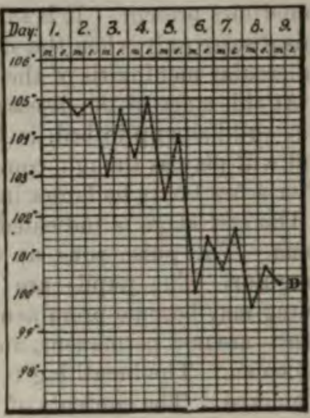

FIG. 196.

Temperature Record in a case of Tubercular Meningitis. The disease was ushered in by convulsions, and terminated in death on the ninth day.

Treatment.—It is unnecessary to refer to all the different measures which have been resorted to for the cure of this disease, for they have all failed. Prophylaxis alone is effective. A child whose antecedents lead us to fear the advent of acute hydrocephalus should be given a healthy wet-nurse from its birth, and the greatest care exercised as to its hygiene and diet for the first few years of life. Children who exhibit the premonitory symptoms, and in whom its development is feared, should be given cod-liver oil, kept out of doors as much as possible, and placed under the most favorable hygienic conditions possible; a frequent change of surroundings and of climate is important.

The treatment after the disease is established is almost entirely symptomatic. The bowels are to be kept open, and absolute quiet enjoined. Ice-bags may be placed on the head; but depletion of all kinds is contra-indicated. Iodide of potassium (pushed to toxic effects),[1] the mercury salts, and soda phosphate have been advocated, but their utility is questionable. I have obtained the greatest benefit from opium and bromide of potash during the stage of excitement, to relieve the restlessness and jactitation. I have never found that any of the plans of treatment proposed for this disease have any power to arrest its progress.

Tubercular meningitis *in the adult* is of rare occurrence, and is found only in connection with general tuberculosis. The pathological changes are the same as those found in children, and its etiology is identical with that of general tuberculosis. When latent, it may be excited by severe mental emotions or over-work. Symptomatically, it differs from infantile hydrocephalus in degree rather than kind, the symptoms being perhaps less violent. In all other respects the description just given will apply in all points to the same disease in adults.

CHRONIC HYDROCEPHALUS.

Chronic hydrocephalus is a cerebral dropsy from some cause not well understood. It is divided into *external* hydrocephalus, in which the serous effusion is in the meshes of the pia mater, and *internal* hydrocephalus, where the accumulation of fluid is in the ventricular cavities. When both coexist, it is called *mixed* hydrocephalus. Chronic hydrocephalus may be intra-uterine or extra-uterine.

Morbid Anatomy.—The essential lesion of chronic hydrocephalus is a serous effusion either into the ventricles or upon the surface of the brain. The fluid consists chiefly of water, containing albumen, sodium chloride, traces of lime and potash salts, epithelial and blood cells, rarely scanty lymph-flocculi and urea.[2] The ventricles may contain from twenty to thirty pounds of fluid.[3] The upper wall of the lateral ventricles may be ruptured; the brain substance is either softened or abnormally tough and resistant. The brain will be enlarged, and the convolutions flattened. In congenital hydrocephalus the bones of the skull are thin, the fontanelles and sutures are enlarged ; or, if united, numerous ossa triquetra are found between them ; and the supra-orbital, temporal, and occipital regions are distinctly depressed. The head may measure from eighteen to forty inches in circumference. The large ganglia at the base are pressed downward. The optic chiasm is flattened, and the pons and cerebellum are compressed ; the various septa and commissures are thinned or ruptured ; and, finally, there may be left only a thin layer of brain matter together with the thalami and corpora striata. When the lateral ventricles are distended and the ependyma thickened and granular, the term *chronic* or *granular ependymitis* is given ; in this condition there may be new tissue formation, and bands of cicatricial tissue join the walls of the ventricles.

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[1] Niemeyer.

[2] *Bright's Reports*, vol. i., p. 433.

[3] Trousseau (*Clin. Med.*) mentions a case in which the amount was fifty pounds.

Hydrocephalus *ex vacuo* is the result of defective development of, or atrophic degenerative changes in, the brain ; the space thus left is filled by a serous, sometimes slightly bloody fluid, seldom in great quantity (hydrocephalus senilis). The membranes (in the very young) are seen studded with ossific granules. If the bones should unite, there is subsequent thickening, and the head is either unsymmetrical, or nearly globular.[1] It is not uncommon to find evidences in the membranes of acute or sub-acute inflammatory processes.

Etiology.—Hydrocephalus may be congenital or acquired. When acquired it usually appears before dentition. A few cases occurring in old people are mentioned by Watson[2] and Golis, and Dean Swift is said to have died of it in the seventy-eighth year of his age ; atrophy or imperfect development of the brain causes it ; and it may arise from chronic passive hyperæmia, weakness of the vascular walls, from compression of the veins, occlusion of one or both lateral sinuses, or the presence of tubercular masses *in* the brain-substance. Rickets and syphilis in children, and dementia and alcoholismus in adults, are regarded as causes. It is met with in tubercular and scrofulous subjects, and it is said to have followed measles and scarlet fever.[3] Inflammatory changes in the ventricles and ependyma are accompanied by hydrocephalus. One hydrocephalic child renders it probable that subsequent children will be hydrocephalic.[4]

Symptoms.—The symptoms vary with the rapidity of its development. If intra-uterine, hydrocephalus develops rapidly ; the head becomes so large that its delivery can only be accomplished by operative procedure. If such children are born alive they die within a few days. In those cases where the disease is slight, the child at birth appears healthy, but after a few weeks the head begins to enlarge ; the sutures do not close, and the fontanelles are persistent ; the forehead bulges so that it overhangs the face, which is pale, small and weazened, giving a dwarfish expression to the child. The limbs do not develop ; the abdomen is distended and tympanitic, and the skin dry and scaly. Fluctuation[5] may sometimes be obtained between the anterior and posterior fontanelles. Often the child is unable to hold its head erect even for a few moments; the pupils are dilated and the eyes protruded. There are periods of apparent improvement, but after a year of gradual decline death occurs in convulsions, from starvation or intercurrent disease.

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[1] Barthez and Rilliet state that in a few cases of congenital hydrocephalus the bones were normal.

[2] *Practice of Physic* ; Sir Thomas Watson.

[3] Tanner's *Practice*.

[4] Hoppe, Niemeyer and others regard hydrocephalus arising from nutritive changes in the capillary walls as analogous to "skin inflammations that produce blebs."

[5] Sir T. Watson ; Dr. Bright.

In another class of milder cases the mental faculties are normal, but nutrition is imperfect; the limbs are small and the muscles flabby. The children are irritable, and at times have fever, nausea and vomiting. After an unusually severe attack of fever they may gain flesh and seem much improved, but the head still increases in size. After a variable time of improvement they again become worse, lose strength, and all the active cerebral symptoms return. When they attempt to walk they totter, stumble and fall. Spasms, epileptiform convulsions and paralysis of certain groups of muscles follow, and they become idiotic. Such children do not die from hydrocephalus but from intercurrent disease. Some of these cases live for four or five years, having periods when they seem to be recovering. When anæmia and asthenia cause death the usual duration is a year.

A few rare cases are recorded where hydrocephalic subjects have lived five, ten, and even thirty years.

Differential Diagnosis.—Congenital, or intra-uterine hydrocephalus cannot well be mistaken for any other malady.

Cranial rachitis does not cause the mental, or even the physical, derangements induced by hydrocephalus; but it induces an unsymmetrical enlargement of the bones.

Prognosis.—The prognosis is always unfavorable. The average duration is one year. Death may result from *any* of the complications, from simple asthenia and anæmia, from meningitis, ependymitis, apoplexy, rupture of the fluid through the brain substance into the epicranial aponeurosis,[1] or from general paralysis. The only condition of recovery is a cessation of increase in the fluid and closing up of sutures; the cases of absorption of fluid and return of mental power are doubtful.[2]

Treatment.—There is very little to be done for this disease. The treatment which has been employed may be divided into external or mechanical, and internal or medicinal. Mercurial inunctions and strapping the head with adhesive plasters have been advised, but are of doubtful utility. Sudden compression of the head may cause death. Pale, flabby children bear it best.

Tapping can be advocated only in *external* hydrocephalus and where no inflammatory or organic changes coexist. The anterior fontanelle is the proper point to insert the aspirating needle; only a few ounces should be drawn at a time, the child being carefully watched during and after the operation. Subsequently the head should be lightly bandaged. Langenbeck passes behind the upper lid through the superior wall of the orbit and enters the anterior horn of the lateral ventricle. But inflammatory action is apt to be excited by any such procedure.

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[1] Rokitansky's case.

[2] Otto states, however, "that new cerebral matter may be deposited in place of re-absorbed fluid."—Rokitansky's *Pathological Anatomy*.

Internally cod-liver oil and the syrup of the iodide of iron and potash should be given throughout the disease. Calomel (gr. ¼–½) daily has been recommended, until purging becomes severe. The food, the hygienic surroundings and the clothing should also receive careful attention. Change of air is also highly beneficial. If rickets coexist phosphatic salts are indicated.

PACHYMENINGITIS EXTERNA.

Pachymeningitis is an inflammation of the dura mater which may be acute or chronic. The inflammation may involve either the external, internal, or both layers of the dura mater. When the external layer is primarily involved, it is called external pachymeningitis; when the internal layer is the primary seat of the inflammatory process, it is called pachymeningitis interna. External pachymeningitis is almost always a secondary inflammation.

Morbid Anatomy.—In the non-suppurative form of pachymeningitis externa the dura is injected, softer than normal, and covered with ecchymotic spots. New connective-tissue formations occur, which lead to thickening and induration of the dura and adhesions between it and the cranial bones. Numerous pigment granules stud the thickened membrane. Osteophytes form and the appearance closely resembles periostitis with exostosis. In many cases bony flakes can be detached from the tough, pale, leathery dura mater.

In purulent pachymeningitis externa suppurative processes are early established and the external layer is softened, disintegrated, thinned, and rendered very friable. A thick layer of new connective-tissue separates the pus from the *internal* layer of the dura mater. These purulent collections are usually of traumatic origin and circumscribed, as inflammation of the dura is rarely diffuse, and the pus detaches the dura from the bone and may lead to necrosis. When the sinuses are involved in pachymeningitis their walls undergo thickening, the intima is roughened, and thrombi form at the seat of the lesion, which may break down and be absorbed or give rise to emboli or pulmonary infarctions.

In old age it is physiological for the dura to be thick, leathery, cartilaginous and of a dull white color. The sheaths of the arteries are thickened.

Etiology.—Idiopathic pachymeningitis externa is of doubtful occurrence. Secondary pachymeningitis may result from injuries to, and caries of the cranial bones or upper cervical vertebræ. Hemorrhage of traumatic origin may separate the dura from the bone and be followed by inflammation. Chronic internal otitis and suppurative inflammation of the orbit may lead to it. An external periostitis may be followed by external pachymeningitis without apparent intervening bone-changes. Inflammation in the venous sinuses, especially the transverse and petrous, may lead to it, especially when the thrombus formed undergoes suppurative changes.

Symptoms.—The symptoms of pachymeningitis are generally very obscure. After an injury of the skull or a chronic otorrhœa, we may suspect external pachymeningitis when there is somnolence, headache, dizziness, photophobia followed by delirium, and perhaps convulsions and coma. In cases attended by thrombosis of a sinus there will be hectic fever and rigors and symptoms simulating an attack of intermittent fever. When metastatic abscesses develop in the joints and internal organs, the headache will be severe and localized, and possibly attended by nausea and vomiting. If there is cerebral pressure, the pulse becomes slow and irregular, rarely *frequent* and *feeble;* the pupils are unequal; the headache, apathy, and somnolence increase and are attended by facial paralysis. Just before death the pulse slows and coma is developed. Circumscribed painful œdema behind the ear[1] and less fulness of the jugular of that side are indicative of thrombosis in the transverse sinus.[2]

Differential Diagnosis.—In one who has received an injury of the skull, with possibly fracture of the base, if the signs of cerebral compression persist, pachymeningitis externa may be suspected. With caries of the cranial bones or otitis interna, the diagnosis can be made from the complicating cerebral symptoms. But in chronic cases the symptoms are often so obscure that a positive diagnosis is impossible.

Prognosis.—Recovery is possible and depends largely upon the cause. Alcoholic pachymeningitis is almost invariably fatal. That due to otorrhœa may end favorably if the pus is evacuated either spontaneously or by operation. The great danger is in extension of the inflammation to the internal layer of the dura mater and to the pia mater.

Treatment.—The treatment is mainly surgical. Trephining may sometimes save life. Rest, a mild diet, a free evacuation of the bowels, cold to the head and warmth to the extremities are the principal means of treatment. Disease of the ear demands immediate attention. I recall a case where, after a deep coma of five days' duration, recovery unexpectedly occurred after a copious discharge of pus from the ear. Should symptoms of suppuration be well marked, alcoholic stimulants, quinine, and opium are indicated and the question of surgical interference will present itself.

PACHYMENINGITIS INTERNA.

Pachymeningitis interna may be acute or chronic.[3]

Morbid Anatomy.—In acute pachymeningitis interna the inner surface of the dura mater is intensely hyperæmic and covered with a layer of fibrin and pus which may be circumscribed or diffused. The substance of the membrane may be thickened by new connective-tissue developments; but the larger part of the inflammatory exudation is upon its free surface. This form of pachymeningitis is apt to be complicated by inflammation of the pia mater. In *chronic* pachymeningitis interna the dura is covered with a layer of organized tissue. This thin, filmy new membrane is very rich in large capillaries with thin walls. It is composed mainly of cells, having very little basement substance, and is usually most abundant at the convexity of the brain.

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[1] *Griesinger* calls this phlegmasia alba dolens *en miniature*.

[2] Gerhardt.

[3] Virchow was the first to interpret and classify the changes which take place in this inflammation

Some pathologists claim that before these changes occur, a thin layer of compact fibrin, which can readily be stripped off, occupies the site where subsequently a hæmatoma is developed. The capillaries in the new tissue are easily ruptured and hemorrhages are liable to occur, forming hæmatomata, ranging in size from small clots to large blood sacs covering the whole convexity.[1] After a time the walls of the new vessels become thicker. In rare instances the blood extravasates in small amounts and is absorbed, and only a thin, transparent well-defined membrane marks the spot where the pachymeningitis existed.

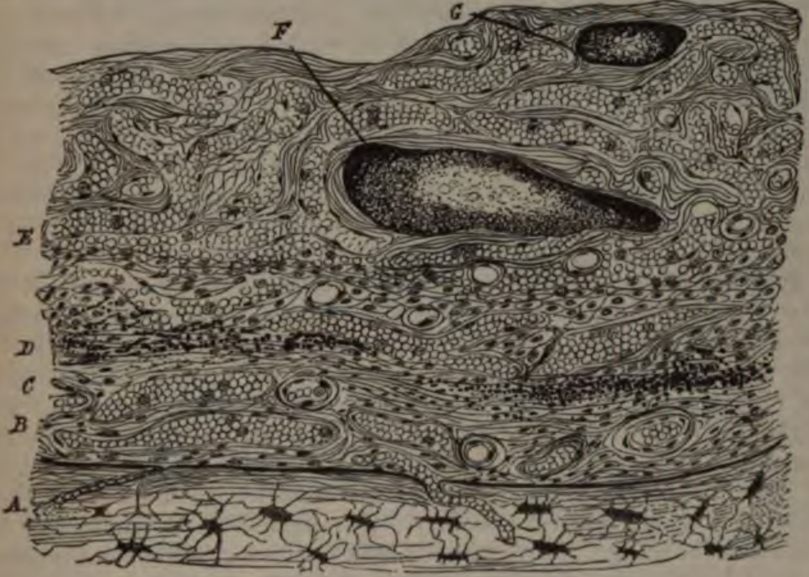

Fig. 197.

Pachymeningitis Interna.

Vertical Section of the Skull and Cerebral Meninges.

A. Section of the skull.
B. Dura mater thickened and connected intimately with C, the first layer of the inflammatory deposit.
D. Deposit of pigment.
E. Superficial layer of exudation containing an hæmatoma F, caused by rupture of the capillaries in the highly vascular tissue.
G. A second and superposed hæmatoma appearing in a new layer of exudation. × 250.

The hæmatoma may become encysted (Virchow's *hygroma of the dura mater*), or its contents may undergo caseous and calcareous changes.[2] In some cases the blood has either dissected between the layers of the wall of the hæmatoma, or else, after one hemorrhage, a new layer of pseudomembrane forms, and a second extravasation is followed by a second tissue formation. The ventricular cavities are sometimes filled with a sero-sanguinolent fluid.

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[1] In opposition to the above description, Huguenin states that a thick (one-twelfth inch) layer of fibrin forms on an *intact dura*. This, he says, is rarely demonstrable; the new, yellow-stained membranes, in which are colorless masses (? white blood corpuscles) of protoplasm, form later.

[2] Rokitansky and Forster.

Etiology.—Both acute and chronic pachymeningitis interna are usually secondary, but in rare instances are of idiopathic origin. The acute form may be secondary to pachymeningitis externa, pyæmia, Bright's disease, or the acute infectious diseases. Chronic pachymeningitis interna is a disease of advanced life, rare before forty, and most frequent between sixty and eighty. Chronic alcoholismus is its most frequent cause. Atrophy of the brain, hydrocephalus, cerebral tumors, and general paralysis and dementia are often followed by pachymeningitis interna. In progressive pernicious anæmia, hæmatoma appears in thirty-three per cent. of all cases.[1] Leucocythæmia, the hemorrhagic diathesis, scorbutus, and splenic anæmia are blood states especially liable to be accompanied by pachymeningitis. Valvular diseases of the heart impairing venous return and atheroma are important factors in its causation.

Symptoms.—The symptoms vary with its extent and the amount of the new tissue formation. When the disease is slight, there are no symptoms; when extensive, most of the symptoms are due to cerebral pressure. At first there is constant headache, dizziness, vertigo, tinnitus aurium, muscæ volitantes, photophobia, constipation, anorexia and insomnia, with slight febrile movements. The intellect is impaired, memory fails, and sometimes there will be a temporary loss of consciousness and partial loss of speech. The symptoms of slight extravasation simulate very closely those of a small cerebral apoplexy. The pupils will be contracted, one more than the other. There may be slight wandering, and when the attack is partially recovered from the mental and bodily conditions are palpably impaired. The temperature may be slightly raised and attended by irregular exacerbations and remissions. The pulse is slow, becoming irregular upon excitement.

Paralysis comes on gradually, as one hemorrhage follows another. Between the attacks localized headache is the prominent symptom. Sometimes slight epileptiform convulsions occur, followed by temporary loss of consciousness.[2] If the hemorrhage in the new tissue is rapid and extensive, patients may die suddenly from cerebral compression; or one slight hemorrhage which gives rise to few symptoms may be followed by a second more extensive bleeding, attended by the ordinary symptoms of apoplexy. Recovery after slight hemorrhage not infrequently occurs: but the patient afterward will be troubled with more or less constant *headache*, *insomnia*, and perhaps by localized paralysis. Moderate-sized hæmatomata have been found at autopsies, where, during life, *no signs* existed.

During the course of acute and chronic pachymeningitis the venous sinuses may become involved in the inflammatory process, causing thrombi which give rise to pulmonary or other infarctions, attended by the usual symptoms: rigors, followed by a temperature of 103° or 104° F., with irregular variations. The pulse at first is rapid, but after a few days it becomes slow. As the case approaches a fatal termination the pulse runs up to 120 or 140, and is small and feeble. The patient becomes delirious and rapidly passes into coma, preceded or followed by convulsions.

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[1] Huguenin.

[2] Pon states that pachymeningitis interna begins, often, with the symptoms of the general paralysis of the insane upon one side of the body.

Differential Diagnosis.—The diagnosis of pachymeningitis interna is always difficult; it may be confounded with *acute meningitis* of the convexity with which it is frequently associated, with *chronic meningitis,* and *softening of the brain.* The diagnosis of a hæmatoma is based on the following conditions, viz.: continued, vertical, localized headache, contracted pupils, strabismus or ptosis, very slight fever, *slow* pulse, a history of one or more apoplectic seizures, or of periods of loss of consciousness followed by dysphagia, facial paralysis or hemiplegia. The diagnosis is always problematical, complicating, as it does, so many cerebral affections, and its symptoms are masked and indefinite.

Prognosis.—The prognosis is bad, although the course of the disease is usually slow. The cerebral symptoms often intermit. Some die from the extension of the inflammation; others from rapid and extensive blood extravasation; some become insane or demented; the larger number die from intercurrent disease. When the venous sinuses are involved patients may die from the effects of the inflammation or from infarctions. The disease lasts, in most cases, from one to three weeks, yet one day and one year are given in a few recorded cases as the limits of this affection.

Treatment.—There is no cure for pachymeningitis interna: all that can be done is to treat symptoms. Absolute rest in a cool, quiet room is to be enjoined. Irritative or inflammatory symptoms demand cold to the head, mild counter-irritation, and heat to the extremities. The bowels are to be kept freely opened, and, at the onset, a brisk purge may be given. As the disease progresses stimulants and a highly nutritious diet are the best means to combat the affection; and anodynes may be necessary to induce sleep and relieve headache. Ergot is indicated on the ground of its physiological action on the vascular system, to prevent or diminish future hemorrhages. Ramskill advocates iodide of potash as the chief remedy, but this and mercury are rarely employed at the present day.

SYPHILIS OF THE DURA MATER.

Syphilitic gummata, developed in the dura mater and accompanied by meningitis, are met with in the advanced stage of syphilis.

Morbid Anatomy.—The lesions differ from the other forms of meningitis, in that the inflammatory product is circumscribed in the form of gummatous tumors, which are composed of small round, oval, and pyriform cells with basement substance. These gummatous masses may degenerate and become cheesy, or be converted into a purulent-looking fluid consisting of serum, degenerated cells, and granular matter. They may be developed either on the external or internal surface of the dura mater, and are usually multiple. Gummatous masses may develop in the substance of the cranial bones and cause more or less destruction of them, or it may be complicated by inflammation of the pia mater, and then gummy masses may develop beneath the pia mater.

Symptoms.—As in the varieties of pachymeningitis, persistent localized headache is the most constant and prominent symptom; convulsions and temporary loss of consciousness not infrequently accompany the headache. The intellect is impaired, and the patient lapses into a dull, stupid, apathetic condition. They may be wildly delirious. In some instances there is loss of sight and hearing. If the gummatous masses attain a large size, facial paralysis and hemiplegia may occur. I have known a patient with syphilis of the dura mater to become hemiplegic, pass into a state of complete unconsciousness, with stertorous breathing, relaxed sphincters, and dilated pupils, remain in this condition for ten days, and finally completely recover.

Differential Diagnosis.—The diagnosis rests entirely on the syphilitic history in one who has any of the external manifestations of syphilis associated with the cerebral symptoms of pachymeningitis.

Prognosis.—The natural termination of this disease is in death. If these patients are subjected to proper treatment before the gummatous masses have become too large or are too far advanced in degenerative changes, recovery is almost certain. Recovery, however, in these cases is rarely permanent, for after the treatment has been abandoned, the disease is apt to return. In one who is addicted to the use of alcohol the prognosis is very unfavorable.

Treatment.—The treatment is that of advanced syphilis. Mercury and iodide of potassium, either together or alternately, are the means to be relied upon. The mercury is best employed by inunctions and baths. My rule is to apply each day a drachm of strong mercurial ointment in the axilla and flexures of the joints until its specific effects are produced. Iodide of potassium must always be given in large doses; from thirty to sixty grains may be given in from four to six ounces of water, three or four times daily until the desired effect is reached, which is the disappearance of the cerebral symptoms. Tonics and cod-liver oil are always indicated, and of service between the periods of the administration of the mercurials and the iodide. Under no circumstances should this class of patients be allowed to use stimulants habitually in any form.

CEREBRAL THROMBOSIS AND EMBOLISM.

The cerebral arteries may be obstructed by emboli or thrombi, the cerebral veins and sinuses by thrombi only. The changes in, and effects produced by a plug in the cerebral vessels, whether embolic or thrombotic in its origin, are identical with similar changes in other parts of the body. The walls of a cerebral artery which is the seat of thrombosis are usually thickened. The thrombosis may be the result of slowing of the blood current from any cause. The results and symptoms of cerebral thrombosis are essentially the same as those of cerebral embolism.

Cerebral emboli may be bilateral; several may coexist; and they have been found in all of the cerebral arteries. The left middle cerebral is their most frequent seat (forty-six in one hundred cases); next the internal carotid, the basilar, and vertebral. Ninety per cent. are in vessels that supply the ganglia at the base. The artery of the corpus callosum is rarely implicated.[1] Embolism in the cortex is rarely attended by serious results, on account of the free anastomoses between the cerebral capillaries and those of the pia mater.

When the left middle cerebral artery is plugged, it being a terminal artery with no anastomoses, well-marked symptoms occur and destructive lesions follow. This artery has the most direct communication of any of the cerebral arteries with the left ventricular cavity. This anatomical fact readily explains the frequent occurrence of embolism in it. The result of cerebral embolism or thrombosis is to deprive the portion of the brain supplied by the obstructed vessel of its nutrition, in consequence of which it degenerates and softens. Softening of the brain is the usual result of embolism; the name embolic softening has been applied to it, to distinguish it from inflammatory softening.[2] Niemeyer describes the initial result of embolism and thrombosis as partial anæmia of the brain,[3] and states that the subsequent softening is analogous to gangrene in the extremities induced by obstruction or obliteration of the vessels. But the difference is, that within the skull the absence of exposure to air precludes decomposition.

If a large cerebral vessel, or a large number are obstructed suddenly, it may cause sudden death, and there will be no time for cerebral softening. Many writers include in the signs of cerebral embolism those of the first stage of softening.

Symptoms.—The symptoms produced by the plugging of cerebral vessels, either by emboli or thrombi, are sudden in their advent. When an artery of considerable size is obstructed there is temporary loss of consciousness, the patient passing rapidly into coma, from which he gradually recovers with complete hemiplegia. If only a small branch of a cerebral artery is plugged there may be only a slight and transient loss of consciousness or confusion of mind, or there may be nothing to indicate its occurrence except sudden loss of speech. During the period of loss of consciousness, if it occur, the face is pale and cold.

Aphasia is common, but not a constant attendant. It may be *complete* or partial, the patient may be able to use only one or two words, as "no" or "table," and employs them to answer all questions. Again, his vocabulary may consist of a number of words, but he cannot use them aright; he calls for his boots when he intends to call for bread.

Aphasia may be of two kinds, *amnesic* or *ataxic*. Amnesic aphasia is where the memory of words is lost, though the capability of uttering them may exist. Ataxic aphasia is where the muscles and parts that produce articulate speech cannot be co-ordinated ; the patient knows just the word he wants to speak but he cannot utter it. The *ataxic* form is commonly associated with hemiplegia of the right side.[1] In *ataxic aphasia* a patient can read, write, and listen intelligently to the speech of others. In *amnesic* he only reads and understands what others say. After repeated attacks of embolism a patient may have both amnesic and ataxic aphasia.

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[1] It is interesting to note that the *vertebral* was oftenest involved in the large number of cases seen by Nothnagel.

[2] Nothing can be definitely stated concerning embolism and thrombosis of the capillaries from pigment (in malaria), leucocytes (in leukæmia), and from fatty granules or salts. They are more pathological curiosities than well-defined diseases.

[3] The only other form of partial cerebral anæmia is from collateral œdema about spots of extravasation.

Within twenty-four hours after the occurrence of a cerebral embolism there may be convulsive movements in the muscles which are afterward to be paralyzed ; epileptiform convulsions frequently occur. If the patient pass into coma he may continue in a comatose state and die within a few days ; or he may recover from the coma with permanent hemiplegia and aphasia. There may be temporary improvement in the hemiplegia, but after the degenerative changes take place in the brain the hemiplegia becomes permanent. In most of the cases where the hemiplegia is permanent the paralyzed muscles become contracted. When the hemiplegia and aphasia are partial, and there has been no loss of consciousness, complete recovery generally takes place. A cerebral embolism may be so slight that there may be aphasia for a few days, and then the patient will completely recover without any other symptom.

Hemiopia and unilateral amaurosis with alternate hemiplegia are symptoms of cerebral embolism ; they are due to extravasations into the optic nerve and to embolism of the arteria centralis retinæ on the corresponding side. Embolic amaurosis may precede cerebral embolism for a few days. The ophthalmoscope shows pallor of the papilla and absence of pulsation in the retinal arteries. Even when the middle cerebral is alone occluded collateral fluxion may cause arterial and venous hyperæmia of the retinal vessels and congestion of the optic disc. If the patient does not begin to improve in twenty-four or forty-eight hours after embolism occurs, a fatal issue may be expected. In such cases the temperature rises to 104° F., remains at that point for a couple of days, and then rapidly declines.[2] There are often evidences of embolism in other parts of the body, as the spleen, kidneys, extremities, etc., etc., which will aid in the diagnosis.

Bilateral embolism usually results from separate attacks. These cases are marked by epileptiform convulsions, temporary aphasia, hemiplegia, or rarely double hemiplegia, accelerated and irregular—perhaps difficult—respiration, unilateral anæsthesia of the conjunctiva, and normal sensibility of the cornea.

Differential Diagnosis.—Cerebral embolism and thrombosis often cannot be positively distinguished from *cerebral hemorrhage*. The symptoms in some cases are the same. If the hemiplegia is upon the right side and there is aphasia the probabilities are in favor of embolism. If there is no aphasia, and the loss of consciousness is prolonged and the facial paralysis is marked, cerebral apoplexy has occurred. If the paralysis is rapidly recovered from, it indicates embolism and not apoplexy.

Cerebral thrombi form in old age without cardiac or pulmonary disease, on account of rigid, calcified and atheromatous arteries; the paralysis is less marked, and aphasia is usually incomplete. If the paralysis improves after a day or two and then gets worse, it indicates embolism.

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[1] Ogle states that in left-handed individuals the centre for language is in the right island of Reil.

[2] Bourneville.

Prognosis.—The prognosis will depend upon the size of the artery plugged; complete recovery is always possible, partial recovery is not infrequent. Still, cerebral embolism and thrombosis are serious conditions, on account of the danger that they will lead to cerebral softening. The prognosis is usually better in those cases where the hemiplegia is partial than when it is complete. In cases where the symptoms at first are mild, but gradually grow worse, the prognosis is unfavorable. It is impossible to determine the extent and duration of the paralysis which sometimes continues after its occurrence. Chronic visceral diseases, senility, and debility or anæmia render the prognosis unfavorable. The occurrence of coma is very unfavorable. Even after rapid disappearance of the paralysis and aphasia there is great danger of another attack.

Treatment.—The plan of treatment in cerebral embolism and thrombosis is a tonic and stimulant one. No depletory or revulsive measures are ever admissible. The action of the heart, and the constitutional appearance of the patient must determine whether alcoholic stimulation is to be resorted to or not. In cases of coexistent cerebral hyperæmia, *local* depletion may be of service. The hemiplegia is to be treated the same as in cerebral apoplexy. Iron, cod-liver oil, and a tonic plan of treatment should follow the disappearance of the paralysis.

CEREBRAL SOFTENING.

Embolism and thrombosis are undoubtedly the most frequent causes of cerebral softening. But I shall adopt the view that there are several varieties of very different causation and anatomical changes, and shall follow the usual classification of *red*, *yellow*, and *white* softening, although this division is somewhat arbitrary and unsatisfactory.

Morbid Anatomy.—*Red softening* is marked by punctate redness or by numerous minute capillary apoplectic foci, with fatty degeneration of the nerve cells and fibres. The pultaceous spot is deep red, shading off into the neighboring brain-tissue with no distinct limit. There may be several of these red foci; as many as twenty have been found in different stages of discoloration and softening. In all cases the centres show most marked changes. There is more or less œdematous swelling of the adjacent brain-tissue, so that upon cutting into it the softened spot will rise above the plane of the section. The vessels are enlarged, often from proliferation of the endothelium, forming masses of varying sizes, frequently within the vascular lymph-sheaths, making a white rim visible on cross-section. There is proliferation of the cellular elements of the neuroglia, and the nerve-elements simultaneously undergo fatty changes. Few pathologists claim that any inflammatory exudation accompanies these changes. There is a débris in and about the focus, consisting of fat granules, altered blood corpuscles, and free nuclei, a few pigment-granules, shreds of brain-tissue, and large granular corpuscles.[1] The nerve fibres become macerated, and their white substance is coagulated and broken up into large masses. There is varicose hypertrophy of the axis-cylinders. This condition is called, by Hayem, *the cloudy swelling of Virchow.* A spot of red softening may become dry and shrunken, or cicatrization may occur. The phenomena of absorption consist in fatty degeneration and caseation, or the formation of a cyst by a process analogous to that described under the head of Apoplexy.

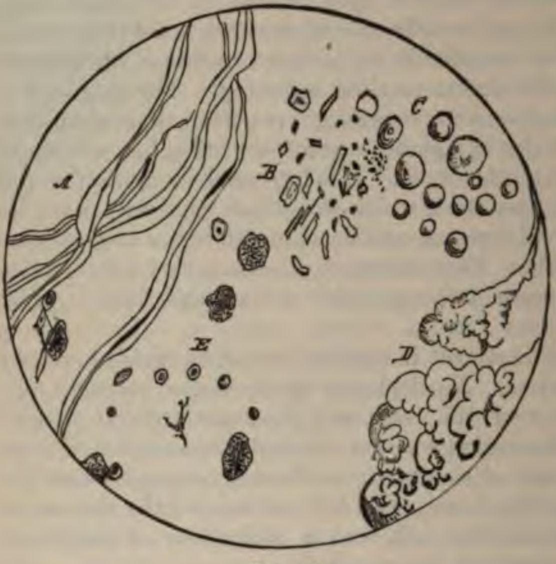

FIG. 198.

Cerebral Softening. Elements from an Apoplectic Focus in Red Softening.

A. Shreds of nerve fibres.
B. Altered blood corpuscles.
C. Fat spherules.
D. Masses of myeline.
E. Free nuclei from neuroglia. × 300.

Yellow softening is usually the result of partial cerebral anæmia from obstruction of the cerebral vessels. It may occur in any portion of the brain, but most frequently has its seat in the *middle* or *posterior* lobes, and in the cortex or corpus striatum. Stasis is accompanied by all the changes described as occurring with an infarction which is followed by fatty degeneration, and it may proceed very slowly or with great rapidity should hemorrhage fail to occur. The coagulated blood in the vessels undergoes a retrogressive change, the fibrin becoming granular. Fatty and granular matter in large quantities surrounds the infarction, which becomes dry and slowly contracts. Corpora amylacea, blood-pigment, and crystals from the altered fatty material are found. A soft, yellow-white mass—often of a sulphur color—is thus formed, varying in size from a hazel nut to an orange. The consistence is variable, but in typical cases it is a gelatinous, moist, and tremulous pulp. A stream of water will readily wash out the focus of softening. These degenerative changes proceed until the focus of softening becomes changed into a mass of reticulated fibres, in whose meshes is a milky fluid. The vessels in the wall of this cyst are covered by fat granules, and are empty or contain a yellowish clot; but their lymph-sheaths are irregularly dilated with pigment, fatty, granular and detached endothelial cells. In this form of softening there is usually a faint line of demarcation between the focus and healthy brain-tissue. The color of yellow softening is due to fatty changes and a deposit of blood-pigment. These foci may cicatrize similarly to apoplectic foci, or result in the formation of a cyst. This process may follow either an obstructive or hemorrhagic infarction. Red softening may also terminate in yellow softening,[1] from gradual absorption of the broken-down corpuscles and pigment matter.

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[1] Gluge's corpuscles are large spherical cells, filled with fat; they are abundant, dark by transmitted and bright by reflected light. The origin of Gluge's corpuscle, so prominent in yellow softening, is as various as that of pus-cells.

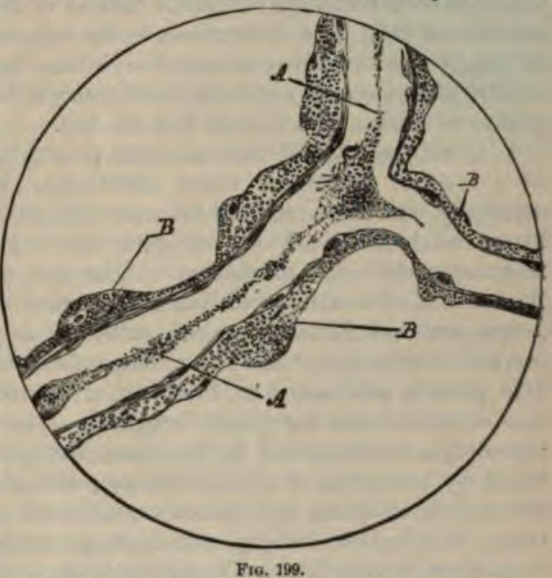

FIG. 199.

Cerebral Softening. Small Blood-vessel from a Focus of Yellow Softening.

AA. Lumen of the vessel containing the remains of a clot.
BBB. Irregularly dilated lymph-sheath containing granulo-fatty matter and detached endothelia. × 300.

White or *atrophic softening* is the form met with so frequently in old age.[2] It is white or resembles healthy brain-tissue; the process is a slow one. Hemorrhage or hyperæmia is rarely present. It is usually met with in the white matter of the hemispheres, and the degree of change may be so slight as to render its detection difficult, or it may be soft and diffluent The specific gravity of the softened mass is less than normal brain substance. White softening is never distinctly limited, but shades off into the adjacent tissue. In chronic softening of the convolutions their form is preserved, but they are markedly atrophied; over them the pia mater is more or less œdematous, and fills up to a certain degree the space caused by the atrophy. Should the vessels in or near a white softened patch be examined, they will usually be found atheromatous or the seat of endarteritis. Thus it is evident that the color has no relation to the pathological changes. Red softening may come from embolism; yellow, directly from thrombosis, embolism, or be a second stage of red; white may be primary, or secondary to yellow. In every case the cause is the primary pathological feature—the color is secondary.

Etiology.—Embolism and thrombosis are most frequently the causes of cerebral softening, especially in old age. It is essentially a disease of old age, for nearly all the predisposing causes of thrombosis are met with in advanced life. Thrombotic softening between thirty and fifty is rare. But embolic softening may occur at any period of life where the predisposing causes of emboli exist. Syphilis and chronic alcoholismus must be ranked next to embolism and age as causes. It has been shown that syphilitic disease of the arteries leads to softening. Although the embolus that induces cerebral softening is usually cardiac in origin, it may spring from an aneurismal clot, from thrombosis in the large arterial trunks, or originate in gangrenous or carcinomatous foci in the lungs. Cerebral hemorrhage is a frequent cause of softening, and it may follow blows on the head or exposure to intense cold or heat (sun-stroke).

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[1] Rokitansky described yellow softening as occurring in well-developed spots. The fluid is acid in reaction from liberation of fatty and phosphoric acids. He further says the same kind of softening occurs about adventitious products in the brain, tumors, clots, etc., etc.—*Path. Anatomy.*

[2] Durand-Fardel and Lancereaux both describe *ramollissement blanc* as the last stage of red softening. Charcot speaks of its frequency in old people with cancer.—*Mal. des Vieillards.*

It is well established that any new growth in the brain is accompanied by a zone of peripheral yellow softening. Fevers, the exanthematous especially, ulcerative endocarditis, necrotic and ulcerative diseases in the lungs and bronchi and osteo-myelitis have led to softening, but are more frequently the cause of abscess.[1] Glanders, puerperal diseases, and the toxic action of mercury and lead are regarded by some as causes.[2]

Symptoms.—Inflammatory red softening is usually attended by well marked febrile symptoms; the temperature may reach 102° or 103° F. The pulse is accelerated at first, but afterward it becomes slowed. The face is flushed and the pupils irregular. There is intense and persistent cephalalgia, accompanied by dizziness, vertigo, and somnolence, and followed by confusion of mind, delirium, convulsions and stupor or coma. The gait is tottering and speech embarrassed. At first there is hyperæsthesia, formication, itching, and neuralgic pains. Later—with the paralysis—there is anæsthesia. Vomiting is often severe and obstinate. Muscular twitchings, contractions, clonic convulsions, and hemiplegic symptoms are present and precede coma. Aphasia may accompany these symptoms. If meningitis complicate the softening, its diagnosis is difficult. The softening may take place rapidly and be accompanied by hemorrhage. In such a case apoplectic symptoms will be prominent. Sudden and deep coma, however, may occur in acute softening without hemorrhage. Death may result in from two to eight days; or, recovery may rarely occur with more or less permanent paralysis.

White softening is oftenest met with in old age, and is usually preceded by despondency, physical weakness, *loss of memory*, and inability for prolonged mental labor.[3] It may come on with acute symptoms, or insidiously. Even when sudden in its development prodromata may have existed very like those of apoplexy. Diminution in the motor power is often an important precursor of softening. When such premonitions occur, the symptoms either gradually increase or advance by sudden exacerbations, with intervals of apparent improvement. The affected side becomes feeble, the hands, feet and fingers are moved awkwardly, there is an unsteady tottering gait, and finally complete paralysis. Death may result from implication of the respiratory centres.

Many cases are ushered in by the symptoms of cerebral *thrombosis* or

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[1] Reynolds and Bastian state that red softening may be caused by "prolonged mental exertion or excitement."

[2] M. Rosenthal, *Dis. of Nervous System*, vol. i.

[4] Durand-Fardel lay stress upon monotony of word or gesture as a valuable diagnostic sign.

embolism, and subsequently, as softening slowly progresses, its characteristic symptoms are developed. In the aged, prodromata may not be prominent, and with momentary or without loss of consciousness the patient becomes suddenly paralyzed and aphasic. This resembles an apoplectic seizure, and is accompanied by headache and vertigo. The features are symmetrical until attempts at expression are made. Again, *convulsions* may occur instead of an apoplectiform seizure, or *delirium* may be prominent with muscular rigidity, spasmodic twitchings, dysphagia, and suffusion of the eye. Stupor and paralysis seem to alternate. The urine and fæces are passed involuntarily, and the patient dies from exhaustion. Bedsores are apt to form about the buttocks.

In all forms of softening there is more or less complete hemiplegia attended either by anæsthesia or hyperæsthesia. When convulsions occur they are followed by increasing stupor and paralysis. If the paralysis begins at the fingers or toes and extends toward the trunk (creeping palsy), it is the chief symptom aside from the failure of mind and memory. After a time these patients have to be fed and watched like children; after eating they sleep until they are aroused again, and they often in their actions and in their mental capacity appear like very young children.

Differential Diagnosis.—Red softening may be mistaken for *acute meningitis*. Acute meningitis is, however, attended by a higher temperature, a peculiar pulse, more intense headache and vomiting, and is marked by distinct stages—headache, delirium and coma.

Yellow and white softening may be confounded with *chronic meningitis* and *cerebral tumor*.

In softening there is usually a history of cardiac valvular disease or of senile atheroma. There is well-defined local pain in *cerebral tumor*, while the headache in softening is dull and diffused. Speech and intellect are less affected in *tumors*; they are *both* markedly implicated in softening. Permanent facial paralysis is usually present with cerebral tumors, and absent in softening. The limbs are principally involved in softening. Epileptiform convulsions occur far more frequently with tumors. The symptoms of softening are usually steadily progressive; while those of tumors are irregular and of longer duration.

The diagnosis between the varieties of softening can only be made by the previous history of the patient.

Prognosis.—Acute red softening may lead to abscess, or be a rapidly fatal complication of a pre-existing cerebral disease; death is rarely delayed beyond the tenth day. Chronic softening is a slowly fatal disease. Death may be due to the softening, to meningitis, asphyxia, pulmonary complications, diarrhœa, acute bed-sores, hemorrhage into the softened spot, or to exhaustion and anæmia.

Treatment.—In all varieties of cerebral softening the most important thing to be accomplished is to improve nutrition. In the acute variety the patient must be kept quietly in bed; cold may be applied to the head, and mild revulsives to the extremities.

In threatened *chronic* softening—in the aged especially—attention is to be paid to the diet. The food must be simple, supporting, and very easy of digestion; the best article of diet is milk. Excitement, active and prolonged mental or physical exertion must be carefully guarded against, and the bowels gently moved each day. Zinc, phosphorus, and strychnia may be given in combination with iron and quinine. The constant current alternating with the Faradic should be employed on the paralyzed limbs. For the relief of insomnia and the nervous phenomena cannabis indica combined with the bromides and chloral may be given. Bed-sores demand prompt treatment, for they frequently hasten the fatal issue. Alcoholic stimulants are indicated in the feeble and aged.[1]

CEREBRAL APOPLEXY.

The term cerebral apoplexy, although often applied to a somewhat uniform combination of symptoms of varied causation, will be confined to non-traumatic hemorrhage into the cerebral substance or meninges and the resulting symptomatic conditions.

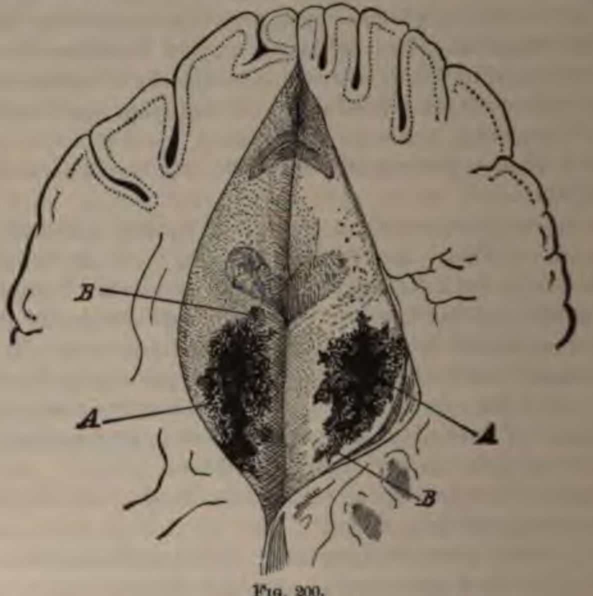

FIG. 200.
Cerebral Apoplexy.
Horizontal Section of the Cerebrum through a Clot in the Left Optic Tract.
A, A. Clots from hemorrhage.
B, B. Area of tissue stained with blood pigment. Lebert.

Morbid Anatomy.—Cerebral hemorrhages are of all sizes, from minute capillary extravasations[2] to large *clots* containing several ounces of blood, the so-called *hemorrhagic foci*. Preceding the hemorrhage the ruptured vessel is the seat of miliary aneurisms due[1] to arterial fibrosis which commences in the perivascular lymph-spaces, and extends to the tunica intima. Globular, saculated, or fusiform dilatations are developed in varying numbers, which are generally microscopic in size, but may be as large as a pin-head, and through their ruptured walls hemorrhage occurs. Minute extravasations, however, play an important part in the development of apoplexy. Such foci are sometimes the result of venous thrombosis, and are probably soon absorbed, or they may accompany cerebral softening, as well as occur in the neighborhood of large apoplectic spots. More rarely an aggregation of these pin-head extravasations forms an apoplectic focus. In capillary hemorrhage, the lymph-sheath may remain intact, or be filled and more or less distended with blood. Fatty degeneration of the walls of the central vessel usually follows. In the other form (hemorrhagic focus), there is found, on autopsy, a clot, varying in size from a pea to a hen's egg, imbedded in the cerebral substance, which is irregularly spherical in the hemispheres, but in the motor tracts it is elliptical or irregular. In some cases an entire hemisphere is ploughed up by a large irregular hemorrhage, which when near the cortex may break through the brain substance, dissecting up or even rupturing the pia mater.

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[1] Cautery, blisters, etc., have often been tried. In none of the cases did benefit ensue; in some, actual increase in the severity of the symptoms followed. Dr. Reynolds regards cod-liver oil as "the most valuable agent in the treatment of chronic cerebral softening." Reynolds' *System*, art. *Softening*, by Reynolds and Bastian.

[2] Capillary apoplexy of Cruveilhier.

The most frequent locations of these extravasations are the intraventricular nucleus of the corpus striatum, the extraventricular nucleus, optic thalamus, cerebellum, and pons—in the order named. The corpus striatum is sometimes pushed up and surrounded by the extravasation; this is made out most distinctly by looking into the ventricles. The ventricles themselves may also be filled by a hemorrhage, or their septa torn and blood escape upon the surface of the brain. In the *aged*, apoplectic foci are not infrequently found between the membranes, in the meshes of the pia, or even superficially. When extravasations are extensive the cerebral convolutions are flattened, the sulci more or less obliterated, the dura is tense, and sometimes there is visible bulging when the hemorrhages are superficial. The adjacent pia mater and uninjured brain substance are anæmic from pressure.

A recent clot is a soft, grumous mass, composed of coagulated blood and brain substance in varying proportions, at whose centre is the opening into the ruptured vessel.[2] It has a ragged wall of cerebral pulp, more or less deeply stained, and covered with fibrinous material, the result of the hemorrhage. Surrounding this is a zone of discolored œdematous brain substance, studded, in many cases, with capillary hemorrhages.

When the apoplectic stroke is not immediately fatal, the following changes may take place in the clot: (1) the fluid parts may be absorbed, leaving the solid elements to undergo secondary changes; (2) the clot and the lacerated cerebral tissue surrounding it may undergo fatty metamorphosis and be absorbed; (3) inflammation may occur in the surrounding brain substance.

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[1] Charcot et Bouchard; *Nouvelles recherches sur l'hemorrhagie cérébrale.* Arch. de Phys.

[2] Rokitansky states that in one form of apoplectic clot the fibrin collects near the centre, and in another toward the periphery of the mass. *Path. Anatomy.*

The subsequent changes which follow the absorption of the fluid portion of the clot are a granular and fatty degeneration of its solid constituents. It may then go on to caseation and subsequent calcareous change, or it may become encysted by the development of new connective-tissue from the neuroglia, forming a firm, smooth, pigmented cyst wall,[1] in which granular and fatty corpuscles are mingled with hæmatoidin crystals. The contents of this cyst are at first a milky or dark chocolate fluid, according to the amount of red globules present, which later becomes thick and creamy, and finally forms a firm, hard caseous mass, or if entirely absorbed there remains a firm, or friable, pigmented cicatrix. Around spots of capillary hemorrhage, the brain is softened and stained; the medullary substance of the nerve fibres is broken up and intermingled with pigment granules and red and white blood corpuscles.

Whether a cyst must have existed previous to the formation of a cicatrix is a question still in dispute. It requires from six months to two years for the cyst to be absorbed and cicatrix to form. There may be a number of these in the same brain, corresponding to the number of apoplexies.[2] Granules and crystals of hæmatoidin are found between the adjacent nerve elements and in the perivascular lymph-spaces, when the deep layers of the cortex are involved. The nerves connected with the motor tracts undergo degeneration, and connective-tissue increase takes place between the atrophied nerve fibres. These degenerative changes after a time extend into the spinal cord, and general atrophy of the brain may follow.[3]

Etiology.—Apoplexy is rare before forty years of age; and after this the tendency steadily increases. Thus age is the most powerful predisposing cause. When it is stated that after seventy the tendency ceases, the small number of those who live after seventy is not taken into account.

It is now generally believed that miliary aneurism is the antecedent state of every vessel that spontaneously ruptures within the cranial cavity. Periarteritis is thus a powerful predisposing factor, causing arterio-sclerosis. Fatty, atheromatous, and fibroid degenerations of the walls of the vessels also predispose to apoplexy. Hence the importance of gout, rheumatism, syphilis, chronic Bright's, and chronic alcoholismus as predisposing causes. Aortic insufficiency, pulmonary emphysema, and left ventricular hypertrophy are important etiological factors.[4]

I have already spoken of the liability to hemorrhage in leukæmia and progressive pernicious anæmia. Scorbutus, typhus, pyæmia, malignant jaundice and chlorosis are conditions in which the blood does not afford adequate nutrition to the vascular walls, and they are then easily ruptured. Men are more liable to apoplexy than women, on account of their active mode of life and greater liability to excitement. Apoplexy occurs more in winter than in summer. The so-called plethoric habit which causes so much anxiety has little significance, for the emaciated valetudinarian is just as liable to apoplexy as he of the opposite condition.[1] Whether atrophy of the brain can produce sufficient dilatation of the cerebral vessels to cause rupture is uncertain. Cerebral softening may, by affording less support to the vessels, predispose to hemorrhage; but is far more frequently a result than a cause of apoplexy.

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[1] Forster states that a connective-tissue wall is not always found, even when death does not occur. *Path. Anatomy.*

[2] Cruveilhier states that he found fifteen in one brain. They cause thickening of the brain.

[3] *Diseases of Old Age*; Charcot and Loomis, N. Y. Wm. Wood & Co., 1881.

[4] Simple ventricular hypertrophy is a physiological condition in old age.

The *exciting* cause of cerebral hemorrhage is usually sudden increase in the blood pressure, although apoplexy may occur without any such increase. Coughing, running, a fall, sudden mental excitement, straining at stool or in passing urine, bending the head far over near the feet, a cold bath, the sexual act (especially in advanced life), large ingestions of alcohol, sudden stopping of bleeding piles, use of opium, and a too hearty and indigestible meal may all induce a stroke in one whose arteries or arterioles are diseased.

Symptoms.—Preceding an apoplectic seizure there may be premonitory symptoms. Vertigo, dizziness, muscæ volitantes, double vision, temporary blindness due to retinal hemorrhages, tinnitus aurium, flushing or pallor of the face, nausea, an abnormally keen sense of smell, or a total loss of it, are, some of them, present in a certain proportion of cases. Epistaxis in one past middle life is an important and dangerous prodromal symptom. These, however, are unimportant compared with loss of memory, tremor, or neuralgic pains, irregular or retarded heart action, difficulty and thickness of speech, lethargy, stupor, change in temper and sense of weight, numbness or formication, which very often are present before an apoplectic seizure, and must always excite alarm whenever they occur in one past middle life. Partial facial palsy is, by some, regarded as a noteworthy precursor.[2] In many cases none of these premonitory symptoms are present, but the seizure is sudden, the patient rapidly passing into a state of coma. In others the comatose state comes on gradually, and is preceded by pains in the head and a feeling of faintness. In rare instances hemiplegia and aphasia are the primary symptoms. Convulsions usher in the attack when large hemorrhages occur into the meninges. With very small hemorrhages there may be only momentary insensibility; the patient recollects everything, though not clearly, and those about him pronounce it a fainting fit, or bad attack of indigestion, as it frequently comes on after over-indulgence at the table.

Usually the coma is sudden and complete, and lasts from a few hours to two or three days.[3] During this coma the respirations are deep, slow, stertorous, and accompanied by a puffing sound; sometimes the face is pale, but more commonly it is red, swollen, and turgid, and as the coma deepens it assumes a dusky, livid hue. Pallor may continue throughout the attack when the hemorrhages are gradual. If the coma lasts from forty-eight to seventy-two hours the temperature is lowered on the second day in some instances to 96° F. The third day it not infrequently rises. The pulse, at the onset of the attack, is slow and irregular: later it becomes frequent and more regular. The pupils are seldom normal; they may be dilated, or, in meningeal apoplexy, contracted. Inequality of the pupils is of much more serious import than equal dilatation or contraction. Sometimes when the pupils are small, they quickly enlarge upon rousing or disturbing the patient. The patient may be unable to swallow, the features become distorted, and, as the paralysis deepens, the pupils dilate; the skin becomes cold and clammy, and the urine and fæces are passed involuntarily. An apoplectic patient may lie apparently dead,[1] yet even in such cases sudden death is rare.[2] Reflex movements, except at the very onset, may nearly always be excited, often more readily than during health. Convulsive movements during the coma are rare. Hemorrhages into the pons and medulla, implicating the roots of the pneumogastrics, are generally followed by death in a few hours. The side that is subsequently paralyzed may show convulsive movements from the commencement, and tetanic spasms in sets of muscles or in single muscles occasionally occur. In many cases the head and both eyes are turned toward the healthy side for a short time.

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[1] If, as H. Jackson supposes, there is an hereditary tendency to apoplexy, it must be transmitted through arterial disease.

[2] Trousseau and Hughlings-Jackson.

[3] Apoplectic coma, according to Niemeyer, Hutchinson, and others, is due to anæmia produced by pressure upon the capillary vessels. This only holds good for large hemorrhages; and small hemorrhages sometimes produce coma.

After the coma, consciousness returns slowly; and in from forty-eight to seventy-two hours headache, restlessness, wandering or delirium may come on. A slight febrile movement, increase of the pulse-rate and respiration, confusion of the mental faculties, and contraction of the flexor muscles, indicate the occurrence of inflammatory changes in and about the clot.

Hemiplegia upon the side opposite to the hemorrhage is one of the most constant attendants of apoplexy, especially in the aged. It may be accompanied by anæsthesia, but this is rarely present without hemiplegia. The hemiplegia is permanent or temporary, according to the extent and location of the clot. As recovery takes place, the thick speech, retracted mouth, deviation of the tongue, and other evidences of facial paralysis gradually disappear. The leg also gains more or less in strength, but the arm is permanently paralyzed. This is more favorable,[3] however, than when the arm recovers and the leg remains paralyzed. Sometimes the face remains semi-paralyzed after the other signs of paralysis have disappeared.[4] Muscular contractures, of varying intensity, which relax during sleep, are rarely absent.

Diminution of electrical excitability is the rule; and the temperature of the paralyzed limb is below normal. The muscles are either hard and rigid, or weak and flaccid. They always show reflex excitability. Anæsthesia soon passes away; but it is claimed that sensation is *never* as perfect on the paralyzed as on the non-paralyzed side. Though anæsthesia and paralysis are commonly distributed over the same region, the former is usually confined to the track of certain nerves.[1] Sometimes the paralyzed parts are hyperæsthetic, the pain being diffused.[2] The organs of special sense are rarely involved. Sight and hearing may be altered, upon increase of intracranial pressure. Hemiopia is not uncommon. Paralysis of the olfactory is rare; but when the chorda tympani is affected taste may be abolished on the *fore* part *of one* side of the tongue.

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[1] Nothnagel.

[2] Wilks: *Guy's Hosp. Reports*, 178, 1866.

[3] Trousseau.

[4] Total loss of motor power is called *paralysis*; partial loss is called *paresis*. Gubler describes some interesting cases of crossed or alternate paralysis, where a left arm and a right leg, etc., were paralyzed. A few cases of paralysis of the facial alone and of the musculo-spiral alone have been recorded.—*Union Médicale*, 1854.

On the second or fourth day after the apoplectic seizure erythematous patches may appear in the sacral region on the paralyzed side. Excoriation then occurs, and the *acute bed-sore,* the most important of the trophic changes, appears as a dry brown crust. The eschar may slowly extend to the sound side.[3] The intellect rarely remains as clear as before seizure, and the disposition changes. The memory, especially for recent events, is markedly impaired, and the will-power is greatly diminished. Some times complete imbecility follows an apoplexy. In the very aged there is a form of apoplexy that is seemingly associated with hemorrhage into, or rupture of, the walls of *the ventricles;* it is accompanied by a general

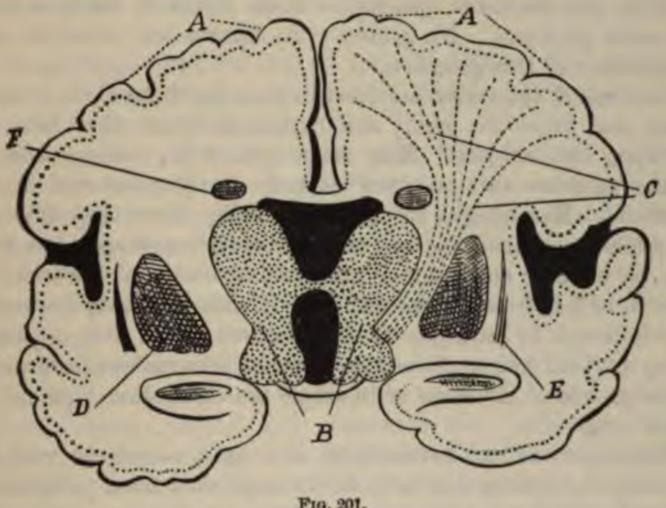

FIG. 201.

Vertical Transverse Section of the Brain through the Optic Thalamus.

A, A. Motor tracts of the cortex cerebri.
B. Optic thalamus.
C. Radiation of internal capsule to the motor tracts of the cortex.
D. Lenticular nucleus.
E. Claustrum.
F. Caudate nucleus.

Charcot.

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[1] Türck states that anæsthesia is *permanent* when the inner part of the lenticular nucleus, the superficial portions of the thalamus opticus and the adjoining portions of the corona radiata are involved. The anæsthesia is also permanent in lesions of the pons and peduncles.

[2] Charcot has laid stress on the arthritic pains that occur in the paralyzed limbs (spontaneous), and Brown-Séquard on the neuralgic pains that are so troublesome during damp, cold weather. The joints are swollen, hot and moist, and there is pain on motion.

[3] Some claim that these eschars depend upon trophic influences due to local hyperæmia. Charcot states that they are due to irritation of trophic centres in the brain. Most authorities, including Charcot and Nothnagel, ascribe little influence to vaso-motorial changes.—*Charcot, Leçons sur les Maladies du System Nerveux.* Paris, 1872.

epileptiform attack, lasting from fifteen to thirty minutes, during which the tongue is bitten and frothing at the mouth occurs. This is followed, apparently, by no serious results, other than the gradual development of extreme debility. Death, however, usually occurs after a longer or shorter period, which varies with the age and constitution of the patient.

Localization of cerebral lesions.

I. Hemorrhage into the *motor ganglia—corpus striatum*[1] and *lenticular nucleus*—occurs in nearly seventy per cent. of all apoplexies. It is attended by hemiplegia, partial paralysis of the face, some anæsthesia, and slight ocular disorders and perversions of the *special* senses. Intelligence is modified, memory chiefly, and there may follow hemi-chorea and athetosis.[2] The head is turned *from* the paralyzed side. To say whether the lenticular nucleus or the nucleus caudatus is alone involved is impossible.

II. When the *thalamus opticus* is alone involved there is anæsthesia and no motor paralysis. No points (yet) known are indicative of exclusive implication of this ganglion.[3]

III. Lesions of the *cortex* are most interesting from their diverse manifestations, and from the study and experimentation that have been expended upon this subject.[4] The *motor zone* of the cortex, however, embraces the anterior and posterior central convolutions and the lobulus paracentralis. Hemiplegia, in nowise differing from that due to lesions of the nucleus lenticularis, may arise from hemorrhage into this part. Aphasia, both ataxic and amnesic, follows destruction of the island of Reil, or of the third left frontal convolution. Hemorrhage into the *paracentral lobule* is followed by paralysis of the arm and leg of the opposite side.[5] Following cortical lesions there appear convulsive epileptiform movements in certain groups of muscles, or in single muscles called "partial epilepsy of cortical origin."

IV. Extensive cortical hemorrhage is usually associated with more or less *meningeal apoplexy*, and is to be distinguished from pachymeningitis. The seat of meningeal hemorrhage may be at the base or convexity, or spread over both hemispheres. The symptoms in children are somnolence, spasms and tremor. Death usually occurs rapidly with convulsions, dyspnœa and sudden attacks of vomiting.[6] In the adult death often occurs very suddenly, and most cases of sudden death from apoplexy are attended by *meningeal hemorrhage*. Rupture of the posterior communicating artery is preceded by signs of compression of the third, fifth and sixth cranial nerves.[7]

V. Hemorrhage into the *pons* is commonly followed by coma and speedy death. Convulsions attend the passage of blood into the fourth ventricle. Incomplete paraplegia, facial paralysis, at times on the same, at others on the opposite side to the lesion, contracted pupils that do not respond to light, disorders of taste, smell, or hearing, indicate apoplexy in the median portion of the pons. Sometimes hemorrhage into the pons attended by slight spasms is followed by partial hemiplegia,[1] or by irregular and difficult breathing. Besides crossed paralysis, we may find hemiplegia, paraplegia and paralysis of all the extremities, with or without facial paralysis; or double facial paralysis with hemiplegic phenomena.[2] The mental symptoms are few, if any. Anæsthesia is common; and may be crossed also. All authorities note that articulation is more difficult and paralysis of the abducens is more likely to occur with this than with any other brain lesion.

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[1] *The caudate nucleus.*

[2] *Top. Diagnos. d. Gehirnkt.* Nothnagel, 1879.

[3] Hammond and Luys alone state that aberrations of the special senses follow lesions of this part of the brain.

[4] Charcot and Pietres state that destruction of the infr. parietal lobe, angular gyrus, of the anterior portion of the first, second and third frontal convolutions produces no motor paralysis.

[5] M. Rosenthal states that psychical disturbances play the chief part in cortical (hemorrhagic) lesions.

[6] *Würt. Med. Corblatt Elsasser.*

[7] Gougonenheim.

VI. Hemorrhages into the *anterior* lobe are commonly attended by hemiplegia, incomplete paralysis of the face, and when on the left side by aphasia.

VII. Hemorrhage into the *middle* lobe is attended by amblyopia, congestion of the retinal veins, and injection of the optic papilla.[3] Nausea, dizziness and headache often occur.

VIII. Hemorrhage into the *posterior* lobe is marked by intellectual disturbances, and usually by the absence of motor and sensory disturbances. When hemiplegia occurs its tendency is to gradually disappear. Frequently no symptoms attend a hemorrhage into the substance of the cerebral lobes.

IX. In hemorrhage into the *cerebellum* vomiting is a prominent symptom.[4] Although it is the *great co-ordinating ganglion*, clots in the cerebellum rarely produce disturbance of *co-ordination*. Sensibility is never disturbed, but there is pain in the *back of the head*. Sometimes the eyes are rolled about incoördinately, and amaurosis and amblyopia occur. In hemiplegia[5] from hemorrhage into the cerebellum there is no lingual or facial paralysis, though there is loss of facial expression. The patients can, and do, lie only in one position; when they are moved they immediately return to it.

X. Hemorrhage into the *lateral cerebellar lobes* is attended by obstinate headache, vertigo, vomiting, amblyopia, amaurosis, dilatation of the pupils, thick and difficult speech, and by hemiplegia on the opposite side.[6] Should the hemorrhage encroach upon any of the great centres, the symptoms will be more pronounced. Injury of the cardio-inhibitory centre would be indicated by irregular heart action, a condition that frequently occurs.

XI. When the *crura of the cerebellum* are involved, the symptoms resemble those of hemorrhage into the cerebellum; most modern pathologists, ascribe all the symptoms of cerebellar hemorrhage to lesions of the crura.

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[1] Gubler and Luys state that crossed paralysis is always attended by apoplexy in the pons. Exceptions have occurred, however.—*Trousseau, Clin. Med.*

[2] Brown-Séquard.

[3] White spots in its centre were noted, in addition to the amaurosis, in Hughlings-Jackson's cases. —*Reynolds' System of Med.*

[4] *Berliner Klinische Wochenschrift.*—Remak, 1865.

[5] *London Lancet*, Nov. 2, 1861.

[6] Rosenthal, *Dis. of Nervous Sys.* vol. i., p. 50-60.

The patients are sometimes forced to rotate about the long axis of the body in some cases toward, in others from, the paralyzed side.

XII. Hemorrhage into the *cerebral peduncle* is attended by hemiplegia and alternating facial paralysis, and by ptosis, mydriasis, and diverging strabismus *on the same side*.

XIII. Destruction of the *corpora quadrigemina* leads to blindness and the pupils become fixed.

XIV. When the *medulla oblongata* is involved, the symptoms are the same as those due to injury of the pons. In addition there is diabetes and albuminuria in many cases. Glosso-pharyngeal and hypoglossal paralysis cause dysphagia and loss of power to protrude the tongue; dyspnœa, irregular heart action, and gastric derangements arise from implication of the pneumogastric.

XV. Finally, when hemorrhage occurs into the *ventricles*, death is usually rapid. Recovery is possible, however.[1] Spasms and contractions of the paralyzed extremities occur in many of these cases.

Differential Diagnosis.—*Apoplexy* may be mistaken for *cerebral congestion, uræmia, alcoholic coma, cerebral embolism, opium poisoning, epileptic* and *hysterical coma*.

Stertorous breathing—a common symptom in apoplexy—is absent in *cerebral congestion*. The pupils are alike in congestion; in apoplexy they are unequal. The coma is of short duration in congestion; in apoplexy it persists for some time. Congestion has a long prodromal period; in apoplexy this is short or absent. Should paralysis be present from congestion it is usually bilateral; while in apoplexy hemiplegia is more or less complete.

The mental faculties are rapidly and completely restored after an attack of apoplectiform congestion, while the reverse is the case in apoplexy.

In *uræmia* the previous history of the patient is important, and there is more or less œdema. Hemiplegia is never present in uræmia; it is rarely absent in apoplexy. Uræmic coma comes on gradually and is usually preceded by convulsions; while the coma of apoplexy is more sudden in its advent, and is followed rather than preceded by convulsions. Casts and albumen in the urine are strong presumptive evidences of uræmic coma.

Profound alcoholic *intoxication* is often mistaken for apoplexy. A patient can be roused from alcoholic coma, but not from apoplectic. There is no stertorous breathing in alcoholic coma; while this is rarely absent in apoplexy. The pulse, in alcoholic coma is feeble; in apoplexy it is full, strong and slow. There is no hemiplegia in alcoholismus, and the urine as well as the contents of the stomach will contain alcohol.

The diagnosis between apoplexy and *cerebral embolism* is often difficult. Both may be preceded by rheumatic endocarditis and valvular disease of the heart, although they are more frequent in embolism than in apoplexy. In embolism there is rarely complete loss of consciousness; and if it occur it is of short duration; while loss of consciousness is the rule in apoplexy, and it usually continues for two or three days. The pulse in embolism is feeble and frequent, and the face is pallid; while in apoplexy the pulse is slow and full, and the face is suffused. Aphasia is the rule in embolism and the exception in apoplexy. The pupils are unchanged in embolism; while in apoplexy they may be dilated, contracted, or unequal. The respiration is normal in embolism and stertorous in apoplexy. There is usually right-side hemiplegia in embolism; if left side hemiplegia exist, it is probably due to cerebral hemorrhage. Arterial degeneration is always present in apoplexy; while in embolism the arteries may be normal. The paralysis is temporary in embolism and recovery is complete; while in apoplexy it is delayed, and recovery is partial. Vomiting is a far more prominent symptom of apoplexy than of embolism. Embolism is probable when hemiplegia occurs suddenly in the young; apoplexy is a disease of middle and advanced life. Premonitory cerebral symptoms are *never* present in embolism; they may be in apoplexy.

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[1] *Diseases of Old Age*; Charcot and Loomis, New York, 1881.

Opium poisoning gives many of the symptoms of apoplexy. Apoplexy is usually accompanied by dilated or irregular pupils; opium always produces regular and generally pin-head pupils. Convulsions may attend apoplexy; they are absent in opium poisoning. The coma comes on more gradually, and is not usually as deep in opium poisoning as in apoplexy. An exceedingly *slow* pulse and respiration indicate narcotic poisoning. *Stertor* and *hemiplegia* attend apoplexy, and the pulse may be irregular.

The coma of *epilepsy* may be confounded with that of apoplexy; but the blood-stained frothing at the mouth, the imprint of the teeth on the tongue, the history of previous convulsions, the rapid recovery, and the age of the patient are sufficient to distinguish epileptic from apoplectic coma.

In *hysteria* the coma is not deep, and cold will restore to consciousness. Stertor is absent, the pupils are mobile or unchanged. In hysterical hemiplegia the patient drags the limb like an inert mass, contractions develop more rapidly than in apoplexy, and the electro-muscular contractility diminishes after it has lasted for a short time. Hysteria is preceded by characteristic hysterical attacks which have been followed by abundant limpid urine. In spinal hemiplegia *sensation* is preserved. The electro-muscular contractility is diminished, and reflex excitability is increased. Sensation is lost on the opposite side, but motion and contractility are intact.

Prognosis.—The prognosis of apoplexy is always grave. The greater the age the more unfavorable it is.[1] Death rarely occurs at the onset of the seizure; but the hemiplegia, the loss of mental power, and the liability to recurring attacks render it, even when not at once fatal, a dreaded condition. It is a favorable sign if the hemiplegia begins to improve very soon after the attack; but if the period of unconsciousness is prolonged, if the coma deepens, if reflex excitability is wholly lost, the sphincters relaxed, the breathing irregular, puffy, and noisy, if the pupils enlarge, or the temperature after having fallen rises rapidly, a fatal termination is indicated. Convulsions in the aged always indicate great danger.

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[1] Durand-Fardel state that ventricular and meningeal hemorrhages are frequent after sixty; hence an apoplexy in one who has passed that age must be regarded as very serious.

The general condition of the patient and the extent and *degree* of paralysis are always important factors in the prognosis. Epileptic seizures may follow a partial recovery from apoplexy. Death may occur from the shock of a large extravasation, from interference with the medullary centres, from asphyxia, and sometimes from inanition.

Treatment.—In one who has the prodromal symptoms of apoplexy, or whose age and condition are such as to favor its occurrence, *prophylaxis* may avert an attack.

The principal prophylactic measures are the avoidance of sudden or violent physical exercise or strain, and of strong mental emotions. The diet should be most nutritious, but non-stimulating, and sleeping and living rooms should be large and well ventilated. Great care should be taken that the functions of the intestines, liver, and kidneys are kept at their normal standard ; and moderate exercise should be taken daily in the open air. Sudden extremes of temperature should be avoided ; therefore hot or cold baths are to be forbidden. The body should always be warmly clothed in flannels.

In the advent of the premonitory symptoms, free purgation and the application of blisters to the neck with the bromide of lithium and oxide of zinc are indicated.

When an apoplectic seizure has occurred, the patient's head is to be elevated, the clothing about the neck loosened, and he is to be put in bed in a cool, dark and absolutely *quiet* apartment, with cold applied to the head and heat to the feet. If the fit occur after a hearty meal, vomiting must be induced and a purge given. Blood-letting is to be the exception ; but if a very robust individual with high arterial tension is seized, and there is evidence of progressive hemorrhage, then six to eight ounces of blood may be taken. The condition of the heart and the arterial tension are the guides as to the propriety of blood-letting. In old age or in the weak, with a pale face and feeble pulse, venesection is never to be practised. The condition of the bladder should be carefully examined, and the urine drawn if necessary. Much of the turgescence of the face is due to the falling back of the tongue, consequently the patient should be placed on his *side*. Sinapisms may be applied to the nape of the neck, calves, and over the stomach, when venesection is not practised.

As the patient comes out of the coma the vital powers must be sustained, the most absolute rest and quiet enjoined, and the bowels kept freely open by mild salines. Milk and beef juice are to be freely administered, and if there is very great feebleness stimulants may be given. Stimulation is demanded very early in old and feeble subjects. Narcotics are indicated when there is great restlessness and insomnia, especially in the aged.

The clot in the brain is now a foreign body : nothing external or internal can remove it ; hence blisters, ointments, drugs, etc., etc., are worse than useless. The *galvanic* current to the paralyzed limbs is indicated if the paralysis persists. It may be passed directly through the brain, and though the absorption of the clot may not be aided by it, it often benefits the paralyzed limbs. It should not be resorted to until three or four months after the seizure; but passive motion, gentle friction, and the application of stimulating liniments to the surface may be practised early on the paralyzed limb. Massage of the paralyzed limb should always form part of the treatment. When electricity is used, three or four seances a week, each lasting from five to eight minutes, are sufficient.[1]

ABSCESS OF THE BRAIN.

Abscess of the brain or *suppurative encephalitis* may occur in any part of the brain. It may be simple or multiple, and may not differ in character from abscesses in the connective-tissue in any part of the body.

Morbid Anatomy.—The white substance of the middle cerebral lobes is its most frequent site. About 16 per cent. of all cases are located in the cerebellum, and about 3 per cent. each in the pons, corpus striatum and thalamus opticus.[2] They may vary in size from a walnut to the involvement of an entire hemisphere. Usually they are from one to two inches in diameter. They are irregularly spherical in shape.

Embolic abscesses are usually multiple. Their walls are irregular and made up of shreddy, disintegrated brain substance, with projections which are found to surround blood-vessels. A limiting membrane may or may not exist. In recent abscesses it is either wanting or incomplete. Some abscesses have a membrane from *their very onset;* they are encapsulated. Usually a zone of red softening surrounds the abscess, and around this is an envelope of œdematous brain substance.[3]

Their contents are usually inodorous and composed of a greenish, creamy pus, fatty and granular matter, the débris of necrosed brain-tissue. Pyæmic abscesses may contain fetid pus. The pus is decidedly alkaline; very rarely is it acid. When *mucin* is present the pus is ropy, viscid, or gelatinous. As an abscess increases in size it causes pressure on the adjacent brain substance. In large abscesses the convolutions are compressed, so that their edges are sharpened and their surfaces flattened. On removing the brain a bulging with a boggy feel is sometimes noticed.

Cerebral abscesses may rupture into the ventricles, or they may make their way to the surface of the brain and cause diffuse suppurative meningitis. In rare instances they discharge into the cavity of the tympanum, the nasal fossæ, or the orbit of the eye.[4] Multiple abscesses are small; they are found scattered throughout the brain. The processes by which the formation of a cyst wall is effected in these abscesses have been described by Rindfleisch as follows: a fibrinous wall, sometimes a quarter of an inch thick, may envelop the abscess. The innermost, lining membrane of this cyst-wall consists of a yellow, smooth layer of cells. Tortuous venous vessels traverse it; it is sometimes called the pyogenic membrane. Next to it is a layer of germ-tissue, irregular in thickness. Externally is a stratiform, spindle-celled tissue, that forms a direct transition into the surrounding brain matter; in spite of which, however, the abscess can be enucleated. A zone of fatty degeneration surrounds the outermost (fibrous) layer of the cyst-wall. The pus, Rindfleisch further states, is greasy, greenish-yellow, acid, and usually odorless. It is to be noted that Gull and Sutton state the pus to be decidedly alkaline and very fetid in old abscesses.

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[1] The subcutaneous injection of strychnia into the paralyzed limb has been recommended, and hypodermics of ergotin have been advocated during the attack and at the commencement of the subsequent coma. Dr. Celborne reports a case where, after an hour and a quarter's persistent practice of artificial respiration by the Sylvester Method, asphyxia was averted.—*Jour. Med. Chirur. Pesth.*, Dr. Foster, *N. Y. Med. Rec.*, 1876.

[2] Hammond states that the gray matter is involved first, the white secondarily. Gull and Sutton state just the contrary.

[3] Rokitansky states that yellow (chronic) softening surrounds cerebral abscess in a large majority of cases.

[4] Niemeyer states that the pus, after reaching the surface of the brain, may perforate outwardly through the bones, provided extensive meningitis has not been excited.—*Text-Book of Pract. Med.*, vol. ii., p. 227.

Hæmatoidin crystals, margarin, and cholesterin are not infrequently found mingled with the pus, and the entire capsule is to be regarded *as a neuroglia production.* Absorption, cheesy degeneration, and the formation of chalky masses are said to occur in cerebral abscesses. The cyst-wall retracts and finally disappears.[1]

Etiology.—Cerebral abscess occurs at all ages. Males are more subject to it than females. Among its chief causes are suppurative otitis and traumatism, especially blows on the head. It may result from suppurative inflammation of the face and scalp, and from suppuration about the orbit or nose. Syphilitic and other diseases of the bones of the skull, the temporal especially, are not infrequently followed by abscess. Pyæmia and glanders are among its frequent causes. Red inflammatory softening is the first stage of abscess. Ulcerative endocarditis and osteo-myelitis are especially liable to give rise to it; the embolus in these cases has a special character. There are cases in which no cause can be found for their development.

Symptoms.—Headache is the most constant and prominent symptom of abscess of the brain. In some cases it is not severe but is constant, in others it is so severe that patients are not able to bear it without an anodyne. It may be so circumscribed as to localize the very site of the abscess. With the headache there is vomiting and dizziness. Delirium and disturbance of intellect may be marked but transitory; it may alternate with stupor. Epileptiform convulsions, and signs of cerebral pressure may end in com Incontinence of urine and fæces is a prominent symptom in most cases.

It is to be remembered that large abscesses have been found in the brai of those who during life, gave no cerebral symptoms. Otitic abscesses of the brain are preceded by all the signs of the (causative) local disease. Headache, vomiting, delirium, *fever* and irregular *chills,* spasmodic movements in the muscles of the face or limbs, then hemiplegia, coma and death —this is the usual course of such an abscess. But cases are reported where an artificial or spontaneous exit to the pus has been followed by recovery.[2] In some cases optic neuritis has been found.[3] Rapid and progressive emaciation usually accompanies cerebral abscess. At times there will be hyperæsthesia and abnormal acuteness of the special senses at the onset;

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[1] Rosenthal, *Diseases of Nervous System,* vol. i.

[2] Schloz.

[3] Hughlings-Jackson.

and this will be followed by sopor, anæsthesia, formication, numbness, etc., etc. If the abscess involves the motor tract, hemiplegia or local paralyses will occur.

When pyæmic abscesses occur in the brain, they are chiefly diagnosticated by the constitutional symptoms. It will begin with rigors and run an acute course; the temperature may reach 105° F. Ague-like rigors and the initial signs of abscess coming on when the conditions of pyæmia exist must lead to the suspicion of multiple cerebral abscess.

Sometimes in chronic abscess of the brain there is a long latent period.[1] During this time epileptiform convulsions, facial paralysis, and hemiplegia and aphasia accompanied by intermittent chills and headache may occur; After which, acute symptoms may be present for a few days and end in death. The acute symptoms differ widely in different cases; there may be localized headache over the abscess, delirium, nausea, vomiting, well-marked signs of cerebral irritation, ending by a fall in temperature and pulse, deep coma and death.

Differential Diagnosis.—It is always difficult to distinguish cerebral abscesses from *cerebral tumor*. Abscess is accompanied by greater emaciation and is of shorter duration than tumor. Local paralyses of long standing are common in tumors, rare in abscess. Rigors and more or less fever usually attend abscess. An ozœnal or otorrhœal discharge, the history of traumatism, or the fact of a *latent period* having existed, is in favor of abscess. Softening may be mistaken for abscess. The age of the patient, the condition of the blood-vessels, the slow development of the hemiplegia, the absence of constant or *intense* localized pains in the head and the gradual loss of mental power, distinguish softening from abscess.

Prognosis.—Acute abscess of the brain is always fatal in from four to twenty days. Chronic abscess terminates fatally from its complications, the commonest of which are meningitis, cerebral hemorrhage, œdema, softening, thrombosis of the cerebral sinuses, serous effusions into the meshes of the pia mater and the ventricles, and pulmonary hypostasis. When abscesses are situated away from the motor tract and surface of the brain, they may exist for years and give rise to no symptoms.

Treatment.—The treatment of cerebral abscess is altogether surgical. The operation of trephining for traumatic abscess, and the treatment of chronic aural disease, are found in modern surgery and in special works upon diseases of the ear. Recently the withdrawal of pus from the brain has received much attention, and marked success has attended surgical operations for the accomplishment of this end. Anodynes are always indicated for the relief of the intense headache.

TUMORS OF THE BRAIN AND MENINGES.

The most frequent intracranial growths are *tubercle, cancer, gummata* and *gliomata*. Apoplexy and abscess, which have much in common with tumors of the brain, are elsewhere described. Some intracranial growths

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[1] Lebert says from one to two months.

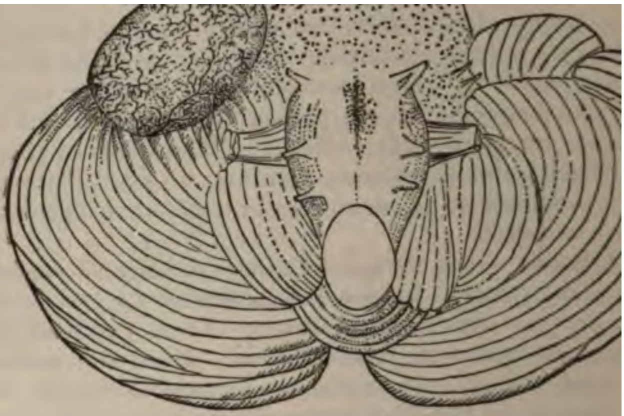

Fig. 202.

Cerebral Tumors.

Sketch showing at A a Fibroma of the Cerebellum, from Lebert.

myxomata, lipomata, cholesteatomata and psammomata ; i
sels as angiomata, aneurisms, and a peculiar tumor in rare i
in the third ventricle—the epithelioma myxomatodes psam
in the *neuroglia*, as gliomata, gummata and fibromata.

Morbid Anatomy.—The commonest form of cerebral tumo
culous. These growths vary in size from a pea to an orange.
and compact, their exterior being gray, semi-transparent,
blended with the surrounding brain-tissue. Their centres a
low. They develop slowly and may calcify. The vessels goi
dilated, and at the centre of the growth they are indisting
bercular tumors occur in the hemispheres, cerebellum, opti

the amount of retrogressive degenerative changes that have occurred at their centres. When more than one tumor is present, they develop symmetrically, and are liable to involve homonymous parts of the brain (Rokitansky).

Gummata, or syphilomata, may appear as soft, red-gray, jelly-like masses, irregular in form, and intimately blended with the adjacent brain substance. They are chiefly composed of round cells, but spindle and stellated cells are sometimes found. They may have an alveolar framework. Capillaries are not numerous in their substance. There may, however, be small points of extravasation. There are rare forms of gummata which consist of a well-defined homogeneous mass, which is dry, friable and cheesy. Atrophied neuroglia and round and spindle cells in a state of fatty degeneration are often found throughout the gummy masses. Syphilomata are generally found at the circumference, and especially at the base of the brain. They may have their origin in the membranes, the vessels, or the neuroglia. The surrounding inflammation joins them to the meninges. If the latter be joined to the dura mater, granules are developed in the pia, which vary in size from a pea to that of a small egg. Syphilitic tumors in the interior of the brain are very rare.[1]

So-called syphilis of the brain may appear, (1) as well-defined hard tumors, (2) as thickenings, adhesions, and contractions or puckerings of the meninges, (3) as disease of the walls of the vessels, and (4) as spots of softening or diffuse gelatinous accumulations.[2]

Gliomata[3] may develop either in the brain substance, or in the meninges along the course of the cephalic nerves, or in the retina. On account of its vascularity, hemorrhages are liable to take place into its substance. At times a light brown coagulum causes the tumor to resemble tuberculous or gummatous growths. It is often difficult to distinguish a glioma from normal brain substance. These tumors may be either hard or soft; soft gliomata contain only a small quantity of intercellular substance. Hard gliomata have bundles of parallel or interlacing fibrillæ as their fundamental substance. They vary in size from minute masses to masses as large as an orange. They grow slowly and are usually solitary. There are fat granules, cholesterin crystals, neuroglia nuclei, a débris of nerve-tissue, and more or less redness and softening in the immediate neighborhood of gliomata.[4] Diffuse gliomatous masses were once thought to be infiltrated cancer of the brain.

Psammomata, or Virchow's sandy tumor, are soft, juiceless sarcomata whose cells are thin, flat, irregular in outline but *very* large. The vessels are in direct connection with the cells. Psammomata usually develop from the dura. They may reach the size of a pea, and are hard, smooth, and spherical. These calcareous tumors often have a concentric or laminated structure.

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[1] Rindfleisch states that syphilomata develop in the brain substance along the lymphatic sheaths and the vessels, and that they produce spots of softening by compression of the vessels and arrest of the circulation.

[2] Niemeyer states that gummata are more frequent as diffuse infiltrations. Virchow, Charcot and Westphal have found gummata in the white substance of the hemispheres, in the thal. opticus, the pituitary gland, the optic tracts, the cerebral peduncles, the pons, and in the cerebellum.

[3] The neuroglia-sarcomata of Cornil and Ranvier.

[4] Ernest Wagner and Obermeier regard hyperplasia of the pineal gland as essentially the same as a gliomatous neoplasm.

Cholesteatoma (called pearl tumors from their lustre) do not always contain cholesterin, but consist of concentric layers of epithelial cells that have partially undergone fatty degeneration. They may have an indistinct fibrous envelope. They rarely exceed one and one-half inch in diameter. They are aggregations of crystalline, pearly masses, the size of a mustard-seed; they grow slowly from the pia at the base of the brain, or in some depression of it. They are pathologically insignificant.

Cysts (independent of cystic developments from echinococci, etc., etc.) are rare. They seldom exceed the size of a pin head, and are found on the walls of the lateral ventricle. They may occur singly or in groups. Transparent serous cysts developing from the vessels of the choroid plexus are not rare.

Medullary or ganglionic *neuromata*—tumors of nerve-cells and ganglia—occur in, and on the brain; they are seldom larger than a pea, and are found on the ventricular surface, in the white substance, or in the corpora striata.[1] A tumor to be a neuroma must contain a large number of nerve-cells.

Sarcomata appear as well-defined, round or lobulated tumors, varying in consistency, and in size from that of a walnut to an apple. They may originate in the cerebral hemispheres, but more frequently they arise from the dura, especially at the base of the skull. There are two forms, the *hard sarcoma* with compact hard fundamental tissue and small cells, and the *soft sarcoma* with loose, scanty intercellular substance and numerous cells of large size. They are separated from the surrounding brain substance by a very vascular zone.

Myxomata appear either as well-defined tumors or as infiltrated masses whose seat and size resemble those of sarcomata. Indeed, to determine whether a tumor is a sarcoma that has undergone mucous transformation, or a myxoma with patches of embryonic tissue, is always very difficult.

A true *myxoma* or *sarcoma* may result from a glioma (*q. v.*), the transition being gradual; these various sarcomatous tumors are found in the hemispheres, the anterior lobes, thalamus opticus, cerebral peduncles, or they may involve the pons and tubercular quadrigemina.[2]

Lipomata are rare, and are only found at the raphé of the corpus callosum and fornix.[3]

Osteomata, new formations of bone, independent of ossification or other neoplasia, are not frequent. Osteoma of the cerebellum has been found to follow encephalitis. Osseous new growths must not be confounded with syphilitic or other exostoses.

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[1] Cornil and Ranvier. *Pathol. Histologique.*

[2] *Transac. Pathol. Society*, Dr. Cayley, vol. xvi., p. 23.

[3] Virchow. Benjamin reports a case where ossification had occurred; Niemeyer describes lipomata as "small lobulated tumors starting from the dura."

Papillomata are the rarest form of cerebral tumors. They are cauliflower-like, budding growths, with abundant milky juice, very vascular, and surrounded by a zone of cerebral softening. A large one—situated on the ependyma of the third ventricle—is described by Cornil and Ranvier.[1]

Fibromata appear as hard, fibrillar tumors, small, and rarely pedunculated. In one case seventeen fibrous tumors were found on the ependyma of the lateral ventricle. The pons and the cerebral peduncle are sometimes implicated by this tumor.

Angiomata, or erectile tumors, are masses composed of vessels of new formation. They are found in the cerebral substance, corpus striatum, cerebellum, and floor of the fourth ventricle. They may be multiple.[2]

Aneurisms are not uncommon; they do not attain a large size. They involve the basilar artery, the vessels in the Sylvian fossa and corpus callosum, the anterior communicating—rarely other than basic vessels; from their position they are liable to compress some of the nerves at the base of the brain. Miliary aneurisms have been considered in the history of apoplexy.

Hydatids or *echinococcus cysts* usually exist as solitary tumors which may attain any size. They are generally located at the centre of the white matter of a hemisphere. The cyst-wall is always absent when the hydatid is in the ventricle. Some claim that it is absent even when located elsewhere in the brain. They grow slowly, producing atrophy of the brain. Five large hydatid cysts have been found in various parts of the same brain.

Cysticerci in the brain form small serous cysts that may occur in any part of the organ, and are rarely solitary. Cruveilhier describes a case where one hundred were found. When the cysticercus is lodged in cavities, it is non-encysted and tends to grow easily into the form of a tape-worm. These parasites may be found dead and changed to a chalky mass in which some of the hooklets are embedded.

Etiology.—Tumors of the brain are twice as frequent in males as in females. Tuberculous tumors are most frequent in children; they usually do not develop until after the second year. Cancer is rare before forty; it is primary in fifty per cent. of the cases. Syphilitic growths are a manifestation of tertiary syphilis. Hydatids occur between the ages of ten and thirty-five; while cysticerci are rarely found before forty. Aneurisms occur in middle life, and are associated with evidences of arterial degeneration.

Symptoms.—Cerebral tumors of large size may give rise to no symptoms, but in most cases their development is attended by more or less marked general or local symptoms. These, however, cannot be stated in any order that is applicable to all cases. I shall only consider the more important and constant of the general symptoms. The most characteristic are headache and disturbance of the intellectual faculties. The most constant local symptom is local paralysis. Headache is generally a prominent and persistent symptom. It is more severe than in any other cerebral disease, except meningitis; it is constant, and is increased by light, sound, or movements of the head. With the headache there are tinnitus aurium, morbid acuteness of hearing, disturbance of vision, strabismus, with more or less perversion of the special senses, local hyperæsthesia, anæsthesia, and impairment of the mental faculties. Vertigo, when the patient assumes the upright position, is almost always associated with the headache, and vomiting occurs at irregular intervals without any apparent cause. There are rarely any febrile symptoms, except when inflammation occurs about the tumors. A slow irregular pulse is of frequent occurrence during the early stage of their development, and the respirations are often irregular and slowed. Spasm of single muscles or groups of muscles and general epileptiform or choreic seizures often follow severe attacks of vertigo.

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[1] *Path. Hist.*, p. 378.

[2] Obermeier gives the name pachymeningitis hemorrhagica bregmatica to angiomatous growths upon the inner surface of the dura.

Hemiplegia is entirely absent in a large number of cases. If present it may come on slowly, or suddenly after an epileptiform seizure; facial paralysis on the same side as the hemiplegia, is present in some cases, and rigidity of the affected muscles is common. Double hemiplegia is not infrequent; one side being implicated some time after the other. If paraplegia exists it indicates a median tumor, usually at the base or in the cerebellum.

If the tumor involves the island of Reil or the posterior portion of the third convolution there will be *aphasia*. The intellectual disturbances are varied. Melancholia and paroxysms of grief or joy are frequent. Incoherence of speech, failure of memory, temporary loss of consciousness, and a gradual passage into a condition of imbecility and helplessness are a part of its history. This is especially liable to occur in cases where the tumors are rapidly developed.

The *choked disc* or congested papilla, and the neuro-retinitis as revealed by the ophthalmoscope, are regarded by some as important in the diagnosis of cerebral tumors. Such conditions may cause amaurosis and amblyopia.[1] If there is loss of sight from involvement of the optic nerves, the pupils will be dilated and they will not contract under the influence of light. If the tumor is situated above the corpora geniculata, although there is loss of sight, the pupils will respond to light. As the tumor increases in size, the paralysis advances from one set or group of muscles to another, and this advancing paralysis is a most important diagnostic sign. Its course is usually from above downward.

On account of the local disturbances which result from the complicating encephalitis, abscess, softening, or œdema may occur and give rise to their own peculiar symptoms. The bowels are usually obstinately constipated. Clinically there may be recognized three classes of cases: (1) those which are attended by no symptoms, (2) those in which the symptoms are slowly developed and intermittent and extend over a number of years, (3) those in which the symptoms come on suddenly and are rapidly fatal.

Differential Diagnosis.—Tumors of the brain may be mistaken for *abscess*,

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[1] Annuske (in *v. Graefe's Archiv*) states that *optic neuritis occupies the first rank among the symptoms of intracranial neoplasia.*

cerebral softening, epilepsy and *chronic meningitis*. The points of diagnosis between the first two have already been considered.

An *apoplectic* or *hysterical* seizure will hardly ever be mistaken for a cerebral tumor.

Epilepsy is a paroxysmal disease; it usually occurs early in life and is rarely accompanied by any of the local phenomena of tumor.

In *chronic meningitis* the pain is not so severe or as constant as in cerebral tumor, the mind is perverted and *weakened*, epileptiform convulsions are rare, and the special senses and facial nerves are *not* implicated.

The differential diagnosis of the different *varieties* of cerebral tumor may be briefly summarized as follows:—*tubercular* growths are met with in early life; they are accompanied by fever, have a tubercular history, or have the evidences of tuberculosis in other parts of the body. They are usually located either in the cerebellum or pons.[1] *Cancer of the brain* may be suspected when the patient is over forty, when there is a marked cachexia with progressive emaciation, when there is an hereditary cancerous history, or when cancer exists in other organs, and when the development of the cerebral symptoms has been *rapid*. Implication of the cranial bones by the tumor always indicates cancer.

Syphilomata are attended by nocturnal headache, by the constitutional signs of *syphilis* or evidences of previous syphilitic disease. Their symptoms remit and sometimes disappear under anti-syphilitic measures. Ptosis and dilatation of the pupil are more often met with in syphilitic tumors than with any other.

Gliomata follow traumatic injuries to the skull, and progress slowly without any interference with the general health.

Aneurism may be suspected in the aged, with the signs of general arterial degeneration.

Cysticerci occur after forty, and produce, at first, subacute epileptiform attacks, which become very frequent. One hundred epileptic seizures have occurred in a day with cysticerci in the brain. Paralysis of the limbs and hemiplegia are *very rare*, while the psychical disturbances are marked, and occur very early.

The diagnosis of *hydatids* is exceedingly difficult. The tumor-symptoms are inconstant; the intellect is unaffected, and there may be œdema of eyelids.

The rules which will aid in the *localization* of *cerebral tumors* are as follows:—*tumors of the convexity*, even when large, may give rise to no symptoms. Usually, however, they cause headache, *motor* disturbances, delirium, convulsions; but *rarely* disturbances of sensation or paralysis.

Tumors of the *anterior* lobes cause diffuse or circumscribed headache, convulsions, and epileptiform attacks, hemiplegia, and *aphasia*. The special senses are undisturbed, except the sense of smell.

In tumors of the *middle* lobes the special senses (especially *sight*) are affected, and there is usually anæsthesia of the surface on the side *opposite* the tumor.

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[1] Hirschberg.

Tumors in the *posterior* lobes cause greater psychical disturbances than those in any other position. Motion, sensation, and the special senses (sight excepted) are more or less disturbed. Vertigo and convulsions are common.

Tumors in the *corpus striatum* and *lenticular nucleus* are accompanied by hemiplegia, convulsions, facial paralysis, difficulty in *articulation*, disturbance of intelligence; but the *special senses* are not impaired. The symptoms correspond to those of apoplexy, but they are of slower development.

Tumors in the *tubercula quadrigemina* are attended by convulsive spasms, paralysis of the motor oculi, disorders of vision on *both* sides, slight paralysis of the face, and unilateral paralysis of the limbs.

Tumors in the *cerebral peduncles* induce headache, vertigo, hemiplegia alternating with sensory disturbances, paralysis of the motor oculi on the same side, neuro retinitis, difficulty of micturition, but no intellectual disturbances.

Tumors in the *pons Varolii* induce crossed paralysis of motion (and sometimes of sensation also), amblyopia, amaurosis, choked disc, dysphagia, strabismus, difficulty in articulation, but *no* convulsions. There is usually paralysis of the bladder.

When the *cerebellar peduncles* are involved the gait is tottering and unsteady; the patient tends to fall to one side, or rotate around the median line.

In *cerebellar* tumors, there will be occipital headache, oscillatory movements, unsteady gait, intense vertigo, strabismus, amblyopia, and amaurosis; but there are no disturbances of the intellect or sensation.

In tumors of the *medulla*, the symptoms not infrequently resemble glosso-labio-laryngeal paralysis. There is dysphagia, disturbances of sensation, convulsions, occasionally saccharine urine, and difficulty in articulation.

Prognosis.—The character of cerebral tumors varies with their structure. The prognosis is always unfavorable; carcinomatous and tuberculous tumors are progressive and early fatal. In hydatids and aneurism, though life may be prolonged, death is a certain result. In syphiloma, life may be prolonged for years by judicious *and timely* treatment. The average duration of cancer is about one year.[1] Echinococci tumors have been cured.[2] In any case of cerebral tumor which is attended by intense pain and progressive emaciation, the course is rapid, and the prognosis unfavorable. Death may occur from continued convulsions, paralysis, cerebral softening, œdema or hemorrhage. Secondary inflammation with abscess, and meningitis, and pulmonary complication, may be the cause of death. Anæmia and exhaustion are common modes of death in specific tumors.

Treatment.—In a case of cerebral tumor where syphilis can even be *sus-*

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[1] Lebert states that three months and five years are the extremes.

[2] Mouline trephined, and Fletcher incised, a frontal tumor, withdrawing the hydatids. These are phenomenal cases.

pected, mercury and iodide of potassium in large doses should be administered. The diet and hygienic surroundings should be carefully regulated, the patient restricted in exercise, the pain and sleeplessness should be relieved by anodynes.

SCLEROSIS OF THE BRAIN.

Independent of cerebro-spinal sclerosis, this is a comparatively rare condition. Cerebral sclerosis is a chronic interstitial inflammation, following hyperæmia of the neuroglia.[1] It may be diffused or multiple.

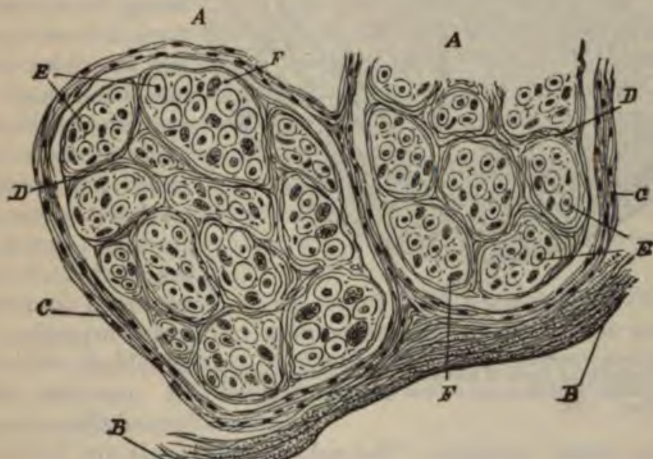

FIG. 203.

Diagram showing the Connective-tissues of Medullated Nerve Structure.

AA. Two bundles of medullated nerves.
BB. Epineurium.
CC. Perineurium.
DD. Endoneurium.
E. Axis-cylinders.
F. Neuroglia cells.

Morbid Anatomy.—The medullary substance is the favorite seat of multiple cerebral sclerosis.

On section, masses of gray, hard, well-defined, transparent sclerotic tissue are found—sclerotic islands varying in size from one-fourth to one inch. They may be so numerous and small as to be scarcely discoverable.[2]

The cut surface of a sclerosed patch is moist with serum; and usually shows small blue or gray-red spots.

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[1] The tissue forming the skeleton framework of the brain is called the neuroglia by Virchow. It is analogous to the connective-tissue framework between the liver-lobules and kidney tubules.

[2] Cornil and Ranvier describe cerebral sclerosis as almost exclusively involving the convolutions, and consisting of a first stage where hyperplasia of the neuroglia produces a vascular, pulpy, gelatinous mass; and of a second stage where atrophy of the new elements is accompanied by development of a vascular structure, hard and resistant.

A microscopical examination of the patch in its soft stage shows active hyperplasia of the neuroglia-cells. Later, compression of nerve-substance occurs from the pressure of the hyperplastic neuroglia tissue, which, at this period, exists as fibrillated connective-tissue, whose fibrils—extremely fine and interlacing in all directions—form a network containing atrophied nerve elements and small round or oval nucleated cells. The axis-cylinders are preserved, and are sometimes markedly hypertrophied at their periphery. At the centre of the mass are found numerous amyloid corpuscles, a few atrophied axis-cylinders, fat granules, and new formed fibres that entirely replace the normal elements. The walls of the vessels are thickened. In the brain any portion of the white substance may exhibit this lesion.

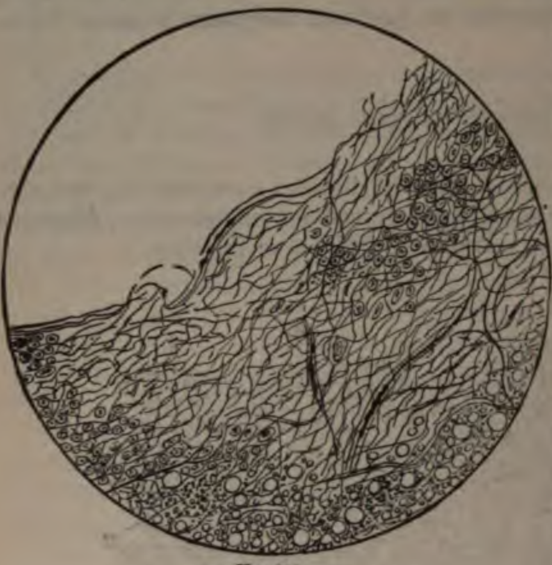

FIG. 204.

Sclerosis of the Brain.

Section of Cerebrum through a patch of sclerosed tissue.

The normal brain structure has entirely disappeared, and is replaced by interlacing fibrils of connective-tissue, in the meshes of which are shown small nucleated cells, atrophied nerve fibres and fat granules. × 300.

Etiology.—The causation of cerebral sclerosis is obscure. It unquestionably is intimately connected with changes in the *vascular* system, for the localities in which it is developed are the terminal arteries, *i. e.*, arteries that do not anastomose, or anastomose slightly. Sclerosis is often found in epileptics and in the insane. It is occasionally met with in advanced life.

Symptoms.—The symptoms of cerebral sclerosis are a gradual enfeeblement of the mental powers, *especially memory*, muscular tremors, headache, dizziness and vertigo. Accompanying these, one group of muscles after another becomes paralyzed. There is no regular order in the development of the paralysis, first a lower, then an upper extremity, then some of the facial muscles are involved. Melancholia, pains in the extremities, and a sense of formication are common. The nutrition is rarely interfered with; many patients gain flesh. Convulsions and disturbances of special sense are rare. Strabismus may be present.

A peculiar symptom is *festination*,—the patient bends forward and trots along like one trying to run after he is tired out. Late symptoms are paralyses of the muscles of deglutition, speech and respiration. In rare instances the first and only symptoms are convulsions of an epileptiform character, followed by hemiplegia.[1] Labio-glosso-pharyngeal paralysis may exist in sclerosis. Electrical reactions are not changed, when the cord is uninvolved. Inanition, emaciation, muscular contractures, and, rarely, an unexplainable rise in temperature, precede death, which occurs in collapse with loss of consciousness.

Differential Diagnosis.—Sclerosis of the brain may be mistaken for *cerebral softening, paralysis agitans*, or *tumors*.

Softening occurs in old age; the paralysis is in one set or group of muscles, and if it extends, does so in an *orderly* manner. There is *anæsthesia*, and the symptoms develop more suddenly than in sclerosis.

Paralysis agitans is marked by rhythmic tremor passing from one upper to the corresponding lower limb; there is a peculiar deformity of the fingers and toes; the facial muscles are not affected, and the patient inclines to the paralyzed side in walking. Paralysis agitans occurs only after the fortieth year, and is accompanied by *no cerebral symptoms.*

Cerebral *tumors* are attended by headache, convulsions, and signs of brain irritation without loss of mental power.

Prognosis.—Sclerosis of the brain may continue from five to eight years, but it is progressive and always fatal. Death may occur from inanition, or complications such as pneumonia, bed-sores, pleurisy, tuberculosis, marasmus, or cerebral paralysis.

Treatment.—Little can be done for this disease except to improve the general health. Yet it should be mentioned that Vulpian recommends chloride of iron, Mitchell the bi-chloride of mercury, Hammond the chloride of barium, and many the phosphite of zinc. Nitrate of silver and strychnia are said to relieve tremor.

HYPERTROPHY OF THE BRAIN.

Cerebral central hypertrophy is an increase in the neuroglia (the nerve filaments and ganglia are uninvolved), and may be partial or general. The term cerebral hypertrophy is really a misnomer.[2]

Morbid Anatomy.—On removal of the skull-cap the brain protrudes beyond the cranial bones. The skull-bones are thinned. If the disease begins very early in life, the head may become as large as in congenital hydrocephalus, and the sutures will be separated. The convolutions and sulci are lessened by pressure, and the membranes are thin and dry. The dura mater may be adherent. The ventricular fluid is absent. Intense anæmia always exists; the brain matter, both white and gray, is *white*, and tough and elastic. The brain is heavier than normal. The cerebrum is usually involved. But hypertrophy of the cerebellum, thalamus opticus, corpus striatum, pons Varolii, and medulla oblongata may also occur.

Etiology.—Hypertrophy of the brain may be congenital. It appears, when *not* congenital, in childhood before the third year. It may be hereditary, *i. e.*, it may occur in several members of the same family. It may be the result of traumatism, lead poisoning, chronic alcoholismus, or epilepsy. It not infrequently accompanies idiocy and insanity. In children, swelling of the lymphatics and thymus gland, and the evidences of rickets precede or coexist with its development. Dwarfs are often the subjects of cerebral hypertrophy.

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[1] Charcot states that cerebral sclerosis not infrequently commences with nausea, headache, vertigo, syncopal and apoplectiform attacks, followed by diplopia, amblyopia, nystagmus, disturbances of mind and of speech.

[2] Virchow in 1862 and 1867 published his article on hypertrophy of the brain; his views were based on the results of autopsies, and to these we are indebted for our knowledge of this subject.

Symptoms.—Virchow makes two forms, *acute* and *chronic*. In the former, headache, epileptiform convulsions, retardation or great acceleration of the pulse, vertigo, delirium, sympathetic vomiting, dyspnœa, and dysphagia occur. In *children* there is weakness, tremor, and a tottering gait, and the head inclines to one side. Convulsive movements of the eye or arm occur. There may be permanent strabismus. The child may be very precocious at first; later he becomes feeble-minded or idiotic. Periodical laryngeal spasm (thymic asthma) may occur. Bulimia is marked; and the child is constantly somnolent. The tongue, often larger than normal, protrudes from the mouth, and children often persistently suck it. Headache is rarely absent. It is steadily progressive, and *ends in coma*, preceded by dilated pupils, slowed pulse, vomiting, and repeated convulsive attacks.

Differential Diagnosis.—It is very often impossible to differentiate between *chronic hydrocephalus* and hypertrophy of the brain. In hypertrophy the child has been, or is, bright and precocious; in hydrocephalus he is *always* stupid. The fontanelles pulsate in hydrocephalus; they do not in hypertrophy. The cerebral souffle may be heard in hypertrophy but not in hydrocephalus.

Prognosis.—It always terminates in death. It may end either by progressive stupor or from complications.

ATROPHY OF THE BRAIN.

Atrophy of the brain may be either *infantile* or *senile;* it is never met with in adult life.

Morbid Anatomy.—In children the disease begins *in utero*. The skull is oblique; one-half is thick, smaller than normal, and misshapen. The corresponding parts of the brain are atrophied, hard, altered in color, and are studded with collections of serum. (Rosenthal.) Atrophy, or absence, of the corpus callosum is the result of defective fœtal development. It is generally accompanied by intra-uterine hydrocephalus. Physiologically, the brain begins to diminish in weight after the sixtieth year; at that time it is one-fifteenth lighter than during early adult life. Hence slight atrophy is physiological in old age. But in *senile atrophy* there is more or less marked diminution in the anatomical elements of the brain, and a loss in the interstitial connective-tissue. The cells of the cortex are swollen and pigmented; and pigmentation also occurs in the walls of the vessels, which often undergo more or less fatty degeneration. The cortex is thinned, and in it are found *corpora amylacea*. The fat in the cerebral substance is lessened, the water increased. Senile atrophy is usually general; but when partial it affects the left hemisphere. There is unequal thinning of the convolutions, and the sulci are large and deep. The meninges are somewhat clouded. The brain is usually tougher than normal, and the ventricles contain from two to twelve drachms of fluid. This is a purely conservative process. The ependyma is granular and nodular. More or less serum distends the meshes of the pia mater. The medullary substance and the corpora striata are riddled with holes, *état criblé*.[1]

On *section* the brain has a leathery toughness; it may be corrugated. The cortex is of a dirty gray color; and the medullary substance is a dull white or *drab* color. Partial atrophy may extend to the cortex, or it may follow the fibres through the peduncles, pons Varolii, and pyramids. A crossed lesion may sometimes be met with, due to atrophy of one cerebral and of the *opposite* cerebellar hemisphere. There are remarkable instances of diminution of the cerebellum to one-half its normal size and weight.

The loss in weight of the brain in the *general paralysis of the insane* is greater than in any other disease. The cerebellum and basal portions of the cerebrum are unaffected; the most striking degree of atrophy being in the frontal lobes, the convolutions exhibiting it most of all. Of all the changes accompanying this form of atrophy those in the *dura* are most marked; it is adherent to the bone, and hemorrhages into its substance are frequently met with. Pachymeningitis, hæmatoma, ruptures and foldings of the dura over the brain are often met with. Some regard these as the specific lesions of general paralysis. The *pia* mater is œdematous and thickened, either continuously or in patches.

Etiology.—Cerebral atrophy may be congenital or occur as a part of senile change. It may follow cerebral hemorrhage or softening, and is occasionally caused by tumors, meningeal inflammation, and internal or external hydrocephalus. Injury or destruction of the peripheral nerves may induce secondary cerebral atrophy. Excess in venery, the opium habit, and alcoholismus are adduced as its causes. It is met with oftener in males than in females. Senile marasmus is its chief cause.

Symptoms.—Senile cerebral atrophy is attended by gradual failure of the mental faculties. Memory is impaired, the special senses are markedly dulled; and the movements, at first unsteady, are soon accompanied by tremor. The patient is somnolent; indeed, he sleeps the greater part of the time. Soon the condition popularly known as "second childhood" is reached. There is often more or less complete loss of power over the sphincters. Atrophic degenerations of that half of the body on the same side as the atrophied *cerebellum*, or on the *opposite* side to the atrophied cerebrum are apt to occur. *Incomplete* paralysis accompanies these atrophic changes. Epileptiform attacks are quite frequent; choreic attacks occasionally occur.

Only a brief mention can here be made of the symptoms of the extensive *atrophy* which occurs in general paralysis of the insane. Headache, dizziness, irritability of temper, weakness of memory pre-eminently, thickness of speech, change in the character of the voice, a feeling of self-importance, grandeur and great riches,—these are very common. Sudden and uncalled-for outbursts of rage are common. It is very much like senile dementia.

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[1] Durand-Fardel and Parchappe.

Atrophy of the brain ultimately involves the medulla, implicates the great life-centres situate therein, and deglutition or respiration is so much interfered with that death results. In general paralysis of both sides of the body there is usually *complete imbecility*.

Differential Diagnosis.—Senile cerebral atrophy may be mistaken for ***cerebral** hemorrhage* and *softening*. The history of the case is essential in its differential diagnosis. Atrophy of the *cerebellum* may be mistaken for *tabes dorsalis* and *multiple sclerosis*. From the former it is diagnosticated by the absence of vesical symptoms and by more intense pains; the anæsthesia about the dorsal vertebræ is a valuable point, as it is always present in tabes and not in atrophy. The intra-uterine variety of cerebral atrophy is easily recognized by the paralyses and spasmodic seizures that occur directly after the birth of the child.

Prognosis.—Congenital atrophies, or those occurring in the early life, usually terminate during the fourth year. Senile atrophy is steadily progressive to a fatal termination. No estimate of its duration can be made. It may be complicated by hypostasis in the lungs, bronchitis, pulmonary œdema, pneumonia, acute bed-sores, or by disease of the bladder or kidneys. In the general paralysis of the insane its duration is rarely more than a year. Death is reached by intercurrent apoplexies, exhaustion from large bed-sores, anæmia or pulmonary complications.

Treatment.—Improvement of the general health is regarded as the most important indication. Some advocate exercise and massage of the paralyzed limbs. Niemeyer recommends cold douches. In atrophy in general paralysis, galvanism, iodide of potash, calabar bean, morphia, and chloral, alone or together, prolonged tepid baths, and attention to the bowels and bladder have been recommended.

DISEASES OF THE SPINAL CORD.

Diseases of the spinal cord and its membranes will be considered under the following heads:

I. *Spinal Hyperæmia.*
II. *Spinal Meningitis.*
III. *Acute Myelitis.*
IV. *Chronic Myelitis.*
V. *Non-Inflammatory Softening.*
VI. *Acute Bulbar Paralysis.*
VII. *Progressive Bulbo-Nuclear Paralysis.*
VIII. *Infantile Spinal Paralysis.*
IX. *Acute Spinal Paralysis of Adults.*
X. *Chronic Anterior Myelitis.*
XI. *Progressive Muscular Atrophy.*
XII. *Cerebro-Spinal Sclerosis.*
XIII. *Locomotor Ataxia.*
XIV. *Spasmodic Tabes Dorsalis.*
XV. *Amyotrophic Lateral Sclerosis.*
XVI. *Pseudo-Hypertrophic Paralysis.*
XVII. *Spinal Apoplexy.*

HYPERÆMIA OF THE SPINAL CORD AND MENINGES.

Hyperæmia of the spinal cord may be active or passive.

Morbid Anatomy.—The most intense active or passive hyperæmia of the spinal meninges may disappear between death and the time of a post-mortem, and a gravitation from position may induce a congestion which is decidedly misleading. In active hyperæmia the arterioles are injected and the parts assume a rosy color marked possibly by numerous points of extravasation. In passive hyperæmia the veins of the cord and membranes are distended with dark blood. Chronic congestion results in thickenings, pigmentation and opacities of the membranes, attended by development of new connective-tissue which may be the starting-point of a general sclerosis.

Etiology.—*Active hyperæmia* may result from muscular exertion, or excesses in venery, from vaso-motor paralysis due to exposure to cold and wet, concussion, and suppression of menstrual or hemorrhoidal fluxes. Hyperæmia is always an attendant of general or local myelitis. It occurs with acute infectious diseases, typhoid, small-pox, scarlet fever, and measles, with chronic malarial infection, and in some cases of rheumatism and puerperal fever. It may also result from poisoning by carbonic oxide, strychnia, nitrite of amyl, alcohol, etc.[1] Intense active hyperæmia has been found in those who have died of spasmodic affections, or who have worked in compressed air, as in caissons.[2]

Passive hyperæmia is caused by any obstructive disease of heart, liver, or lungs, and by mechanical pressure upon venous trunks, by tumors, fluid effusion, etc.

Symptoms.—The onset of active hyperæmia may be sudden, while passive hyperæmia generally comes on slowly and often insidiously. In most instances there is pain along the spine, especially in the lumbar region, which radiates down the thighs and is increased by movement and pressure. There is hyperæsthesia of the lower limbs associated with itching, burning, or formication, and reflex irritability is augmented.

Hyperæsthesia, sharp pains, spasms, and symptoms of irritation would rather point to *active* hyperæmia, while numbness, anæsthesia, heaviness of the limbs and vesical paresis are more commonly associated with congestion.[3] In rare cases the disease is so sudden in its onset that the patient may awake to find himself in a state of incomplete paraplegia. During the whole course there is no fever and little, if any, change in the pulse. Dyspnœa occurs when the nerve roots are involved high up in the cord.[4] Quite often during attacks of spinal congestion, persistent priapism occurs and the iron-band sensation about the waist is a frequent symptom.[5]

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[1] Magnan has experimented with absinthe on animals, and found it to produce intense hyperæmia when given in large doses.

[2] *St. Louis Med. and Surg. Jour.*—Dr. Clark.

[3] Rosenthal states that violent emotions and sometimes the *dorsal decubitus* increase the rachialagia.

[4] Steiner reports a case where facial paralysis occurred.—*Archiv der Heilk.* 11, 1870, p. 233.

[5] Fabre has observed pain, anæsthesia, hyperæsthesia, slight paresis, and, rarely, convulsive phenomena occurring in the last stages of heart disease, which seemed to be due to *passive* hyperæmia of the cord.—*Gaz. des Hôp.*, 1876, No. 147.

When convulsions occur in such diseases as tetanus there is intense hyperæmia of the gray matter of the cord.[1]

Differential Diagnosis.—Hyperæmia of the cord may be mistaken for *spinal anæmia, meningitis, myelitis* or *apoplexy.*

In *anæmia* the symptoms are relieved by the recumbent posture, while they are increased in hyperæmia. Anæmia occurs suddenly, as from embolism and thrombosis. Women suffer most frequently from anæmia, men from hyperæmia. Vesical complications follow congestion, but not anæmia.

In *inflammation of the cord* there will be fever, paraplegia, paralysis of the sphincters, loss of electro-contractility, with bed-sores and subsequently wasting of the muscles.

The onset of *spinal apoplexy* is sudden, paraplegia is complete within a few hours, and accompanied by anæsthesia, paralysis of the bladder and rectum, the early development of gangrenous bed-sores, and in most cases by the symptoms of cystitis and myelitis.

Prognosis.—The prognosis is favorable, although the condition is one which has a marked tendency to become chronic. Complete recovery is slowly reached, except in those cases where the cause is permanent.

Treatment.—If its cause can be reached, it should at once be removed. Severe and sudden congestion demands local abstraction of blood by wet cups along the spine or leeching about the anus. The patient should be kept quiet on his side. In recent cases ice-bags to the spine, hot foot baths, and a brisk purge will relieve the pain. Ergot and belladonna and hot douches along the spine are highly advocated in chronic passive hyperæmia. In reflex hyperæmia, or hyperæmia due to vaso-motor disturbances, electricity may afford relief.

SPINAL MENINGITIS.

Spinal meningitis may be acute or chronic.

Morbid Anatomy.—Acute inflammation of the spinal meninges is generally diffuse, and runs a course similar to acute cerebral meningitis. The pia mater is hyperæmic, swollen and studded with ecchymoses. The exudation takes place into the meshes of the membrane, and the effusion may be sero-fibrinous, or purulent. The pia mater is thickened, opaque, and œdematous, and a turbid fluid fills more or less completely the spinal canal. Although the exudation is more abundant upon the posterior surface, it usually envelops more or less completely the whole cord, whose substance may either be pale and anæmic, or exhibit changes of commencing myelitis and softening. The roots of the nerves are embedded in the exudation, and present changes similar to those in the cord. The exudation may be wholly absorbed, and the membranes and the cord return to their normal condition ; but more frequently the inflammation becomes *chronic.*

In *chronic spinal* meningitis there are found the opacity, thickening, adhesions, and puckerings so characteristic of chronic interstitial inflammation in similar membranes. The membrane is tough, dark, bluish-gray in color, pigmented, and contains calcareous plates. It is adherent at various points to the thickened dura mater (pachymeningitis spinalis). The fluid in the spinal canal is always increased in amount; it may be clear serum, or contain lymph flocculi, blood, or pus. The irregular and localized adhesions and retractions produce sclerosis of the cord, and also induce anæmia, atrophy, and degeneration of the nerve roots.

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[1] Feinberg.

Etiology.—Spinal meningitis is a disease of youth and early adult life, and may follow a traumatism, as in a fall, blow, dislocation, fracture, or other injury to the vertebræ, or concussion, which is thought to be a frequent cause. It may arise from extension of inflammation from the cerebral meninges, from any disease of the spine, such as caries, cancer, etc. Prolonged exposure to cold—especially damp cold—or brief exposure to intense cold when the body is heated, as well as exposure to intense heat, will induce spinal meningitis. Operations for spina bifida have been followed by rapid and fatal spinal meningitis.

Rheumatism is said to be an occasional cause, and some authors regard all febrile and infectious diseases as liable to be complicated by it.[1] Syphilis, venereal excesses, alcoholismus, chorea, tetanus, and hydrophobia may each, in rare instances, induce spinal meningitis;[2] and scrofulosis, tuberculosis and wasting diseases are very apt to be complicated by it.

The *chronic* form is often a sequel of the acute, and is very apt to accompany alcoholismus, syphilis, impeded venous return, and diseases of the cord. The latter are the most frequent of all causes. Chronic or acute inflammation of the cord, or any neoplasm that encroaches upon the spinal canal, will lead to *localized* chronic meningitis. Excessive use of tobacco or narcotics, and anti-hygienic surroundings are predisposing causes.

Symptoms.—When spinal meningitis is associated with cerebral inflammation its symptoms are less distinct than when it is uncomplicated. Severe pain in the back is the earliest and most prominent symptom. The pain at first is localized about the seat of the inflammation, but later becomes diffused and shoots down the legs and arms. It is constant, and is made sharp and lancinating by motion, so that the patient holds himself in a fixed position with rigidly contracted muscles; pressure along the spine may increase the pain. A chill or distinct rigor accompanies the pain, and is followed by rise in temperature, nausea, vomiting, and a sense of general malaise. The fever is never high and the pulse-rate is frequently below the normal. The muscles along the spine become rigid, and if the cervical region is involved there is opisthotonos. Convulsive twitching of groups of muscles is attended by the most excruciating pain. The surface of the body becomes hyperæsthetic in the area of motor derangement, and reflex activity is increased. In a few instances all the extremities are involved, but usually there is only incomplete paraplegia. There is constipation, and the abdomen has the well-known boat-shaped appearance.

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[1] C. B. Radcliffe, in Reynold's *System*.

[2] Koehler states that any pulmonary or cardiac disease that impedes proper venous return affords a marked predisposition to spinal meningitis.

At the commencement of the attack there is a constant desire to micturate. Later, paralysis of the bladder and retention of urine accompany the para-paresis, so that catheterization must be resorted to. If the paralysis involves the respiratory muscles there will be dyspnœa, and the temperature will rise to 106° or 107° F., and be followed by coma and death. If the meningitis is limited to the lower portion of the cord, the case will be protracted, but marked by periods of slight improvement. In such cases bed-sores may develop, with incontinence of urine, and death finally occur from exhaustion. Sudden and profuse sweats may result from vaso-motor implication.

Chronic spinal meningitis is generally a sequel of acute, although it *may* develop without any acute symptoms. When the acute passes into the chronic form, pain and rigidity of the spine remain after the other symptoms have subsided. The limbs are hyperæsthetic, and the seat of burning, formication, or itching. There may be a sensation as of a tight band about the waist, accompanied by weight and uneasiness in the limbs, which may develop into incomplete paraplegia. The bowels, at first, are constipated, but later the passages may be involuntary. The bladder is frequently paralyzed, incontinence of urine occurs, bed-sores form, and a well-marked marasmus is developed. Finally the paralysis will vary in degree with the posture of the patient—and also from day to day.

Differential Diagnosis.—Acute spinal meningitis may be confounded with *myelitis, tetanus,* and *muscular rheumatism.* In *tetanus* the locked jaw, the peculiar implication of the facial muscles causing the *risus sardonicus,* and the intense cutaneous hyperæsthesia, with recurring paroxysms without paralysis, are in marked contrast to the symptoms of spinal meningitis. In the latter disease there is great pain on motion, little or none on pressure, and muscular spasm is produced by attempts at movement rather than by irritation. There is, usually, a traumatic history in tetanus.

Rheumatism in the muscles of the *back* is accompanied by local pain only when movements are made; but there is never that rigidity of the spine which is present in meningitis, nor the cutaneous hyperæsthesia, paralysis, spasms, or febrile phenomena.

Spinal irritation may be mistaken for meningitis, but the pain on pressure confined to one spot, the absence of pain upon ordinary or slight motion, and the disposition to a sudden transference of the diseased action from one organ or part to another, with possibly the occurrence of hysterical symptoms, will be sufficient to distinguish between them.

Chronic meningitis may be mistaken for *chronic myelitis.* In meningitis *pain* is a far more prominent symptom than in *myelitis;* and it is increased by motion but not by pressure, the reverse of which occurs in myelitis. Paralysis is never complete in meningitis, and anæsthesia and muscular atrophy are rare as compared with myelitis.

Prognosis.—Acute spinal meningitis runs a variable course; should death occur within twenty-four or thirty-six hours the case is to be regarded as epidemic. The usual duration of the acute form is from seven to ten days. The majority of cases are fatal, more especially those in which the membranes of the cervical cord are involved. In these cases, death results from paralysis of the muscles of respiration, and will be preceded by intense dyspnœa and cyanosis. Even when recovery occurs, convalescence is tedious; and though the general health is restored, pain, paralysis, and stiffness or atrophy of muscles are apt to continue for many months. Rarely is convalescence rapid. In many acute cases death occurs from simple asthenia.

Chronic spinal meningitis runs a very protracted course, and may terminate in death from exhaustion, anæmia or marasmus. It progresses by stages; and although the prognosis is not so unfavorable as in the acute form, complete recovery is rare. Both acute and chronic are most severe in the very young or the very old and enfeebled.

Treatment.—The same principles guide the treatment of acute spinal meningitis as were advised in acute cerebral meningitis. The patient should be placed in bed in a cool room and a brisk purge administered. Ice or counter-irritation may be applied along the spine, and from the onset the patient should be kept in a condition of semi-narcotism. Ergotin and belladonna hypodermically are said to produce contraction of the arterioles and restrain the inflammatory process. The internal administration of the iodide of potash with mercury is advocated. Warm baths are grateful to the patient, and produce a sedative effect, and have seemed to me to be of greater service than all other measures. The nourishment should be highly nutritious but never stimulating. When symptoms of heart failure or asthenia come on, stimulants are indicated. Careful attention to the condition of the bladder should never be neglected.

In *chronic spinal meningitis* counter-irritation over the spine, and derivatives to the surface are to be employed as long as the inflammatory processes are in progress.[1] Warm douches are excellent adjuvants, and in some cases are followed by marked benefit. Iodide of potash and mercury—the latter both internally and by inunction—are more clearly indicated in chronic than in acute cases. The galvanic current is often of service in preventing the muscular atrophy and contractions which are sequelæ of the paralyses.

ACUTE MYELITIS.

Myelitis is an inflammation of the substance of the spinal cord, and may be limited to the gray or white matter; it runs an acute or chronic course, and involves the whole or isolated portions of the cord.

When the gray matter alone is involved, it is called *central myelitis;* when the white matter and the meninges are involved it is called *cortical myelitis.*

When once established the disease may be ascending, descending, or transverse in its extension.

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[1] Brown-Séquard advises sinapisms, stimulating ointments, and oils, moxa, and in severe cases, white-hot irons to the spine.

Morbid Anatomy.—In acute myelitis the portion involved is softened and discolored to an extent corresponding to the amount of vascular dilatation and transudation of red blood cells. Hemorrhage takes place into the softened spots, although in many cases it is doubtful whether the hemorrhage preceded or followed the softening. In most cases the cord is enlarged, and on section, blood points and spots of ecchymoses are seen. The veins especially are distended and surrounded by a layer of red and white blood corpuscles.

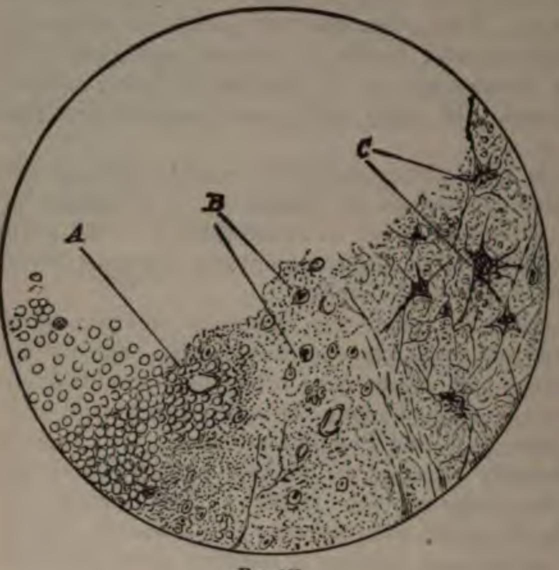

FIG. 205.

Acute Myelitis.

From a Section through the Dorsal Spinal Cord, including portion of the Anterior Gray Cornu.

A. Patch of ecchymotic tissue.
B. Nerve fibres with swollen axis-cylinder.
C. Hypertrophied and pigmented ganglion cells, with ampullar dilatation of nerve fibrils.

Microscopically there will be found swelling of the cells of the neuroglia, ampullæ-like dilatations of the axis-cylinders, hypertrophy of the cells in the anterior horn of the gray substance, and an albuminoid granular degeneration of the nerve fibres. The nerve cells not infrequently show pigment degeneration, and the ganglion cells are clouded and swollen.

These morbid processes result in entire disappearance of the normal anatomical elements of the cord. The adjacent membranes will be congested, thickened, opaque, and adherent to the cord, while collections of blood or pus underneath the membrane may cause it to present a nodulated appearance. These changes are most marked in the gray matter of the dorsal and lumbar regions. Acute red softening soon becomes yellow from fatty degeneration, from changes in the coloring matter of the blood, and from diminution in vascularity. Later, as its consistence diminishes, a pale yellow or white diffluent mass is left.[1]

Etiology.—Acute myelitis is a disease of children and young adults. In children it takes the form of acute anterior polio-myelitis or spinal paralysis. Exposure to excessive heat or cold, intense and prolonged muscular exertion, and excessive venery are said to predispose to it; and in children, dentition is regarded as a predisposing cause. Myelitis may be excited by traumatism, lying on the damp ground, and exposure to sudden chilling of the surface when overheated. Whether suppression of the menses, checking hemorrhoidal fluxes, or profuse perspiration of the feet can cause it is uncertain. Pressure on the cord, from tumors or displacements of the bony parts, whether occurring suddenly or developing slowly, will induce myelitis, or it may be excited by extension of inflammation, especially from spinal meningitis.

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[1] Erb states that this softening is due to fluid exudation from the vessels, and destruction of the nerve fibres.

Myelitis also arises during the course of small-pox, measles, scarlet and typhoid fevers, acute articular rheumatism, malignant pustule, puerperal fever, and syphilis. Continued jarring of the spine from travel on railways will induce it.[1] Visceral disturbances—*especially of the genito-urinary* and *digestive organs*—and diseases of the joints are said to act as reflex causes. In many cases the myelitis comes on without any assignable cause.

Symptoms.—Acute myelitis usually commences with slight febrile symptoms, pain in the back, a peculiar sensation of an iron band around the waist, and the pulse is frequently feeble and irregular. Anorexia, headache and general malaise usually precede the attack. The power of motion in the lower extremities is rapidly lost, and soon complete paralysis occurs, which is usually both sensory and motor. Patients will complain of a sense of numbness in the feet; they cannot feel the ground under their feet, and they have a sensation as if something was crawling over their legs. Retention of urine and fæces, which marks the onset, gives place to incontinence, from paralysis of the sphincters. Tremulous and spasmodic movements often occur in the limbs that are subsequently paralyzed; and at the commencement of the paralysis their temperature is elevated. Electro-muscular contractility is diminished.

The pains in the back are increased by pressure, and localized at certain vertebræ. The application of heat or cold over the sensitive spot produces pain; and a warm or cold sponge at the junction of the normal and anæsthetic parts produces a *burning* sensation, felt in a line around the body. When the paraplegia is sudden and *complete*, hemorrhage into the softened focus may be suspected. The paralysis frequently extends rapidly upward, and when the cervical cord is involved paresis and anæsthesia of the arms, irregularities of the pupils, dyspnœa and dysphagia will be present; the pain in these cases is located in the neck. The itching, burning, or boring pains in the limbs and the sense of formication that precede the paraplegic symptoms are rarely influenced either by pressure or motion. Reflex action is diminished or lost, and its abolition is an indication of the extent to which the gray matter is involved.

Trophic or vaso-motor disturbances appear early, causing acute bed-sores, œdema of the paralyzed limbs, effusions into the joints, and more or less muscular atrophy. The urine becomes alkaline and often bloody. Retention is of frequent occurrence and results in cystitis and pyelitis; uræmic symptoms may appear, and sepsis often occurs from the bed-sores and gangrenous inflammation. Among the later manifestations are darting pains, spasmodic twitchings and contractions, either of isolated groups or of all the muscles in the paralyzed part.[2] This marks its passage into the chronic stage. In some few cases hemi-paraplegia is induced by myelitis. The disease is always progressive. In some classes the paraplegia may be so rapidly developed that in forty-eight hours the patient will be unable to lift or move his legs.

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[1] Ollivier, Hine, and Leyden regard mental shock, especially from fright or anger, as a cause.

[2] The spinal epilepsy of Brown-Séquard is a spasm of all the muscles of the lower extremities generally following transverse myelitis.

Differential Diagnosis.—*Acute myelitis* may be confounded with *acute spinal meningitis, hysterical paraplegia* and *paraplegia* from *reflex urinary irritation.*

In *meningitis* there is acute pain *on motion,* with rigidity of the muscles of the back; in myelitis there is *no pain* on motion and the muscles are flaccid and relaxed. Paralysis in spinal meningitis is incomplete, but paraplegia or hemi-paraplegia is always present in acute myelitis. Cutaneous and muscular hyperæsthesia, with febrile and cerebral symptoms, exists in meningitis, but is absent in myelitis.

Hysterical paraplegia is diagnosticated by the attendant hysterical symptoms, globus hystericus, large flow of limpid urine, jactitation, etc. It is not a true paraplegia, and generally occurs in young women.

In paraplegia from reflex irritation, genito-urinary troubles will *precede* the paraplegia; in myelitis the urinary symptoms *follow* the paraplegia. In reflex urinary irritation the paraplegia is incomplete and does not extend upwards; in myelitis it is complete and increasing. There is no paralysis of the sphincters in reflex irritation; in myelitis it is an early and marked symptom. There is no girdle sensation, no formication, or sense of swelling and heat in reflex paraplegia; while these symptoms are always present in myelitis. The urine is acid in reflex, and alkaline in myelitic paraplegia. The muscles are atrophied in myelitis; and normal in reflex paraplegia. Myelitis of the *cervical* portion of the cord is attended by paralysis of *all the extremities,* increase in reflex irritability, dysphagia, dyspnœa, vomiting and impaired speech. When the whole cervical region is involved the upper extremities are first implicated, and they *lose their reflex irritability.* The pharyngeal, thoracic, and ocular symptoms are also marked. The pulse is rapid and irregular.

Prognosis.—In acute myelitis death may occur in twelve to thirty hours, or be delayed two or three weeks. When the disease is protracted a month it becomes chronic. Complete recovery is rare; incomplete recovery occurs quite often. Cervical myelitis is the most, dorsal the least unfavorable. Bed-sores, cystitis, nephritis, and pyelitis, or high fever and sudden and complete paralysis, render the prognosis exceedingly unfavorable.

Treatment.—The most important thing in the treatment of acute myelitis is absolute rest. Ergot and belladonna have been highly recommended, but I have never obtained any positive results from their use. Blisters and other counter-irritants, electricity and strychnia are contraindicated. Spinal bags filled with hot water have seemed to me to give the greatest relief to this class of patients. Diuretics and mild cathartics should be given; and catheterization practised from the outset. If the myelitis is of syphilitic origin, iodide of potassium may be of service, but not otherwise. A supporting, nourishing plan of treatment is to be adopted from the onset. To prolong life, complications must be prevented as far as possible. Bed-sores must be prevented by great cleanliness and the daily use of the galvanic current; cystitis may be avoided by the frequent use of the catheter and the washing out of the bladder.

CHRONIC MYELITIS.

Under this term are included a variety of changes in the cord, of which white softening is perhaps the most frequent.

Morbid Anatomy.—When chronic myelitis is the sequela of acute, the change to white softening marks the entrance into the chronic stage. In this stage, by a process precisely similar to that which occurs in the brain, a cyst is formed. It is divided by numerous septa of connective-tissue, and contains fluid resembling chalk and water. After absorption the cicatrix is gray, shrivelled, and pigmented. Less commonly, though by no means infrequently, there is hyperplasia of the neuroglia, and a dense, gray, sclerosed focus remains. Large cells with numerous processes, called Deiter's cells, are seen in this sclerosed tissue, and the ganglion cells are found atrophied. Large quantities of corpora amylacea are formed. Usually the cord is hard and gray, but in many cases it appears to the naked eye perfectly normal, while the microscope reveals chronic myelitis.[1] It is slightly diminished in volume, and the atrophy may be uniform, or irregular, and at scattered points.

Chronic inflammation of the meninges with progressive atrophy of the roots and trunks of the peripheral nerves is met with in chronic myelitis. There is increase in the connective-tissue of the neuroglia, and degeneration of the nerve fibres. The ganglion cells are hard and pigmented, and large Deiter's cells are abundant. The axis cylinder remains intact for a long time.[2] Fat cells are everywhere present, and in cases of very long standing large excavations in the substance of the cord may occur.

It is impossible to distinguish interstitial from parenchymatous myelitis.[3]

Etiology.—All the constitutional causes that were enumerated as causes of acute may be included under the remote causes of chronic myelitis, and of these chronic alcoholismus, sexual excesses, and reflex disturbances are more liable to result in chronic than acute myelitis.

Symptoms.—The symptoms of chronic myelitis are so complex that Charcot calls it a "polymorphous" disease. It is usually insidious in its onset; and in its development disorders of sensation precede motor disturbances. Pains in the limbs simulating rheumatism are gradually associated with muscular weakness; and tingling, formication, numbness of the limbs, with the girdle sensation, are followed by an unsteady gait. Local anæsthesia alternates with hyperæsthesia. Weakness of the bladder and constipation are both the result of muscular weakness. These symptoms are followed by paraplegia, muscular atrophy, cystitis, and chronic bed-sores. Slight tremors and twitchings of the muscles are not uncommon. Patients with chronic myelitis always complain of cold feet.[1]

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[1] So-called *gray degeneration*.

[2] Charcot and Leyden.

[3] Leyden, in the *Zeitschr. f. Klin. Med.*, Berlin, 1879, No. 1, p. 1-26, recites a most interesting case, where numerous large round nucleated cells were found pushing apart nerve fibres in the posterior dorsal region of the cord of a man who had been poisoned in a caisson. Recently "*systems*" have been described in the cord, and some pathologists have classified diseases of the cord on this physiological basis. Leyden describes two forms of system disease of the cord, where chronic myelitis is the sole lesion, *i. e.*, tabes dorsalis, and atrophy of the motor parts of the cord. These two combined give a *combined system disease*. Regeneration of destroyed nerve fibres in the cord is *possible* though very rare.

There is usually progressive emaciation and cachexia. Some cases remain stationary for months and even years; but the majority reach a fatal termination through successive exacerbations and remissions.

Differential Diagnosis.—Chronic myelitis may be confounded with *spinal apoplexy, spinal meningitis,* or *locomotor ataxia.*

It is distinguished from *hemorrhage* by the sudden advent of the hemorrhage, and from *meningitis* by the absence of pain.

In *locomotor ataxia* the double heel-and-toe tread, the neuralgic pains, the preservation of motor power, of control of the sphincters and sexual force, all stand in contrast to the signs of chronic myelitis.

Prognosis.—The prognosis in chronic myelitis is always unfavorable. It may continue from two to ten years, but in no case can there be complete recovery. It may remain stationary also; but the functions are never restored. Death results from cystitis and pyelitis, bed-sores, and other complications.

Treatment.—Rest is the most important remedial agent. When a cause—such as lead poisoning, disease of bladder, uterus, etc.—can be reached it must be removed. Dry cups daily to the spine are usually of service. Ergot, belladonna, nitrate of silver, iodide of potash, arsenic, phosphorus, and strychnia have all been recommended and benefit claimed for them, as may be said of hot or cold baths at natural springs.[2] The bladder must be emptied twice or three times daily and the bowels kept freely open. The galvanic current is considered beneficial, or at least harmless. Friction, shampooing, and *massage* of the paralyzed limbs prevent wasting of the muscles.

NON-INFLAMMATORY SOFTENING.

Our knowledge of this rare condition is vague. Indeed, until recently, its existence was denied.

Morbid Anatomy.—The site, extent and limitation of non-inflammatory softening are the same as in myelitic patches. Myeline, broken-down nerve-tubes, large granulation corpuscles, are all found in the patch. Radcliffe describes white softening as non-inflammatory and due to anæmia.

Etiology.—Slowed blood-current, a tendency of the blood to spontaneous coagulation, and disease of the walls of the blood-vessels are regarded as causes of non-inflammatory spinal softening.[3] It is sometimes met with within the month after childbirth, in the late stages of syphilis, after great bodily exertion, sexual excess, and exposure.

Symptoms.—The symptoms of non-inflammatory softening do not differ essentially from those of chronic myelitis, spinal hemorrhage, and spinal tumors. Its invasion is generally gradual, and the complexity of symptoms varies according as the foci are circumscribed or diffuse, central, lateral, or completely transverse. If the softening extends completely across the cord, there is complete paralysis of the lower extremities and of the abdominal muscles. The limbs are cold to the touch and their temperature is sub-normal. The toes are turned inward, so that as the patient lies in bed the feet form a cross. The skin becomes dry and rough, and the muscles are flabby although not wasted. There is almost complete abolition of reflex movement. Early in the disease there is retention of urine, which is followed by incontinence and partial retention, and the usual sequelæ of cystitis and possibly pyelitis or ammonæmia; the kidney may become studded with minute abscesses.

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[1] Erb states that catheterization and dressing the bed-sores produces varied movements in the paralyzed limbs.

[2] Erb, Rosenthal and others.

[3] Dr. Moxon calls attention to the fact that the blood supply at the lower end of the cord—where softenings are most frequent—is peculiar and easily interfered with.—*Brit. Med. Jour.*, vol. i. 1881.

Differential Diagnosis.—Its slow onset and the previous history enable us to differentiate between non-inflammatory softening and *spinal hemorrhage.* It is not difficult to determine the extent of the lesion; but to determine whether it is central or peripheral, anterior or posterior, is always difficult and often impossible.

Prognosis.—The prognosis is always unfavorable. There is great danger of intercurrent diseases, especially pneumonia, local or general meningitis, and inflammation of the genito-urinary tract, or septicæmia. In rare instances the paralysis may gradually disappear and partial recovery take place.

Treatment.—No plan of treatment is successful. The bowels and bladder must be attended to, and the latter is best washed out with a one-half per cent. solution of chlorate of potash. The galvanic and Faradic currents may be used.

ACUTE BULBAR PARALYSIS.

While acute bulbar paralysis involves a nervous distribution similar to that of the chronic form, its morbid anatomy is very different.

Morbid Anatomy.—At the autopsy there will be foci of softening and extravasation from thrombosis and embolism. Erb states that there is an acute bulbar paralysis not due to these causes, but which is in reality a primary *acute myelitis bulbi.*

Etiology.—The etiology of acute bulbar myelitis is unknown, aside from the causes of apoplexy, embolism, and thrombosis elsewhere in the cerebrospinal system.

Symptoms.—Its onset is very *sudden;* the prominent symptoms are headache, dizziness, and sometimes loss of consciousness (apoplectiform variety). Cough, dyspnœa, and hiccough are often present, and sometimes there are convulsions and weakness in the limbs accompanied by tingling sensations. In other cases coma and asphyxia precede the rapidly fatal issue. Paralysis is often best marked in the distributions of the bulbar nerves. This may affect the facial, trigeminus, accessorius, or glossopharyngeal. In many cases the paralysis is "crossed," the hemiplegia being on the opposite side from the paralysis of the bulbar nerves. Very rarely the hemiplegia is crossed, the arm of one side and the leg of the other suffering. In occlusion of the basilar artery the carotid pulse is unusually full. The paralysis is usually paraplegic, but may be hemiplegic. Occasionally it will begin severely on one side, and after a few days pass to the other one as the primary paralysis improves.

Paralyses of the muscles of the pharynx and fauces are common in all forms of bulbar disease. Rosenthal states that, in addition to dyspnœa, Cheyne-Stokes respiration often appears as a characteristic symptom of medullary hemorrhage.[1] When the extravasations extend into the fourth ventricle, polyuria and albuminuria are observed.[2] In embolism, improvement is common, but in hemorrhage it is rare; thrombosis of the vertebral arteries pursues a more chronic course, but with similar results.

Prognosis.—If the patient recovers from the primary effects of the lesion, the prognosis of the paralysis is better than in the chronic form. The prognosis is better when the disease is associated with or due to syphilitic infection. In a few acute cases the paralysis is permanent, although it has no tendency to increase. In sudden and complete obstruction of the basilar or both vertebrals the prognosis is exceedingly bad. Limited or capillary hemorrhages render the prognosis unfavorable.

Treatment.—This does not differ from the treatment of similar conditions elsewhere in the brain, which has been considered under apoplexy and cerebral softening.

CHRONIC BULBAR PARALYSIS.

(*Glosso-labio-laryngeal Paralysis.*)

This is a progressive, symmetrical paralysis of the lips, adjacent facial muscles, tongue, pharynx, and sometimes of the larynx.

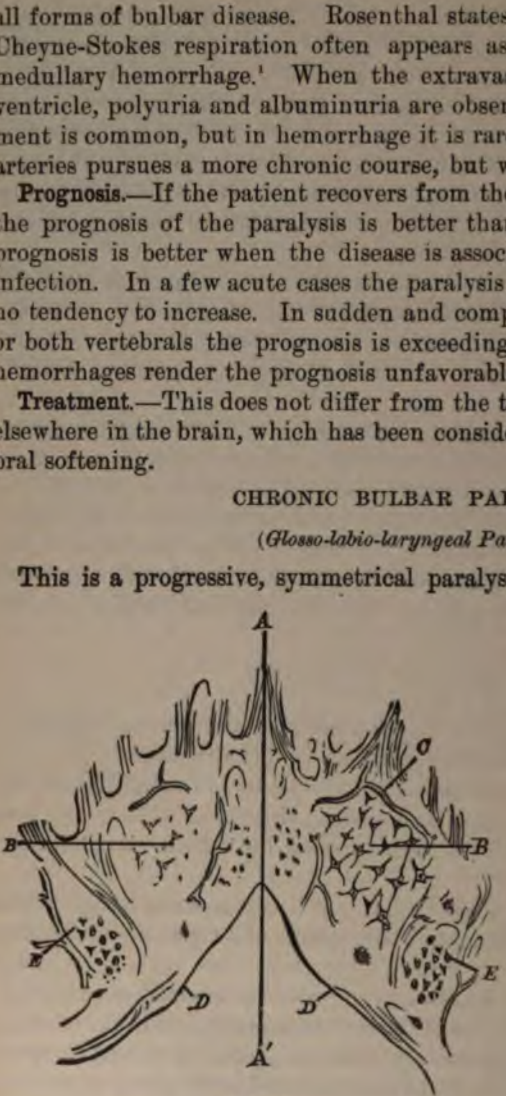

Fig. 206.

Chronic Bulbar Paralysis.

Transverse section of the bulbus on a level with the middle of the nucleus of the hypoglossus.

AA′. Line dividing the section centrally. Morbid condition on the left hand.
B. Ganglion cells forming nucleus of hypoglossus.
C. A vessel forming front and inner boundary of B.
D. Floor of fourth ventricle.
E. Nucleus of pneumogastric.
On the left the nucleus of the hypoglossus is nearly obliterated, while that of the pneumogastric is unaltered. Charcot.

Morbid Anatomy.—The medulla may be atrophied and show spots of gray discoloration which have a sclerotic feel. There is degenerative atrophy of the gray nuclei in the floor of the fourth ventricle; with atrophy and gray discoloration of the nerve roots from the medulla, especially of the facial and hypoglossal nerves.[3] The ganglion cells and the nerve-nuclei lose their stellate form and become shrunken, smaller, and of a dull ochre color. The prolongations and nuclei are rudimentary or even completely atrophied. The cells are filled with pigment and granular matter, the nucleus and nucleolus present a vitreous, shining appearance,[1] and are separated from each other by large spaces. Atrophy and disappearance of the motor ganglion cells is always to be noted. It may be the sole lesion or be accompanied by increase in the neurolgia, when fat and granular corpuscles, numerous corpora amylacea, Gluge's corpuscles, and spider-cells will be found in the newly-developed tissue. The walls of the vessels are thick, and show more or less fatty change. The decrease in size of the gray nuclei is a measure of the intensity of the symptoms that existed during life.

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[1] See also accounts by Traube in the *Berlin. Klin. Wochen.*, 1869 to 1874.

[2] *Gazette des Hôpitaux*, 1862.

[3] Recent physiological investigations show that the lower facial nucleus and the hypoglossal nucleus are closely connected.

Similar bilateral lesions may be found in the nuclei of the pneumogastric, spinal-accessory, glosso-pharyngeal, facial, trifacial, motor oculi, and, very rarely, of the trigeminus.[2]

The muscles are pale and the fibres frequently show granular degeneration; but sometimes fatty tissue is in excess. The fibres may be thin, and the tissue between them contain the pigment products of degeneration, so that the muscles, though degenerated, will preserve their normal bulk.

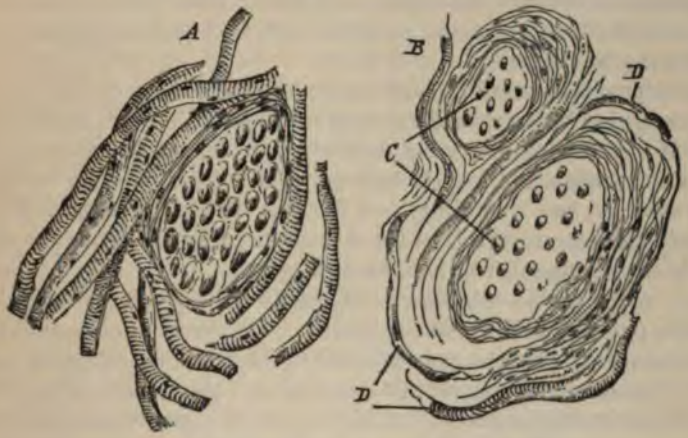

FIG. 207.
Chronic Bulbar Paralysis.

A. Microscopical appearance of normal muscle from the tongue.
B. Same muscle taken from a case of Glosso-labio-laryngeal Paralysis.
C. Fasciculi of muscular fibres in transverse section.
D. Atrophied fibres seen longitudinally. × 300.

The muscular fibres show increase in their nuclei and changes precisely similar to those in progressive muscular atrophy.

The nerves going to the muscles exhibit sclerosis of the neurilemma, slight traces only of the axis-cylinders remaining. The same degenerative atrophy is found in the nerve roots coming from the bulb. This disease is rare before the fortieth year of life; it is essentially a disease of old age. Males are more subject to it than females.

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[1] Yellow degeneration of Charcot.

[2] Duchenne.

Etiology.—Its etiology is always obscure. The neuropathic tendency seems to exercise some influence in certain cases. It is said to occur with syphilis and rheumatism. Bad hygiene, exposure to cold, excessive anxiety, close mental application, and prolonged physical exertion are all adduced as causes.

Symptoms.—The earliest symptoms of bulbar paralysis are imperfect movements of the tongue; the speech is indistinct, and enunciation of the palatal and dental sounds is imperfect. Often the tongue cannot be protruded as far as normal, nor the lips brought together as perfectly or separated as promptly as in health. Whistling and whispering are impossible. The lower part of the face becomes expressionless. The lips remain separated and the saliva is either tenacious or dribbles from the mouth, which is so drawn as to give the face a woe-begone expression. Speech may be entirely lost.

When the palate muscles are involved, deglutition becomes difficult, and as the soft palate hangs motionless, not closing the posterior nares, the food regurgitates through the nose, or lodges in the upper part of the pharynx and collects between the cheeks and the alveolar arches; portions are also apt to fall into the larynx. Another effect of palatal paresis is to give a nasal twang to the voice.

If the laryngeal muscles become weakened and closure of the glottis imperfect, coughing becomes ineffective and phonation is interfered with. The muscles of mastication are rarely involved until late in the disease, but exhaustion and emaciation from insufficient food are developed early. The muscles atrophy, and tremblings and fibrillar twitchings occur. When the respiratory centres are involved there is a sense of fulness and constriction in the chest accompanied by attacks of dyspnœa.

Lesions in the cardio-inhibitory centres are followed by attacks of syncope and a pulse of 140 or 160 per minute. There is an abnormal amount of saliva secreted, either as paralytic saliva[1] or from irritation in the medulla.[2] The laryngoscope reveals paralysis of the vocal cords. Should bronchitis occur expectoration is difficult, and if pneumonia or any other severe pulmonary affection develops it almost always terminates fatally. The muscles show the reaction of degeneration; sensibility is unchanged, but reflex actions are greatly diminished or destroyed. Atrophy of the muscles at the back of the neck—the trapezii especially—is not infrequent. In some few cases the paralytic symptoms may be preceded by dull pains in the back of the head and neck, giddiness, queer sensations in speaking, and loss of reflex irritability in the pharynx and larynx.[3]

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[1] See M. Foster's *Physiology*. Art. *Sub-maxillary Gland*.

[2] Pflüger's *Archiv*, Bd. 7.

[3] Kirchoff reports a case where bulbar paralysis was produced by a *unilateral* lesion, and from the autopsical results in this case the symptoms must be attributed to a lesion of the lenticular nucleus. A new and peculiar variety of bulbar paralysis has recently been described by Erb. An analysis of the symptoms shows that the parts chiefly involved are the motor oculi communis, the motor portion of the trigeminus, the spinal-accessory, and the upper cervical nerves; and those more slightly affected are the facial (the upper branches to the face), the hypoglossal, and probably also the glosso-pharyngeal nerves. The nuclei of origin of these nerves are all situated in the floor of the fourth ventricle and in its immediate neighborhood in the pons Varolii. Erb supposes that, in the affection under consideration, the lesion is situated in the upper half of the fourth ventricle and spreads more deeply into the substance of the medulla, affecting nerve fibres as they pass upward from the nuclei of origin in the fourth ventricle.

Differential Diagnosis.—Progressive bulbar paralysis may be mistaken for *tumors in the medulla, double facial palsy, embolism and thrombosis of the medulla, medullary apoplexy, embolism* of one of the *vertebral arteries, progressive muscular atrophy* attacking the *face*, and *general paralysis* of the *insane*.

In *tumors of the medulla*, we find neuralgia, clonic convulsions of the muscles of the face and tongue, disturbances of the smell and hearing, headache, vomiting, dizziness, and epileptiform attacks, the disease being either *unilateral* or decidedly marked only on one side.

In *double facial palsy*, *all* the branches of the facial nerve are involved; movements of the tongue and deglutition are normal.

In *embolism* or *thrombosis* of the *medulla*, the sudden onset of the symptoms with either hemiplegia or paraplegia, taken in connection with the age of the patient and the condition of the arteries, will be the chief points of diagnosis. In embolism improvement is possible.

In *bulbar hemorrhage*, loss of consciousness, epileptiform convulsions, vomiting, prominence of unilateral symptoms are combined with bulbar paralysis of sudden advent. The previous history will aid in the diagnosis.

Embolism of the *vertebral arteries* is accompanied by sudden (apoplectiform) onset of the symptoms of bulbar paralysis, hemiplegia, anæsthesia, variations in the paralyses, and co-existing disorders of sight and hearing.

In *progressive muscular atrophy*, paralysis *follows* the atrophy; in bulbar paralysis it is the reverse. Moreover, the thenar and hypothenar eminences are involved early, even should muscular atrophy first attack the tongue, lips and palate.[1]

In *general paralysis* of the *insane*, the cerebral disturbances and the fact that other muscles are involved besides those in the region of the mouth and palate, will establish the diagnosis.

Prognosis.—The prognosis is grave; although a temporary arrest may occur, genuine bulbar paralysis invariably terminates in death. The amount of dysphagia and dyspnœa and the rapidity of development will determine the relative gravity of the case. Its average duration is about two years. Bulbar paralysis may be complicated by progressive muscular atrophy, amyotrophic lateral sclerosis and disseminated sclerosis. Death occurs from starvation, paralysis of the heart or respiratory organs, or intercurrent pulmonary diseases. Sometimes coma ends the scene, and there is a slight rise of temperature.

Treatment.—A nutritious diet and the best hygienic surroundings, with quinine, arsenic and nitrate of silver are the means which have been most extensively employed. The German physicians condemn, and the English advocate, the use of strychnine and phosphorus. Ergot, belladonna, and iodide of potash may be given. Direct galvanic or Faradic currents applied to the paralyzed parts have been recommended. In some cases a stomach tube may be used to prolong life, and perhaps *gastrostomy* may be demanded.

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[1] Duchenne.

INFANTILE SPINAL PARALYSIS.

Infantile spinal paralysis or *acute anterior polio-myelitis* is an inflammation of the anterior cornua of gray matter of the cord. It may occur in adults, but is almost always exclusively confined to children.[1]

Morbid Anatomy.—The *early* changes are those of inflammatory softening; medullary hyperæmia and vascular exudations are the incidental occurrences.[2] Extensive changes may exist, and yet the gross appearance of the cord be unchanged.

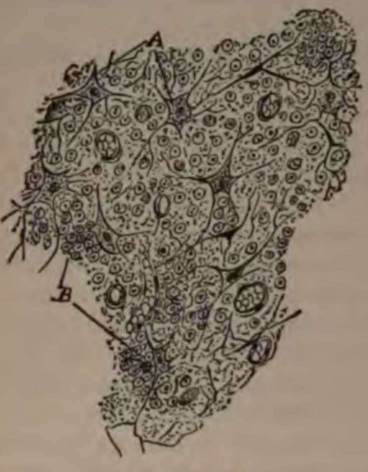

Fig. 208.

Anterior Gray Cornu of Spinal Cord in Early Stage of Infantile Spinal Paralysis.

A. Large multipolar ganglion cells.
B. Neuroglia cells in a state of active proliferation. × 300.

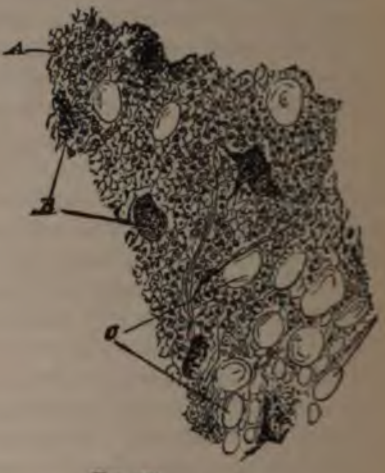

Fig. 209.

Anterior Gray Cornu of Spina Cord after establishment of the Sclerotic Process in Infantile Spinal Paralysis.

A. Dense nucleated connective-tissue.
B. Large masses of pigment—the remains of ganglion cells.
C. Corpora amylacea. × 300.

Microscopically there will be seen, in its early stage, all the changes of acute interstitial inflammation, and the neuroglia nuclei are in active proliferation. Later a sclerotic process is established, and new connective-tissue developed, in which are multitudes of nuclei and corpora amylacea. Pigmentation is more or less marked, and the ganglion cells, that have lost their processes, may remain only as irregular spherical masses of pigment. Thickening and increase of neuroglia in the anterior columns result in more or less atrophy of the nerve fibres. The antero-lateral columns of the cord may be invaded, but the posterior usually escape.[3] The anterior roots of the spinal nerve are shrunken, atrophied, and degenerated. They are gray and translucent. The vessels undergo considerable enlargement, and their walls are thickened. The motor nerves are involved secondarily to the cord at an advanced period of the disease. The *muscles* which are implicated rapidly undergo fibroid changes and atrophy. Their transverse striæ are indistinct, and the nuclei become abundant; the muscular fibres may wholly disappear.[1] The muscular fibres do not *always* suffer this degeneration, but sometimes they undergo fatty degeneration, or the muscles are so infiltrated with oil globules that they retain their normal size, and may even exceed it; this is a pseudo-hypertrophy.[2] The *bones* are retarded in development, somewhat flexible, and contain more fat than usual. The *tendons* become atrophied, and the *joints* lose their compactness.

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[1] Niemeyer calls it essential palsy. Erb describes it as a more or less diffuse myelitis of the anterior gray substance, which reaches its greatest intensity in the *lumbar and cervical* enlargements of the cord, and, as a rule, leaves no permanent and irremediable alteration except at those two points.

[2] Rosenthal.

[3] The most careful microscopical examination fails to decide what modern pathology is still earnestly discussing, viz.: whether acute polio-myelitis is an interstitial or a parenchymatous inflammation. The majority favor the *latter* view, the ganglion cells being its supposed starting-point.

Etiology.—This is essentially a disease of the first three years of life, the usual time of occurrence being between the sixth and fourteenth months. It attacks equally children of both sexes, the robust as well as those of feeble and cachectic constitutions. Cold, dentition, and traumatism are among its doubtful causes. It has occurred in two or more offspring of the same parents, and once in twins[3] after an attack of measles. Many regard acute febrile diseases as an important factor in its causation. It is developed, if at all, during convalescence from such fevers.

Symptoms.—The onset of infantile spinal paralysis is *sudden.* A child has well-marked febrile movement attended by dizziness, headache, restlessness, nausea, vomiting, and sometimes delirium, convulsions and coma. Accompanying these symptoms there is more or less pain in the back. In many cases the febrile symptoms only last a few hours. Following a convulsion or attack of unconsciousness the child becomes *paralyzed,* or in some cases paralysis may come on suddenly without *a single premonitory symptom;* the child goes to bed perfectly well and wakes with paraplegia. If only one lower limb is involved at first, the other soon becomes so; and it is not unusual for *all four* extremities to be affected simultaneously. The arms alone are rarely involved. The paralysis is not accompanied by loss of *sensibility.* It reaches its maximum in from ten hours to six or seven days, and begins to diminish in about two weeks after its commencement. The paralyzed muscles become flaccid, relaxed, and attenuated, and if the paralysis is persistent they atrophy and undergo degeneration. The surface of the body is cold and of a purplish color. The limbs may preserve their normal contour; but they are soft and often tender to pressure. The tendon-reflexes and reflex action in the muscles of the paralyzed part are entirely lost, and they fail to respond to the Faradic current. These alterations in electrical excitability are results of *the reactions of degeneration.*

Paralysis at the onset is general, but later it is localized in one group or in single muscles. The muscles on the back of the forearm and front of the leg, in the foot, and the extensors of the leg, are more apt to be affected, but the paralysis may involve only the deltoid, tibialis anticus, sterno-cleido-mastoid, or the extensor longus digitorum. The joints are loosened, and the bones, especially the long ones, are smaller and *shorter* than those in the unaffected limb. The temperature of the paralyzed parts is often 5° or 8° F. lower than normal. The deformities and unnatural attitudes that result may simulate talipes; and all varieties of contracture occur as late manifestations. The epiphyses atrophy and subluxations sometimes occur. The general health of the patient is usually good, and there is nothing that interferes with long life, except the paralysis and deformity. In severe cases there is at the onset loss of control of bladder and rectum. In such cases slight vesical weakness usually continues during life. The normal sensibility of the skin is preserved throughout.

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[1] Erb lays great stress upon the replacement of muscle tissue, in this disease, by fat, so that the muscles have the faded-leaf appearance of a typhoid heart.

[2] Erb.

[3] Moritz Meyer.

Differential Diagnosis.—Infantile spinal paralysis may be mistaken for *progressive muscular atrophy, pseudo-muscular hypertrophy, rachitis, temporary infantile paralysis, myelitis,* and *hemiplegia.*

Progressive muscular atrophy begins insidiously and is slowly progressive; spinal infantile paralysis begins suddenly, and after a time a certain amount of improvement occurs. Progressive muscular atrophy is rare in children before the fifth or seventh year of age. It commences by palsy about the lips and mouth, and the electro-contractility of the affected muscles is lost only in proportion to their atrophy and degeneration, the *uninvolved fibres responding to the current.* This fact taken in connection with the age of the patient will usually enable one to make a diagnosis.

Pseudo-muscular hypertrophy begins *without* fever; the motor power at first is only weakened, and the trunk and *extremities* are involved late in the disease. The electro-muscular contractility is preserved, and there is always increase in the volume of the muscles. In walking the patient spreads the feet far apart, and there is a peculiar incurvation of the vertebral column not seen in infantile spinal paralysis.

Rickets is attended by no change in electro-muscular contractility, is preceded by no cerebral or pyretic phenomena, and there coexist developmental and other changes that cannot fail to determine the character of the deformity.

In *temporary paralysis* there are no signs of softening or atrophy of muscles, there is no change in electro-muscular contractility, and the paralysis is recovered from in twenty to thirty days.

In *myelitis,* trophic disturbances and genito-urinary complications are sufficient to distinguish it from infantile spinal paralysis.

Hemiplegia from acute cerebral affections in childhood can generally be distinguished from acute anterior polio-myelitis by loss of intelligence and speech, strabismus, paralysis of half the face, dilated pupils, and normal electrical contractility in connection with disturbances in sensation, stiffness of the joints, spasmodic contractures with absence of fever, and muscular atrophy.

Prognosis.—There is little or no danger to life in acute anterior polio-myelitis, even when the attack commences with very active symptoms. A mild, or even a severe, onset may be followed by complete restoration of the function and power of the paralyzed muscles:—so-called temporary spinal palsy. Usually the improvement is such that the function of the few muscles that remain permanently paralyzed and atrophied is performed by the muscles not involved. All the paralyzed muscles in which Faradic irritability is not completely lost are restored.[1]

Treatment.—In the acute stage rest in the recumbent posture is the most important element of treatment. Beyond this the treatment is the same as for acute myelitis. After the febrile symptoms have subsided—usually by the fourth week—measures must be adopted to restore the function of the paralyzed muscles. The early and persistent use of the galvanic current hastens the recovery in those muscles whose electric contractility is but slightly diminished, and will often arrest the wasting and restore them to a normal condition. When electric contractility is entirely lost little benefit can be expected. The longer the use of electricity is delayed the less the chances of recovery. Even if it fails to cure it has a tendency to prevent deformity. Saline and thermal baths and the water treatment of various kinds are recommended. Massage, friction, shampooing, inunctions, etc., are to be combined with the electric and hydropathic plans of treatment. The diet must be such as to bring nutrition to its highest point. Minute quantities of strychnia injected hypodermically have been found beneficial. Iron, arsenic, quinine and phosphorus are indicated—as tonics—in nearly every case.

ACUTE SPINAL PARALYSIS OF ADULTS.

Duchenne and Moritz Meyer first observed that this disease not infrequently occurred during adult life, with pathological changes identical with those of the disease in infancy.

The **etiology** is obscure; cold, wet, and the debility found in convalescence from fevers, pneumonia, malarial poisoning, etc., have been suggested as causes.

The **symptoms** at the onset are modified by the greater stability of the adult nervous system, and there is less restlessness, delirium, fever, etc. The cerebral symptoms may be very slight, transient, and easily overlooked and followed by paralyses, which go on to partial recovery as in the infantile form. There are no bone deformities or arrested developments; and the joints do not become lax. Tingling, numbness, and formication occur in adults at the onset, and gastric symptoms are more frequent.

Differential Diagnosis.—Absence of spasms and of trophic disturbances, diminution of reflexes, normal sensibility, non-interference with the sphincters, the sudden onset and the subsequent improvements suffice to distinguish this disease from all other affections of the cord.

Chronic atrophic spinal paralysis resembles it, but the abrupt invasion of anterior polio-myelitis is absent in the former malady. If this point in the history be wanting a differential diagnosis may be impossible.

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[1] Seeligmüller records two cases where progressive muscular atrophy occurred late in life in those who in infancy suffered from acute anterior polio-myelitis.

The *prognosis* and *treatment* are the same as in children.

CHRONIC ANTERIOR POLIO-MYELITIS.

Duchenne was the first to describe (1853) this disease under the above name. It has since been called subacute and chronic inflammation of the gray anterior horns, chronic atrophic spinal paralysis, and subacute spinal paralysis.

Morbid Anatomy.—The morbid anatomy of this disease is still obscure. So far as can be stated from the few recorded autopsies it is simply a chronic myelitis of the anterior cornua; the neuroglia is increased; the blood-vessels are thickened, the anterior nerve-roots are atrophied, and there is an abundance of granular and fat cells in the diseased district. Recently vacuoles have been found in the ganglion cells of the anterior horns. Almost entire disappearance of these cells was the chief lesion in one case.[1]

Etiology.—It is a disease of adult life from thirty to fifty, and excesses of any kind, or exposure to cold and wet, are said to exert an influence on its development similar to that in other spinal affections.[2]

Symptoms.—In some subacute cases slight fever and shooting pains in the back accompany the development of paralysis of the lower limbs. In others the patient first notices weakness and heaviness in his legs, followed by paralysis either of groups of muscles or of the whole limb. The muscles become flabby and progressively waste away. They are sensitive to the galvanic current, but respond little, if at all, to the Faradic. The irregular distribution of the paralysis is characteristic. As the muscles are undergoing atrophy, fibrillary twitchings are often noticed. Tendon-reflexes and skin-reflexes are both abolished; but sensibility is unaffected. There is vaso-motorial disturbance, indicated by cold and blue extremities. The temperature of the affected limbs is lowered. Later, the upper limbs are involved. The paralysis first attacks the flexors or extensors on the forearm, and gradually involves isolated groups of muscles, or the whole limb. The fingers and hand, however, suffer most. When the disease has reached this stage the wasted muscles will no longer respond to the galvanic current. The rectum, bladder, and sexual power are undisturbed. When the process extends to the cervical region dyspnœa is present, and if the medulla becomes involved deglutition and articulation are affected, and great exhaustion is induced, asphyxia closing the scene. The general health remains good, and the mental faculties are unimpaired.

Differential Diagnosis.—Chronic atrophic spinal paralysis may be mistaken for *progressive muscular atrophy*, *amyotrophic lateral sclerosis*, *acute ascending paralysis*, and the *acute spinal paralysis of adults*.

In *progressive muscular atrophy* paralysis *follows* the wasting; the reverse is the case in polio-myelitis. Portions of the muscles only are involved in progressive muscular atrophy, and it begins in the muscles of the thumb. Reflex action is retained, and the progress is much slower than in chronic polio-myelitis. Moreover, the susceptibility to the electrical currents is never wholly lost in progressive muscular atrophy.

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[1] *Arch. de Physiologie*, 1876.

[2] Erb suggests that its co-existence with chronic lead poisoning is the result of an inflammatory action called forth by the saturnismus.

In *amyotrophic lateral sclerosis*, though the upper extremities may be wasted, there is a characteristically different combination of paralysis *without* wasting, and with more or less rigidity in the lower extremities. The reaction of degeneration is far more marked in chronic anterior polio-myelitis. Reflex clonus and exaggerated tendon-reflexes are absent in chronic polio-myelitis, and present in amyotrophic lateral sclerosis.

In *acute ascending paralysis* the atrophy is not marked ; electrical reactions of nerves and muscles are normal, reflex action is preserved for a long time, and bulbar symptoms with vesical disturbances are not uncommon. Acute ascending paralysis is of short duration compared with chronic anterior polio-myelitis.

In *acute spinal paralysis* of adults the paralysis, which is sudden in its onset, is more extensive, and after a short time there is improvement in motor power ; while in chronic atrophic spinal paralysis there is a *distinctly* progressive unremitting spread of the disease from part to part.

Prognosis.—In rapidly progressive cases the prognosis is bad, but in those that are slowly developed and partial the prognosis is better, and sometimes complete recovery may take place, or certain muscles or groups remain paralyzed and atrophied while others improve. After a long time the disease may be spontaneously arrested and the patient remain paralyzed the remainder of life. The most unfavorable cases are those in which the cervical region and the medulla become involved, death occurring with symptoms of bulbar paralysis. The usual duration is from a few months to three or four years.

Treatment.—Electricity and a nourishing diet, with rest, give the best results. Dr. Bastian suggests that counter-irritation may do good in the early stages. Sulphur, mineral, and brine baths and the cold water treatment are advocated. It is a question whether either iodide of potassium or ergot is beneficial. Modern literature, though extremely rich in theories, is devoid of facts which can aid in the treatment.

PROGRESSIVE MUSCULAR ATROPHY.

As the name indicates, this disease is a progressive and chronic wasting and atrophy of the muscles, and results from trophic changes due to a central nerve-lesion.

Morbid Anatomy.—The morbid anatomy of this affection differs little from that in spinal paralysis of children. Its essential lesion is atrophy of certain groups of nerve-cells in the anterior cornua of the cord. Sometimes atrophy of the anterior horns is associated with a sclerotic condition of the lateral columns. The general changes are the same as in the late stage of anterior polio-myelitis. The central canal of the cord is sometimes dilated and filled with fluid. On microscopic examination the ganglion cells show pigmentation to a marked degree, with more or less atrophy. They are surrounded by indurated tissue. The blood-vessels are often dilated, and surrounding them is a zone of granular disintegrated or diffluent material, the so-called *mixed exudation*. All these changes may be found in both gray and white matter. The anterior roots of the spinal nerves are atrophied, and show more or less gray degeneration. Sometimes all except the neurilemma has disappeared. The muscles over the body are not equally involved; indeed, bundles of fibrillæ in the same muscle are affected in different degrees. This uneven character of the atrophy *is peculiar to* this disease. The muscles simply waste, and become pale or of a faintly yellow hue. They are harder and firmer than normal. The striæ disappear only after great reduction in size. The interstitial structure is increased and filled with numerous lymphoid cells. Fatty and granular degeneration may occur later, with fatty infiltration, and if the fat-globules are present in large quantity the muscle *may not be reduced in size*. Granular disintegration is soon followed by transformation of the muscular tissue into fine fat-granules.[1]

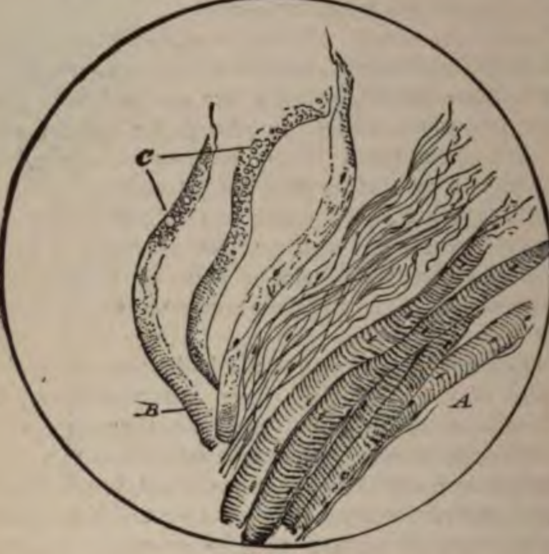

FIG. 210.
Teased Fibres from the Abductor Pollicis in a case of Progressive Muscular Atrophy.
A. Fibres from a normal bundle.
B. Fibres from a fasciculus adjoining A, atrophied and showing fatty degeneration. × 300.

In some cases progressive muscular atrophy has occurred without appreciable changes in the cord, and given rise to the belief that such changes were secondary to the muscular atrophy. It is very generally accepted, however, that the central lesion is the primary and characteristic change.

Etiology.—Progressive muscular atrophy is chiefly met with in adult males in middle life. Heredity and consanguinous influence can no longer be doubted.[2] Excessive physical labor, exposure to cold and wet, are said to excite it. Those who habitually use one set of muscles are perhaps predisposed to the disease. Injury to the spine is an important causative factor.

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[1] Virchow calls fat in the fibres *parenchymatous*, and fat in the interfibrillary tissue the *interstitial* form of degeneration.

[2] Leyden states that in hereditary cases the lumbar muscles and those of the lower limbs are *first* attacked; that it *may* appear in childhood, and that several members of the same family may be simultaneously affected.

Symptoms.—The invasion of progressive muscular atrophy is irregular and variable. It usually comes on insidiously, the first indication of its presence being a wasting and loss of power of some muscles or group of muscles. When regular in its course the wasting begins in the muscles of the hand; first the ball of the thumb of the right hand, then the hypothenar eminence and the interossei are attacked, in the order named. Marked atrophy of the interossei causes the hand to have the characteristic bird-claw look. The left hand is soon involved, and the wasting then slowly ascends, attacking the muscles of forearms, arms, shoulders, the pectorals, and latissimus dorsi, with symmetrical alternation. The arms may be reduced to skeletons of limbs, and the wing-shoulder is not uncommonly seen.

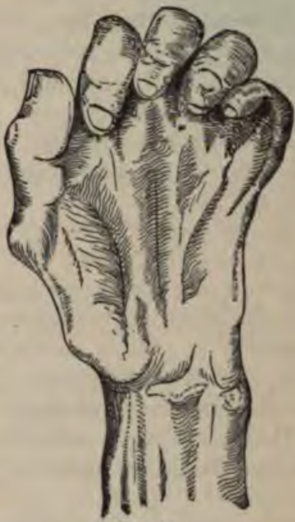

Fig. 211.
Sketch of a Hand in Progressive Muscular Atrophy.—Charcot.

When the muscles of the trunk are involved, those of respiration and deglutition are very likely to become implicated early. Sometimes the starting-point is in the thoracic muscles. Although the legs become extensively involved later, the wasting seldom begins there. These atrophic changes advance very slowly, and the patient will remember that he had for weeks or months a feeling of slight numbness or formication, and that his fingers have seemed clumsy. They also may complain—just before atrophy begins—of a sensation of cold air being blown on them; the hand and arm are very easily fatigued, and wandering pains not infrequently precede the wasting of the muscles. The parts to be attacked or those just involved are *colder* than normal. A peculiar fibrillary tremor—transient oscillatory movements in the fibres of the affected muscles—is present early. It occurs unknown to the patient, and may be excited by gently blowing or tapping on the skin. When the atrophy becomes extensive this ceases. The muscles respond promptly to the Faradic and voltaic currents, with a force in proportion to their bulk. The wasting in the muscles produces very different appearances according to the group involved; should those of the face become implicated, as frequently happens in children, the expression is stolid, grave, and unchangeable. Often, the head falls forward, and the saliva dribbles from the mouth. The speech may be faltering; and the tongue small and shrivelled. Mastication and deglutition may become difficult, and as the muscles of respiration are involved dyspnœa is urgent and asphyxia or pulmonary complications result fatally. Many state that the pupil on the affected side is much smaller than its fellow, and reacts to light but slightly.[1]

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[1] Rosenthal, Schneemann, Voisin, Menjaud and Bergmann.

In a certain number of cases agonizing pain along the nerves leading to the affected muscles occurs and is a prominent symptom throughout. Late in the disease atrophy of muscles proceeds so far that absolute immobility of a member is the result.[1] The general health is unimpaired, and the intellect is clear.

Differential Diagnosis.—Progressive muscular atrophy may be mistaken for *acute anterior polio-myelitis*, for *palsy* due to *injury of a nerve*, *lead palsy*, *malarial paralysis*, *post-paralytic atrophy of muscles*, and *sclerosis of the lateral columns.*

In injury of a *nerve* the atrophy is confined to the muscles supplied by that nerve, and is *not* progressive. In injuries of mixed nerves, sensation will also be lost.

In *lead palsy* the history of exposure, the blue line about the gums, and *the colic*, with the fact that the extensor muscles of the hand, rather than those of the thenar and hypothenar eminences, are first atrophied, causing the drop-wrist instead of a claw hand, and that their electric contractility is greatly diminished, are sufficient for a differential diagnosis.

In *malarial palsy* there is no muscular wasting, no tremor, and there are the attendant well-known malarial symptoms.

Muscular atrophy sometimes follows *paralysis*. But this fact alone, when the muscles do *not* respond to Faradization, excludes progressive muscular atrophy.

Symmetrical *sclerosis* of the *lateral columns*—amyotrophic lateral sclerosis—is, according to Charcot, distinguished by its rapid course, the ultimate affection of *all* the limbs, and the almost constant extension to the bulbar nuclei, by the prolonged preservation of electro-muscular contractility, and by permanent spasmodic contractures of the paralyzed and atrophied limbs. The symptoms of muscular atrophy are preceded in *amyotrophic sclerosis* by paralysis, and accompanied by rigidity; this does not occur in muscular atrophy.

Prognosis.—Progressive muscular atrophy is always a grave disease. Its course is slow and irregular; it may appear in the muscles of the hand, and years elapse before it extends. There is little hope of checking its advance, even if the treatment is commenced at its onset. The disease is arrested spontaneously in a few instances within two or three years. Complete recovery is rare. One year is the average duration. when recovery takes place. Its average duration is five years.[2] An hereditary element in the etiology renders the prognosis unfavorable, and when the disease is prolonged several years, or the muscles of respiration and deglutition become involved, a fatal termination is rarely long delayed. Inanition, bronchitis, pneumonia, and hypostatic congestion are the causes of death.

Treatment.—In the cases that have come under my observation no plan of treatment has had any beneficial effect. If an exciting or predisposing cause can be reached it should at once be removed. Damp, cold, and over-exertion should be avoided, and if syphilis be suspected, an anti-syphilitic treatment is indicated; and cod-liver oil, phosphorus, arsenic, and the mineral tonics are to be given with a highly nutritious diet. The body is to be warmly clothed in flannel; friction, *moderate exercise,* shampooing, massage, and inunctions are undoubtedly beneficial, if persevered in. Warm baths at natural springs are strongly recommended. Galvanism, however, is probably the most efficient remedy. The current should be applied along the spine, especially in the cervical region, and directly to the affected muscles. Faradization alternating with the constant current often leads to improvement and a temporary arrest of the disease.[1] The cramps and neuralgic pains are best controlled by hypodermic injections of morphine.[2]

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[1] Herpes has been observed along the line of a nerve going to an atrophied muscle, and Rosenthal mentions hypertrophy of the bones with concentric osseous atrophy, arthropathies, and bed-sores as rare trophic disturbances.

[2] Roberts, in *Reynolds System of Medicine.*

CEREBRO-SPINAL SCLEROSIS.

Morbid Anatomy.—On opening the spinal canal the cord is seen to be studded with well-defined nodules of sclerotic tissue which have given it the name of *nodular sclerosis.* These nodules are distributed irregularly throughout both the gray and white matter. They vary in number, and range in size from minute microscopical objects to the size of a walnut. They present a yellowish red, glistening appearance, are slightly elevated, semi-transparent, of a jelly-like or cartilaginous feel, and are marked by fine white lines. The meninges over the nodules are thickened and opaque, but seldom adherent to the substance of the cord. The sclerosed patches are well defined and easily distinguished from the normal tissue in which they are imbedded; still there is no abrupt transition from healthy to diseased tissue.

A *microscopical* examination shows the *centre* of the nodules to be a dense mass of very fine fibrillated connective-tissue, containing fat granules, corpora amylacea, Deiter's cells and small axis-cylinders which are glossy and brittle. The persistence of the axis-cylinders is regarded by Charcot as peculiar to dissem-

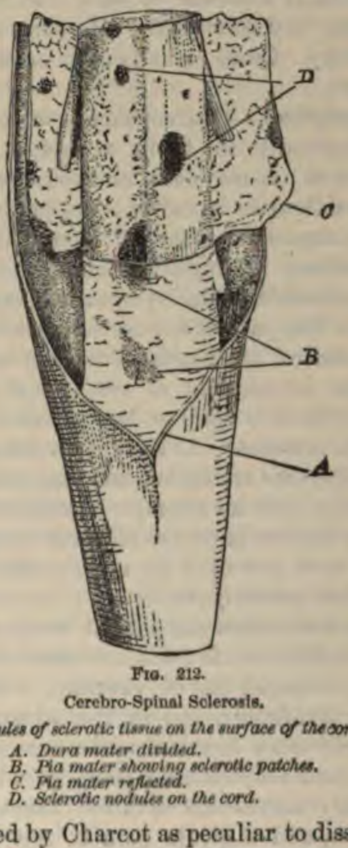

FIG. 212.

Cerebro-Spinal Sclerosis.

Nodules of sclerotic tissue on the surface of the cord.

A. Dura mater divided.
B. Pia mater showing sclerotic patches.
C. Pia mater reflected.
D. Sclerotic nodules on the cord.

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[1] Remak recommends galvanization of the fibres of the sympathetic nerve.

[2] Lockhart Clark hints that blistering and other forms of counter-irritation to the spine, *late in the disease*, deserve further trial.

inated sclerosis. Near the periphery the nerve tubes are surrounded by connective-tissue fibrillæ running parallel with the inclosed nerves, and there is commencing hyperplasia of the neuroglia, proliferation advancing more or less rapidly according to the age of the nodule. The walls of the vessels are thickened and infiltrated with numerous fat and lymphoid cells, but their lumen is notably diminished.[1] The nerve cells in the gray cornua are either primarily or secondarily involved and undergo cloudy swelling, followed by pigmentation or granulo-fatty degeneration,—the yellow degeneration.

Thus it appears that sclerosis is a primary, and multilocular chronic interstitial myelitis or encephalitis a secondary condition.

Etiology.—The recognized exciting causes of multiple cerebro-spinal sclerosis are damp and cold, sudden chilling of the body, traumatism, and severe, long-continued brain work or physical exercise. Continued jarring of the body is also thought to produce the disease, and it is said to occur in nervous people, with hysteria and after acute febrile diseases. It is essentially a disease of early life, few cases occurring outside the limits of fifteen and thirty-five. Heredity is said to play an important part in its etiology. Quite recently cases are reported as occurring in very young children.

Symptoms.—Charcot makes three varieties of this disease, according as it is confined to the brain, or cord, or involves both. The latter is the more common; it may come on insidiously or be sudden in its development. If it is insidious in its advent the patient complains vaguely of headache, *vertigo*, muscular weakness, mental disturbances, and queer feelings as formications, itchings, burnings, etc., in the limbs. The symptoms which are referable to the sympathetic system are nausea, vomiting, and cardialgia. The patient notices very soon a loss of co-ordinating power; he cannot control his hands in writing or his feet and limbs in walking. There is also impairment of the special senses. If the spinal element is prominent, there is more or less paresis of all the extremities, with contractures of the muscles. As soon as an attempt is made to use the paretic limbs, they become tremulous and contracted. This tremor is peculiar in not occurring until an attempt is made at *voluntary* motions, and at once ceasing when the parts are allowed to rest. It is called the *shaking tremor*. The more powerful the mental effort the more marked is the tremor. *Even the head participates in it.*

In some cases the patient becomes childish or morose, and the cerebral symptoms in such cases are identical with those of cerebral softening. During prolonged fits of yawning, sobbing, or laughing the *respirations become stridulous*, and in the advanced stages the voice is changed. The patient talks in a low monotone or whisper, dividing his words into syllables, and emphasizing them as when scanning a line of poetry. If the sensory nerves are involved there are pains in the course of the affected nerves, and a girdle pain is felt about the abdomen. Amblyopia, nystagmus, diplopia, and inequality of the pupils evidence invasion of the base of the brain and optic tracts. In the advanced stage vesical symptoms, acute bed-sores, and loss of sexual power and control of the sphincters become marked symptoms. Sometimes a sudden apoplectiform attack followed by paretic symptoms ushers in the disease. The course of the disease is peculiar. As its development is by stages, it may gradually progress for several years, and then remain stationary for a long period.

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[1] Charcot, Cornil and Ranvier maintain that in the sclerotic islands nerve-elements are *always* present. Frommann and Erb hold the reverse.—*Ziem. Cycl.*, vol. xiii.

Differential Diagnosis.—Disseminated sclerosis of the brain and cord may be mistaken for *paralysis agitans*, or *locomotor ataxia*, and when ushered in by apoplectiform symptoms may be mistaken for *cerebral hemorrhage*.

In *paralysis agitans* the *fine* tremor exists when the patient is at rest, and is not accompanied by shaking of the head; while in the shaking of disseminated sclerosis the head is always involved, the symptom ceasing as soon as the patient is at rest. Paralysis agitans is rare before forty; multiple sclerosis is rare *after* thirty-five. Changes in the voice and speech and ocular symptoms are present in disseminated sclerosis and absent in paralysis agitans.

In *locomotor ataxia* the peculiar tremor, impairment of voice and speech, and nystagmus that belong to disseminated sclerosis are *absent*. In the former disease we notice the peculiar iron-band sensation, vesical symptoms, the Menière's vertigo, the very slow and late appearance of paretic symptoms, the lightning-like and agonizing neuralgic pains, and the peculiar double beat in walking, the heel being put down first, all of which are in marked contrast to the symptoms of multiple cerebro-spinal sclerosis.

When disseminated multiple sclerosis is ushered in by loss of consciousness which rapidly deepens into coma, with marked hemiplegic symptoms, it may be mistaken for *cerebral hemorrhage*; but in sclerosis the temperature is very high during these peculiar attacks—104° or 105° F.—the hemiplegia passes off as the patient returns to consciousness or in a few days after, and the temperature rapidly falls to normal.

Prognosis.—This disease is usually of long duration, five to ten years being its average. It is rare for death to occur in coma. There is no well-authenticated instance where recovery has occurred. During the stage of its development and greatest activity deceptive remissions occur; but after six or seven years emaciation sets in, a marasmus is developed, and the patient is apt to die from intercurrent disease.

Treatment.—The best method of treatment yet proposed is the restorative; the object is to improve nutrition. Among the drugs that have been used, especially by Charcot and his followers, are chloride of gold, phosphate of zinc, nitrate of silver, chloride of barium, potassium iodide and bromide, arsenic, belladonna, calabar bean, and ergot. The galvanic current is the best means of administering electricity. Opinion is divided as to the benefit obtained from hot or cold baths and thermal springs, or inunctions and massage. Pain not infrequently becomes so severe as to demand hypodermic injections of morphine.[1]

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[1] Leyden reports a case of almost complete cure from galvanism and the baths of Rehme.—*Beit. z. acute u. chron. Myelitis. Zeitsch. für klin. Med.* Berlin, 1879, I., p. 126.

upper lumbar and lower dorsal regions, and extend upward and downward. It is possible in long standing cases for the medulla and the second, third, sixth and eighth cranial nerves to be involved, and for the entire cross section of the cord, at various points, to be shrunken, hard and gray.

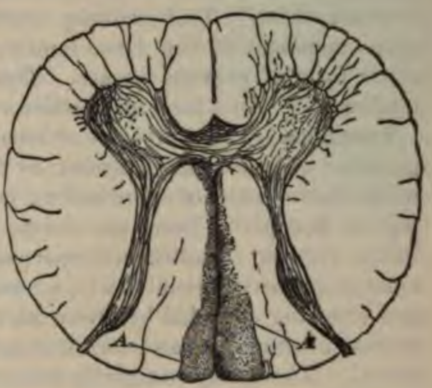

FIG. 214.

Locomotor Ataxia.

Section of Spinal Cord in the Cervical Region.

A.A. Sclerosis of the columns of Goll.

As a rule, the sclerosis ceases at the *restiform bodies.*[1] If Goll's columns are involved it is a secondary degeneration. The *posterior roots* of the spinal nerves show gray degeneration and atrophy to the naked eye.[2] On microscopical examination there will be found evidences of a large amount of dense and delicate connective-tissue containing nuclei, granular and amyloid corpuscles, in which are very few atrophied nerve fibres, which have lost their medullary sheaths.[3] Large branching cells are found throughout the sclerotic tissue. The posterior columns are seen fused together by connective-tissue in the pia mater, which dips down into the fissure. The walls of the capillaries and small vessels are thickened and rigid, and their calibre is diminished. Their sheaths are filled with oil globules. They are also markedly pigmented.[4] The sclerosis travels from the cord to the posterior roots of the spinal nerves, which show atrophy. The sciatic, crural, and brachial nerves have been found sclerosed and atrophied.[5]

Etiology.—Locomotor ataxia is more frequently met with in men than in women, the proportion being six to one. It occurs oftenest between the ages of twenty and fifty. In a neuropathic predisposition it may be induced by anything that seriously depresses the nervous system. Cold and wet, bad hygienic surroundings, excessive mental or bodily exertions, —onanism, and excesses in venery, especially,—depression of spirits, an insufficient or improper diet, the impoverished blood states that occur with or follow wasting acute or chronic maladies, prolonged lactation, syphilis,[6] and, according to some, excessive use of tobacco,—are among its predisposing causes. Blows on the spine, the suppression of menses or old hemorrhoidal fluxes may excite it. It is a question, when it follows pneumonia, rheumatic fever, or diphtheria, if there is any causal relationship between them.[1] Its hereditary tendency is shown by its attacking two or more members of the same family, other members suffering from some other form of nervous disease. This is an example of the "neuropathic tendency."[2] In a large proportion of cases there is no assignable cause.

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[1] Charcot describes *the external bands* :—two bands near the posterior cornua in the outer part of the posterior columns. They run parallel with the posterior horns of gray matter.

[2] Charcot has drawn attention to the condition of *the joints* in some cases of locomotor ataxia. "Sur quelques arthropathies," etc.—*Archiv. de Physiologie*, I., 1868, p. 161.

[3] Cornil and Ranvier insist that the axis-cylinder *always* exists, but that it requires a peculiar mode of preparation to demonstrate this.

[4] Lockhart Clark states that he has sometimes found the extremities of the posterior cornua, and even the central gray substance, more or less damaged by disintegration. The question has been raised whether the *initial lesion* may not be in these cornua.

[5] Friedreich.—*Virchow's Archiv.*, Bde 26, 27.

[6] Erb found a *syphilitic history* in twenty-seven out of forty-four cases.

Symptoms.—The symptoms of locomotor ataxia may be divided into three periods: a period characterized by disturbances of sensation, a period in which there is loss of coördinating power, and a period of paralysis. During the first period there are sharp, tearing, lightning pains in the lower limbs, dysuria, incontinence, spermatorrhœa, nocturnal pollutions, excitement of, or loss of sexual desire, a sense of weariness in the limbs, and nausea and vomiting attended by severe and paroxysmal aching in the stomach. A sense of numbness and formication in the limbs is not uncommon in this period. There may be a girdle sensation, not only about the waist, but also in the limbs—chiefly about the knee and ankle. In some cases there will be evidence of arthropathies and symptoms much resembling those of active cerebral hyperæmia. Rectal and urethral colics, bronchial spasms, and nephritic symptoms resembling those of renal colic, are not infrequent. The pains during this period are usually in the feet and legs; but they may have their seat in the back, stomach, intestines, or bladder. If they are situated in the internal viscera, the functions of those viscera are disturbed. Wherever they may be seated, at first they do not come on often, and are of short duration; but as the disease advances, the attacks become more frequent and are of longer duration. The muscles of the eyes may be affected, causing double vision or strabismus, which may last a few days or weeks and then disappear; or changes may occur in other nerves which lead to loss of sight. There may be temporary or permanent dilatation of one or both pupils.

The disturbances of surface sensation are manifold; sometimes the patient will complain of a sense of numbness in the hips, sometimes a pricking sensation, or a sensation of some soft substance between the feet and the ground; one portion of the surface may be anæsthetic, another hyperæsthetic. After a varying period, the *ataxic* symptoms appear, and the muscles are no longer moved in their natural way. The gait becomes unsteady, the patient walks like one intoxicated; there is a sense of heaviness in the limbs, and if the feet are brought close together, and the eyes closed, the body sways to and fro and sometimes falls. After a time the patient is compelled to watch his feet while walking. Later on he throws out his feet and legs in the most grotesque manner; for when the will acts the muscles contract far more than the patient intends. If the upper extremities are involved, he is unable to dress himself; he cannot pick up a pin, button his garments, or hold anything in his fingers. The movements of his hands and arms are forcible, irregular, and jerking. The gait during this period is peculiar; the heel is brought down first, then the toe: there is a double beat to the step. Quiet or steady movements are impossible. At times the loss of coördination is so great that for days the patient is unable to walk, and then the coördinating power is partially restored. One extremity may be involved after the other, or both be attacked at the same time. During this period there is a marked loss of sensation, especially in the feet and legs; these patients are often unable to tell when their feet touch the floor. Sensitiveness to pain is diminished, and it may be several *minutes* before the prick of a pin is felt. The sensory disturbances of the first period are increased, and the sight is more impaired. In this period there is loss of the reflex action of the muscles of the lower extremities, especially the muscles of the calf of the leg. The abolition of the patellar tendon-reflex is one of the diagnostic signs of the disease. It occurs very early in the disease, and it is often impossible to say when it first occurred. Cessation of reflex mobility of the pupil (Argyll Robertson) is also an early symptom. Loss of the sense of temperature, a greater or less loss in electro-muscular contractility, and, in the irritative forms, increase in galvanic excitability, are not uncommon. During this period there may be developed a peculiar affection of the joints; the joints most frequently affected are the knee, hip, shoulder, and elbow. The joint rapidly swells, and the synovial sac fills with fluid; after a time disorganization of the articular surfaces takes place and may be followed by destruction of the ends of the bones. In some instances the swelling suddenly disappears and the joint is not disabled.[1] Degenerative changes in the anterior horns are thought to be the cause of these joint symptoms. In a few cases skin eruptions of various kinds make their appearance.[2] In the *last* period paralysis occurs, and then are developed muscular atrophy, bed-sores, and those vesical and renal symptoms that are so apt to lead to death.

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[1] The most recent theory of its causation is that of Kahler and Pick, who regard those cases which follow acute infectious diseases as the result of the accumulation of fungi in the central nervous system producing nutritive disturbances.

[2] Carre records eighteen cases of tabes in the same family in three generations.—*M. Carre, Thèse* Paris, 1862.

Nearly all the symptoms of locomotor ataxia appear intermittently, and the progress of the disease is rarely continuous. During the third period there is always complete impotence. In some cases the face has a pale yellow color, which is most marked during cold weather.[3] During this last period sensation about the rectum is lost; hence the patient is apt to become exceedingly filthy unless great care is exercised. This condition is accompanied by almost constant dribbling of the urine. Intelligence, memory, and the higher cerebral functions are rarely, if at all, impaired. In a few cases of locomotor ataxia the patients become color-blind. Locomotor ataxia is a non-febrile disease, but during the initial period febrile symptoms may occur, and are then especially important as indicating a rapid progress in the disease.[4] The former is neither an early nor a late symptom. Pierret says that all possible nervous disturbances of hearing may precede ataxia. After reaching the second period, the disease may for a long time remain stationary, or it may temporarily improve, but complete recovery is rarely, if ever, reached. In the long and slowly progressive cases fluctuations always occur, with improvement in summer and exacerbations in winter.

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[1] Blum states that the great friability of the bones that results in spontaneous fracture is due to rarefying ostitis.—*Des. Arth. d'ori. nerv. Thèse.* Paris, 1875.

[2] Charcot says that they follow the track of nerves that have been the seat of pain.

[3] Eulenberg attributes dicrotism of the pulse in ataxics to loss of vascular tone of spinal origin.—*Berlin. Klin. Wochen.*

[4] Among the most recent contributions to this disease is Erb's paper, wherein he ascribes great importance as a symptom to *spinal myosis*, *i. e.*, reflex immobility of the pupil.

Differential Diagnosis.—Locomotor ataxia may be confounded with *paraplegia, multiple cerebro-spinal sclerosis, cerebellar lesions, chronic myelitis, hysterical ataxia*, and *chronic spinal meningitis*.

Paraplegia is a true paralysis; ataxia is not, and it is readily proved that in the latter disease muscular force is not diminished. In paraplegia the limbs are not thrown about in walking,—they are merely dragged. In paraplegia there is little or no resistance to artificial movement, while in ataxia there is great resistance in bending the limbs against the will of the patient. The nutrition of the muscles is markedly impaired in paraplegia, and normal in ataxia. Neuralgic pains are absent in paraplegia and present in ataxia. Strabismus, ptosis, etc., are present in ataxia and absent in paraplegia.

The differential diagnosis between multiple sclerosis and ataxia has been given.

Cerebellar disease has for its characteristic symptom vertigo; this is rare in ataxia. A patient with cerebellar disease can stand and walk better with his eyes shut than open, has unimpaired cutaneous sensibility, and the movements, while uncertain, are not so abrupt, vehement, and jerky as in ataxia; they resemble rather the stupid movements of a drunken man. The absence of neuralgic pains, of vesical and sexual weakness, and the prominence of headache, vomiting, and convulsions in cerebellar disease will be sufficient for the diagnosis.

In *chronic myelitis* there are no disorders of coördination. The patient suffers paresis, or even complete paralysis of the lower extremities, while in ataxia there is no paralysis, muscular power being undiminished. The limbs are *dragged* simply in chronic myelitis; they are thrown forcibly about in ataxia. Ocular symptoms are absent in chronic myelitis, present in ataxia. Contractures, spasms, paralysis of the bladder, cystitis, and the early formation of bed-sores, together with the absence of intense neuralgic pains, will also serve to distinguish chronic myelitis from ataxia.

In *hysterical ataxia* the history, and the occurrence of the disease in a female, with the subsequent course of the disease, will enable one to distinguish it from ataxia.

In *meningitis* there is pain,—increased on pressure,—slight paralysis but no incoördination, no flinging of the limbs in walking or moving, no abolition of tendon reflexes, and no ocular symptoms such as are present in ataxia.

Prognosis.—The usual course of locomotor ataxia is progressive.

The prognosis as to its duration is uncertain. The disease sometimes ceases of itself, leaving the patient in a disabled condition, but still giving him years of life. The slower the development the longer the duration. The prognosis is more unfavorable when it occurs with a history of nervous disease in the family, when the early symptoms are serious and constant, and when constitutional symptoms (especially emaciation) become marked. Complications likewise render the prognosis unfavorable. Complete recovery is possible but not probable. The duration of the disease varies from six months to as many years.

Treatment.—The efficacy of treatment depends upon the stage at which it is commenced. Undoubtedly, if the disease can be early recognized, its advance can in many cases be checked.

Of the drugs recommended, nitrate of silver is perhaps the one most extensively used. It should be given cautiously; about one grain *a day* in divided doses. The galvanic current is nearly always of service. Some cases will be benefited by the iodides, others by the bromides. Strychnia, phosphorus, arsenic, the chlorides of gold, sodium, and barium, the phosphide of zinc, belladonna and ergot all have been recommended.[1] The diet and mode of life should be such as to conduce to the highest degree of health and nutrition. Cod-liver oil and phosphorus may be given as adjuvants to a nutritious diet.

The patient should remain at rest as much as possible. Under no circumstances should he be allowed to expose himself to cold or wet or to sudden changes in temperature. Flannel should be constantly worn next the skin. Simple thermal baths seem to do harm, but *saline* thermal baths sometimes give good results. Sulphur, chalybeate and mud baths have been recommended. Erb recommends, as better than all, the cold water cure. He advocates the wet pack for the neuralgic pains. Bleeding or depletion of any sort is contraindicated, even in the initial stages. For the gastric derangement bismuth will generally be found efficacious. Constipation must be overcome by mild cathartics. For the vesical weakness or for incontinence, Faradization of the bladder, bromide of potash, camphor, and lupulin are advocated. In order to preserve the nerve force and prevent exhaustion, crutches are very useful, as they prevent the muscles from being constantly overtaxed.

SPASMODIC TABES DORSALIS.

Under this name Charcot has described what Erb calls *spastic spinal paralysis.* It has also been called tetanoid pseudo-paraplegia.

Morbid Anatomy.—As far as can be stated there is symmetrical sclerosis of the lateral columns, chiefly of their posterior portions. The induration shades off imperceptibly into the normal tissue of the columns.

This degeneration does not differ microscopically from that seen in sclerosis of the cord; it often extends in varying degrees the entire length of the cord. Anterior polio-myelitis and posterior sclerosis are frequently associated.

Etiology.—Spasmodic spinal paralysis is more common in males than in females, and is rare except between the ages of twenty and fifty; it rarely occurs in children. Traumatism and exposure to wet and cold are named as its causes.

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[1] Lockhart Clark recommends morphine, cannabis indica and belladonna *with* silver nitrate when the latter irritates the bowels or bladder.

Beyond this its etiology is obscure.

Symptoms.—Beginning very insidiously, the first symptoms noticed are weakness and paresis of the lower—rarely of the upper—extremities. These patients drag their limbs. This is followed by twitchings and stiffness of the muscles, and later there is so much muscular rigidity that locomotion is embarrassed or rendered impossible. Exaggeration of the tendon-reflexes is an early and important symptom, and is associated with marked ankle-clonus, in which the muscles of the calf or the whole limb are put in a state of tremor when the foot is flexed, or when the patient puts his toes to the ground. As the muscular rigidity increases these signs diminish. Later, general muscular tremors or shiverings unaccompanied by temperature changes may occur, in which all the muscles partake. They may be excited by cold or follow excitation of ankle-clonus when they do not occur spontaneously. If the patient is able to walk, he has the typical *spastic* gait; the adductors keep the thighs close together, the toes are dragged, and as the heel is brought down the extensors of the foot contract spasmodically and may throw the patient forward, lifting him on his toes. Sensibility and skin-reflexes remain normal.

Electric reaction of the *muscles* is unchanged; while that of the nerves is lowered to *both* currents. In the advanced stage of the disease the muscles of the abdomen, back, or upper limbs may become involved. In the latter case the fingers and hand are strongly flexed; the forearm is pronated and semiflexed, and the arm is fixed to the side. After a varying period paralysis of the affected parts occurs, and the contractures become more marked; the legs are permanently extended, and the foot assumes an equino-varus position. Pain rarely accompanies the contractures,[1] and the nutrition of the affected muscles is *not* impaired.[2]

Differential Diagnosis.—Spastic paralysis may be confounded with *tabes dorsalis, chronic anterior polio-myelitis, multiple sclerosis, peripheral paralysis,* and *transverse myelitis.*

In *locomotor ataxia* the ataxic symptoms, the double beat and stamp of the walk, the absence of tendon-reflexes, the general pains, the bladder symptoms, and the absence of paralysis and contractures are in direct contrast to the symptoms of primary lateral sclerosis.

In *chronic anterior polio-myelitis* atrophy follows the paresis and the muscles lose electric excitability. Tendon-reflexes are absent. In spastic paralysis rigidity follows paresis, and tendon-reflex and electric excitability are exaggerated.

Multiple sclerosis, when it is located in the lateral column at the onset, is practically spastic tabes. When the sclerotic process attacks other portions of the cord, or when cerebral disturbances occur, it assumes its distinctive characteristics.

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[1] Erb states that pain in the back and limbs attended by formication and other paræsthesiæ not uncommonly *precedes* the motor weakness at the beginning of the disease.—*Virchow's Archiv.*, b. 70. 1877.

[2] Recently, Stumpell calls attention to the relaxation of the muscles which occurs in spastic paralysis when the legs are not irritated by their own weight.

In *peripheral paralysis* there are disturbances of sensation and nutrition; the disease develops *unsymmetrically*, and reflex excitability and electro-muscular contractility are rapidly lost.

In *tranverse myelitis* trophic disturbances, vesical derangements, and alterations in sensation are early and marked symptoms. They do not occur in spastic paralysis.

Prognosis.—In uncomplicated spastic paralysis the prognosis is good. Some claim that complete recovery is possible, and in most instances the symptoms can be ameliorated. The disease may progress slowly for years, and then remain stationary indefinitely; or it may become complicated by bulbar or glosso-labio-laryngeal paralysis, and prove rapidly fatal.

Treatment.—In addition to the treatment proposed for chronic myelitis the galvanic current is most useful. Iodide of potash, arsenic and cod-liver oil in small doses, with careful attention to rest and diet, are to be recommended. Shampooing, rubbing and massage afford great comfort, and calabar bean may be given for the cramps. Nerve stretching has also been employed.

AMYOTROPHIC LATERAL SCLEROSIS.

(*Spastic Paralysis.*)

Charcot calls this disease the deuteropathic form of progressive muscular atrophy. Pathologically and clinically it is a complex of progressive muscular atrophy and spasmodic spinal paralysis.

Morbid Anatomy.—The sclerotic process begins in the cervical region, and although at first it is limited to the lateral columns, it soon attacks the anterior cornua and leads to destruction and atrophy of the large ganglion cells. It also extends downwards into the dorsal and lumbar lateral columns, and almost invariably upwards so as to involve the medulla, when the signs of bulbar paralysis are induced and followed by a fatal issue. This process has its seat in the same portions of the cord as the secondary descending degeneration of Türck, and new bands of dense connective-tissue join the degenerated lateral columns with those portions of the anterior horns that are involved.[1] In the floor of the fourth ventricle the cells of the nucleus of the spinal-accessory, facial, and hypoglossal nerves are degenerated. The anterior roots and peripheral nerves are atrophied. Trophic changes in the muscles are identical with those of progressive muscular atrophy.[2]

It is stated that interstitial growth of neuroglia is sometimes found without marked degeneration or atrophy of the nerve fibres, but that the whole system of fibres and ganglion cells which unite the motor centres in the cortex with the muscles is involved.[3]

Etiology.—Nothing more can be said regarding its etiology than has already been stated concerning the origin of the two diseases of which it is a compound.

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[1] *Archiv. de Phys. Nor. et Path.* 1879.

[2] Rosenthal states that their inflammatory character is more marked in amyotrophic lateral sclerosis, and that hyperplasia of the perimysium is more pronounced.

[3] Flichsig and Pick state that the whole system of nerve fibres and ganglion cells which unite the motor centres in the centres of the brain with the muscles are affected in amyotrophic lateral sclerosis.

Symptoms.—The disease begins with weakness, paresis, and then actual paralysis in the upper extremities, associated with muscular atrophy, which is usually diffuse and rapidly progressive. Fibrillary spasms and twitchings of the affected muscles are well marked; but electrical contractility is preserved. Sensibility is not impaired, but the muscles become rigid and contracted with the arms flexed.[1] In a few months the lower limbs are involved in the paralysis and rigidity, with exaggerated tendon-reflexes and contractures. Subsequently the muscles atrophy and show the reaction of degeneration and fibrillary spasm, while the contractures diminish. This is followed by the symptoms of bulbar paralysis.

Differential Diagnosis.—Amyotrophic lateral sclerosis may be mistaken for progressive muscular atrophy. But in the latter disease the slow course, absence of bulbar paralysis, and *partial* affection of certain groups of muscles, are in marked contrast to the symptoms of the former. Moreover, in amyotrophic lateral sclerosis the atrophy is preceded by paralysis.

Prognosis.—The prognosis is decidedly unfavorable; death results in from one to three years from bulbar paralysis. It is not, however, preceded by paralyzed sphincters, vesical troubles, bed-sores, or other trophic lesions.

Treatment.—Residence in the open air at a high altitude and strict attention to the general health are of first importance. Beyond this the treatment is identical with that of other forms of spinal sclerosis.

Mitchell recommends cod-liver oil, iron, strychnia, and dry cupping along the spine, with massage.

PSEUDO-HYPERTROPHIC PARALYSIS.

This is a progressive muscular paralysis occurring chiefly in boys.

Morbid Anatomy.—The German pathologists generally regard this disease as a chronic myositis with hyperplasia of interstitial connective-tissue. Gowers, however, in a recent monograph, describes the substance with which the muscle is filled and its fibres replaced as a new growth. Fat accumulates in the new growth to such an extent as to induce atrophy, and the muscles undergo granular and fatty metamorphosis.

As a final stage Charcot mentions waxy degeneration of the muscular elements. If a portion of the affected muscle is examined it will be found of a pale red or yellow color, according to the date of the disease.[2] No lesions of the spinal cord have been demonstrated in this disease, although it is always described with diseases of the cord.

Etiology.—Age and sex are the most constant predisposing causes, over eighty-eight per cent. of the recorded cases occurring before the tenth year. Hereditary influence appears most powerfully on the mother's side. The neuropathic tendency is more marked than in any other nervous disease. The recognized exciting causes are cold, falls, and convalescence from acute febrile disorders.

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[1] Erb states that as atrophy of the muscles progresses they may undergo simultaneous lipomatous hypertrophy.

[2] Charcot, Cohnheim and Eulenberg found no changes in the cord. Others have found spots of sclerosis and atrophy of the ganglion cells in the anterior horns. Lockhart Clark and Gowers have discovered extensive disintegration of the gray matter at the centre of each lateral half of the cord and of the anterior commissure.

Symptoms.—M. Duchenne makes three stages: *first,* a stage of weakness without increase in the size of the muscles; the muscles chiefly affected are those of the legs, especially the gastrocnemii, lower part of the back, and the erectores spinæ. *Second,* a stage in which hypertrophy appears and weakness extends to the upper extremities. During this stage for a year or more the child may evince no symptoms beyond a progressive weakness. He is easily and quickly tired, raises himself with increasing difficulty, and when erect does not stand firmly. He soon begins to show a peculiarity of gait and attitude. He walks with a swaying, unsteady step, and as he stands the shoulders are thrown backward and the spine is sharply bent in the lumbar region. The hyperplastic and degenerative changes in this stage produce the pseudo-hypertrophy of the muscles, which become firm and hard with increased loss of function. When the child is placed in an erect position the increase in size becomes very marked. In the supine position the soles of the feet are approximated and the joints of the lower extremities are flexed. Similar hypertrophy sometimes affects the muscles of the upper half of the body; but more commonly they are wasted, and thus the protuberant belly and the thick, firm calf and thigh afford a striking contrast to the emaciated muscles above the diaphragm. The children walk only with the greatest difficulty, or possibly they cannot stand without support, and the act of sitting or rising becomes difficult. Sometimes it is impossible for them to maintain even a sitting posture. The antero-posterior curvature of the spine and the displacement of the shoulders are much exaggerated, and the toes often undergo a claw-like deformity.

Gradually some muscles become soft and fatty while others remain firm and hard, and the child passes into a stage of complete paralysis of the trunk and upper extremities in which all the muscles that were hypertrophied atrophy, and the patient becomes completely helpless. Formication is not uncommon at first, but neither anæsthesia nor hyperæsthesia is present at any time. Electro-contractility of the muscles is unimpaired until the advanced stages of the disease. The upper extremities seldom suffer pseudo-hypertrophy, but may exhibit true progressive muscular atrophy. The deltoid and triceps are usually the only muscles enlarged. In nearly all cases the disease progresses symmetrically. The skin of the affected parts is sometimes bluish, dry, and thinned. The tongue and the muscles of the face may become enlarged, and some consider the cardiac hypertrophy that is often present as of similar origin.

In a number of cases the mental faculties have been impaired, but the general health is usually good, and the sphincters are never involved.

Prognosis.—The prognosis is unfavorable, and when progressive muscular atrophy is superadded it is especially so. Cases of recovery have been reported. Its duration varies from a few months to several years. Intercurrent disease is generally the direct cause of death.

Treatment.—Duchenne's treatment is regarded as the most efficient. Local electricity, shampooing, and massage, if employed before the hypertrophic changes occur, may arrest its development.

ACUTE ASCENDING PARALYSIS.

This peculiar disease has no well-recognized anatomical lesions, but is regarded by most observers as a purely functional disease.[1] Many authorities have recently regarded it as an acute infectious disease, in which the infectious element causes functional rather than organic changes, and may, in different cases, affect different parts of the nervous system. Some cases closely resemble forms of peripheral neuritis.

Etiology.—Acute ascending paralysis is a disease of adult life, more common in men than in women. Exposure to cold, emotional influences, venereal excesses, syphilis, acute febrile disease, and poisoning from corrosive sublimate[2] have each been followed by it.

Symptoms.—For the first few days there is possibly a slight fever, accompanied by a sense of weariness and numbness and darting pains in the limbs, chiefly in the feet and in the tips of the fingers. This is followed by paresis, then actual paralysis of the distal portions of the lower extremities. The paralysis gradually extends upward, until in a few days paralysis of the lower extremities is complete. Soon the trunk muscles are implicated; the patient can neither turn nor sit up in bed. The upper extremities are then involved, the paralysis extending from the finger tips to the shoulder joint. Sometimes there is a distinct interval between the paralysis of the trunk and the upper extremities. In about seventy per cent. of cases the muscles of the neck and the diaphragm are involved, and finally bulbar paralysis is superadded.

In some cases the disease pursues a reverse course, palsy of the extremities following the symptoms of bulbar paralysis. The paralyzed limbs are lax and the muscles flaccid; but they do not undergo atrophy, and the electrical reactions of nerves and muscles continue perfectly normal. Sensibility is little if at all affected, the sphincters are not involved, cutaneous nutrition is unimpaired, and there are few, if any, vaso-motor or trophic disturbances. Reflex action diminishes after the first two or three days. There is usually constipation and difficult defecation, on account of the paralysis of the abdominal muscles. The intellect is never disturbed. In over thirty per cent. of the cases, when, or before, the arms are implicated, the disease is arrested, and soon recovery of power begins to manifest itself, the parts first paralyzed being last restored. It is said that recovery has taken place even after the paralysis has reached the nerves of the bulb. As a rule there are no pains complained of in the paralyzed parts.

Differental Diagnosis.—Acute ascending paralysis may be confounded with *acute myelitis, acute spinal paralysis of adults*, and, when slowly evolved, with *chronic spinal paralysis*.

Acute ascending paralysis is differentiated from acute ascending *myelitis* by the slight disturbances of sensation which attend it, by the preservation of electrical excitability, and by the absence of motor irritation and trophic disturbances.

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[1] Dejerine claims to have found in two cases an alteration in certain fibres of the anterior roots (parenchymatous neuritis). The myeline was broken up into fragments. Multiplication of the nuclei in the white substance of Schwann, and disappearance of the axis-cylinders were noted. The majority of the fibres were unaltered. The same lesions were found in the intramuscular nerves of the affected members.

[2] Ketly in the *Pester Med. Chir. Pres.* Nos. 8-9. 1878.

Acute ascending paralysis differs from *acute spinal paralysis* of adults in the absence of atrophy of the paralyzed muscles; the electrical reactions remain normal, and it is more rapidly progressive. The medulla is not involved in acute spinal paralysis, whereas about seventy per cent. of cases of ascending paralysis end in bulbar symptoms.

Rapid atrophy of the muscles and the reaction of degeneration are prominent symptoms of *chronic spinal paralysis*, which are absent in acute ascending paralysis. In the latter disease there is a far greater tendency to extend to the medulla.

Prognosis.—Acute ascending paralysis is generally fatal. It may last several weeks; but its average duration is from ten to fifteen days. The more rapid its progress, and the earlier the medulla is involved, the more unfavorable the prognosis. Improvement may take place even in the most acute cases. Death occurs from the same causes as in bulbar paralysis.

Treatment.—All that can be done in this disease is to maintain the nutrition. Electrical currents may be applied to the affected muscles. Sulphur baths, iron, arsenic, strychnine, and iodide of potash are recommended for the more slowly progressive forms, but clinical experience does not sustain the claim of beneficial results which have been obtained by their use.

SPINAL APOPLEXY.

Spinal apoplexy is not of frequent occurrence, except when due to traumatism or to some pre-existing disease of the cord.

Morbid Anatomy.—A meningeal hemorrhage may extend the entire length of the cord; primary hemorrhage, however, usually occurs into the gray matter, and if slight, may only involve one side. The white matter is never alone involved, but about fifty per cent. of spinal hemorrhages are circumscribed. In a few cases punctate capillary hemorrhages are found studding the gray substance. A clot of varying size, one-fourth to one inch in diameter, is found in the central portion of the cord containing débris of nerve-tissue, corpora amylacea, fat granules, and pigment. This blood sac commonly lies with its long axis parallel with the cord. The centre may have undergone softening, and the wall is formed of ragged nerve-tissue. About a clot in the white substance, the tissue is

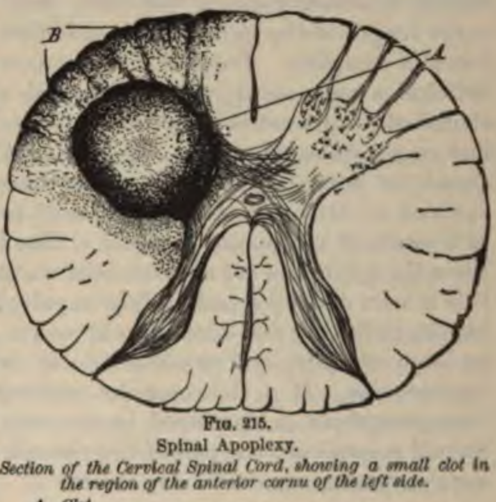

FIG. 215.
Spinal Apoplexy.
Section of the Cervical Spinal Cord, showing a small clot in the region of the anterior cornu of the left side.
A. *Clot.*
B. *Meninges adjacent to the clot showing hyperæmia.*

always more or less deeply tinged with blood. When the extravasation involves the periphery of the cord there will be hyperæmia of the adjacent membranes. Capillary aneurisms have usually been found in the spinal vessels at the seat of the apoplexy; and Liouville has found ampullary dilatation of the large vessels, thickening of their walls, and proliferation of their nuclei. Charcot states that there is swelling of the nerve cells and axis cylinders. The clot may undergo retrogressive changes and result in softening, abscess, or a cicatrix. Erb describes softening of the gray substance as a not infrequent sequela of spinal apoplexy. White softening accompanied by gelatinous œdema surrounds the blood tumor and merges imperceptibly into healthy cord substance. Ch. Bastian states that inflammatory softening may start from the clot and extend up or down the cord.[1]

Etiology.—Spinal hemorrhage is most commonly the result of traumatism, and especially of severe concussion.[2] It may result from rupture of vessels in or near neoplasia, in foci of myelitic or other softening, or in any chronic spinal disease. Small hemorrhages may occur with scorbutus, purpura, and in the hemorrhagic diathesis. Atheromatous, fibroid, and fatty degeneration of the blood-vessels, nuclear proliferation, and minute aneurismal dilatations predispose to spinal hemorrhage. Age and sex are also predisposing factors, as it is most frequently met with in men between the ages of fifteen and forty. Anything that induces or predisposes to active hyperæmia acts as a predisposing cause.[3]

Symptoms.—Sometimes the symptoms of myelitis, spinal irritation, or active spinal hyperæmia precede the extravasation, but usually it comes on suddenly and causes complete paralysis of both motion and sensation below the site of the hemorrhage, without loss of consciousness. It is attended by severe pain in the back, that may be localized or extend the entire length of the spine. This sometimes disappears when the paralysis becomes complete. Pressure does *not* increase it. At the onset spasmodic twitchings may occur in the paralyzed parts; and all reflex motion is abolished—the muscles being completely relaxed. Priapism and dyspnœa may occur when the clot is high up. When hemorrhage occurs in the dorsal or lower cervical region, the temperature of the paralyzed limb —which at first is sub-normal—rises 2° to 3° F. higher than the axillary as a result of vaso-motor paralysis. The bladder is at first paralyzed, but when the sphincters are also involved the urine passes involuntarily. Cystitis is soon developed and pyelitis rapidly follows. The fæces are passed involuntarily and bed-sores appear early. The paralyzed parts begin to undergo atrophy, and while so doing exhibit *the electrical reaction of degeneration.* If a clot occupies *one-half* the gray matter at any point, *hemi-paraplegia* is developed in the limb of the same side as the lesion. Should it implicate the root of the phrenic nerve, intense dyspnœa and perhaps instantaneous death may result.

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[1] Charcot and Hayem regard this lesion as always consecutive to myelitis.

[2] Sir William Gull relates a case where small extravasations were found on the anterior and posterior cornua as well as in the posterior columns of the cord. The case was the result of a fall.

[3] Erb claims that variola hemorrhagica, typhoid, yellow, and malarial fevers are causes of spinal hemorrhage.

Differential Diagnosis.—Apoplexy of the cord may be confounded with *meningeal apoplexy* and *thrombotic softening*. In *meningeal apoplexy* sensory paralysis is absent or but slightly marked; after the initial motor paralysis, improvement is marked and speedy; pain, hyperæsthesia, and *irritation symptoms* are prominent; and bed-sores, cystitis, and pyelitis do not occur. All these are in distinct contrast with the symptoms of spinal or intramedullary apoplexy, Vaso-motor disturbances are absent in *meningeal*, but present in spinal hemorrhages.

Thrombotic softening produces *incomplete* paraplegia, without loss of sensation. The absence of sensory and motor excitement is regarded as diagnostic. *Hemorrhage* not infrequently occurs in a spot of *myelitic softening*, but in such cases the paraplegia *follows* irritation, pain on pressure, fever, vesical symptoms, and the girdle sensation. In apoplexy paraplegia is the *first* symptom, the other symptoms coming on at greater or less intervals.

Prognosis.—Charcot states that a true spinal apoplexy is always fatal. The prognosis is certainly exceedingly unfavorable when the onset is severe or when the thoracic and respiratory muscles are implicated. Death may occur in six hours. Incomplete recovery is possible, with paralysis and atrophy of the muscles of the lower extremities. Septic fever, cystitis, and pyelitis are its complications; and death may occur from exhaustion and marasmus. The chief danger, if life is prolonged a few weeks, is from myelitis and extensive softening.

Treatment.—Absolute rest in the *prone* (not supine) position, is most important. Blood-letting, purgatives, or revulsives are not allowable. Ice-bags should be applied along the spine. Bromides and opiates may be employed to insure rest. Attention to the bladder is an important element of treatment. The treatment is the same as for cerebral apoplexy with transference of local measures from the head to the spine.

TUMORS OF THE SPINAL CORD.

As tumors in the spinal canal arise from the same causes and present the same anatomical appearances as similar growths in the brain, it is only necessary to consider their clinical phenomena.

Symptoms.—These will vary greatly in their nature and in the order of their development, with the seat of the tumor, its extension, and the amount of intercurrent changes in the adjacent tissues.

I. Tumors which primarily involve the substance of the cord are more common in the gray matter, and are attended by a gradual abolition of function. The changes are due more to pressure than inflammation, so that pain is a less common and prominent accompaniment. The earlier symptoms are those of paresis, either with hemiplegia or paraplegia of the parts below the tumor, according as the growth involves a lateral half or the entire substance of the cord. When the lesion is lateral, the paralysis may be crossed or mixed; motor paralysis of one side may be attended by anæsthesia of the other. The paralysis is rarely complete at first, but is progressive, though liable to remarkable remissions, and eventually becomes complete. As these tumors extend and involve the meninges and roots of the nerves and are attended by inflammation, peripheral pains and muscular spasms may develop, while the œdematous softening or ascending and descending degenerative changes may cause atrophy and wasting of the muscles. Tumors of the substance of the cord may thus resemble muscular atrophy, tabes dorsalis, or other forms of sclerosis.

II. Meningeal growths and tumors developed exterior to the membranes pursue a less latent course. They involve the roots of the nerves early, and are productive of more marked inflammatory changes. Hence the early symptoms are those of both sensory and motor irritation. There are burning, lancinating, and crushing pains, which are irregular and liable to severe exacerbations, which may be attended by hyperæsthesia and cutaneous eruptions. There are muscular twitchings and spasms which, as the nerves are more seriously affected or the cord becomes compressed, pass on to paresis or complete paralysis with muscular atrophy and wasting. The sensory symptoms at the same time give place to numbness and anæsthesia.

Spinal tumors of all forms produce more or less myelitis, and with this the reflex excitability is greatly increased and may cause contraction and rigidity.[1] Trophic changes are late symptoms. In all forms of spinal growths the symptoms are liable to sudden and marked changes either favorable or unfavorable. A sudden œdema or hemorrhage may cause extensive paralysis, which may be permanent or slowly recovered from. Or a rapid and marked improvement may be speedily followed by a more complete and widespread paralysis. When the new growth involves the vertebræ and results in destructive changes, as frequently occurs with cancer, the early symptoms of pain and hyperæsthesia are usually severe and the paralysis rapidly becomes complete.

Differential Diagnosis.—*Inflammation, hemorrhage,* and *softening* of the cord are excluded by their abrupt onset, and the fact that their symptoms are more general and uniform in their development than those of a tumor.

The nature of a tumor can seldom be determined, and the diagnosis rests almost entirely upon the history and the presence of some adventitious growth in other organs.

Prognosis.—Complete or even partial recovery is not to be expected. The paralysis and muscular atrophy are progressive. No estimate of their duration can be made.

Treatment.—Every tumor should be treated as syphilitic, as this is the only kind amenable to treatment. When the vertebræ are involved, supporting appliances are indicated. Beyond this the treatment is purely symptomatic.

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[1] Schüppel claims that the spine is always curved towards the side upon which the tumor is situated, and that the combination of irritative and paralytic symptoms of striking inconstancy is diagnostic of tumor.

SPINA-BIFIDA AND HYDRORACHIS.

Spina-bifida is a congenital malformation due to arrested development of some portion of the spinal column. It is usually associated with dropsy of the spinal cord, or hydrorachis. Internal hydrorachis is a collection of serum in the central canal, causing atrophy or destruction of the spinal medulla. External hydrorachis is an effusion into the subarachnoid space. If the spinal canal closes, it is called *H. incolumis;* if not, *H. dehiscens.*

Morbid Anatomy.—Usually two or three spinous processes and laminæ are deficient, the rudimentary portions of the vertebral arches are spread out and irregularly expanded, and the membranes protrude through the aperture as a hernial sac. The tumor, which is oval or spherical and at birth about one inch in diameter, occurs almost invariably in the lumbar or lumbo-sacral region. It is tense and fluctuant, being filled with cerebrospinal fluid. Pressure on the tumor increases the tension, and may produce symptoms of cerebral pressure. The skin over the tumor, although sometimes normal, is usually thin and transparent. The nerve-trunks forming the cauda equina often traverse the interior of the sac. The skin at the apex of the tumor is sometimes destroyed and the sac exposed. The point is excoriated or covered with pus and granulations, and the ulceration may go on to perforation. The dura mater always forms one layer of the sac.

Etiology.—Nothing is known concerning the etiology of the hydrorachis which accompanies spina-bifida.

Symptoms.—The symptoms of hydrorachis are obscure. It may cause pressure on various parts of the cord and cause paresis or absolute paralysis and wasting of the muscles. When associated with spina-bifida, the tumor is the diagnostic symptom. If the effusion is in the central canal and the cord is thus implicated, the lower limbs are usually paralyzed as well as the bladder and the lower bowel. Convulsions, spinal inflammation, or rupture of the sac with escape of its contents, usually precede death, which usually occurs within a few weeks after birth. When the fluid oozes away gradually, relief follows; spontaneous and complete cure may then occur, the tumor contracting to a small nodule, and the aperture in the canal closing more or less completely. When the dropsy is external to the cord and the skin thick, the tumor may increase in size without causing serious disturbance, and may even reach the size of a child's head, and life be prolonged twenty or thirty years.

Other forms of arrested development often accompany spina-bifida.

Prognosis.—It is very rare for patients with spina-bifida to reach puberty. The majority of cases terminate fatally a few days or weeks after birth. The prognosis is favorable when the base of the tumor is narrow, the skin over it thick and normal, and when it is situated in the sacral region.

Treatment.—The process of spontaneous cure has been successfully imitated by small tappings, frequently repeated, and followed by light compression. Only a small portion of the fluid should be withdrawn at any operation, and the puncture should always be made at the side of the tumor so as to avoid injury to nerve-trunks. Cases have been successfully treated by the injection of small quantities of iodine.

Other operative measures, such as compression of the neck of the tumor by means of a clamp or ligature, and excision, have occasionally been employed with success; but no such attempt should be made except when the tumor has a very narrow base, and is situated over the sacrum.

DISEASES OF THE PERIPHERAL NERVES.

Aside from those conditions which distinctly affect nerve-trunks, some others are so prominently manifested through motor and sensory disturbances, as properly to come under this head.

I shall here consider—

I. *Peripheral Neuritis.*
II. *Localized Spasms and Paralyses.*
III. *Chronic Lead Poisoning.*
IV. *Chronic Mercurialism.*
V. *Paralysis Agitans.*
VI. *Facial Paralysis.*
VII. *Eclampsia and Infantile Convulsions.*
VIII. *Tetanus.*
IX. *Neuralgia.*
X. *Megrim.*

PERIPHERAL NEURITIS.

Within a comparatively recent period quite a number of conditions formerly supposed to depend upon disease of the nervous centres have been found to be associated with inflammatory and degenerative changes in the nerve-trunks. They are grouped under the general term peripheral neuritis.

Morbid Anatomy.—In the acute forms of the disease distinct inflammatory processes can usually be recognized. The hyperæmia is sufficient to give the nerve-trunk a distinct pink color, and even extravasations may be present. The exudation produces marked swelling, and when the round cell infiltration, which is always present in acute cases, becomes more prominent, the nerve-sheaths and interstitial tissue show distinct purulent infiltration. Changes in the nerve-fibres vary with the extent and severity of the inflammation. Always more or less compressed, the parenchymatous changes may vary from slight softening and cloudy swelling to fatty degeneration or complete disintegration of the medullary sheaths and axis-cylinders. When vascular and exudative changes have not been followed by destruction of the nerves, the process may terminate in simple resolution. Often, however, even in these cases, and always when there has been loss of tissue, repair takes place by formation of new connective-tissue, which may develop quite evenly or in irregular patches (*neuritis nodosa*), and cause, by its subsequent contraction, varying degrees of interference with nervous function. When nerve-fibres have been actually destroyed, partial restoration may take place, but it is complete only when there has been little loss of tissue.

Chronic neuritis is less frequent. In it the vascular and exudative changes are subsidiary and even unappreciable. A development of new connective-tissue may be the only evidence of pathological changes, but, more frequently, the inflammation is so purely parenchymatous as rather to deserve the name of degeneration. The changes are those of fatty metamorphosis and atrophy rather than simple cloudy swelling. Permanent injury of the nerve-trunks, rather than regeneration, is the termination of these changes.

Etiology.—Direct traumatism, pressure from dislocated bones, fractures, or tumors, and extension of inflammation from adjacent tissues, may each induce a local neuritis. It is only when such conditions are complicated by sepsis that this neuritis becomes wandering or metastatic (*neuritis ascendens, aut aberrans, aut migrans*).

Most cases of primary neuritis appear without apparent cause. Many forms of neuralgia and paralysis occurring as complications of rheumatism, gout, lead, arsenic, or mercurial poisoning, and allied conditions, are believed to depend often upon neuritis, and these poisons are thus included among its causes. Alcohol must be recognized as an unquestioned cause, especially of chronic neuritis. Aside from this, little is known. Exposure to cold, sexual excess, and other "scapegoats" of that class, are made responsible for many cases when no evident cause offers.

The determination of a specific cause of tetanus has led to the belief that many cases of peripheral neuritis have a similar specific nature.

Symptoms.—Acute neuritis may be either *primary* or *secondary*.

Primary neuritis is usually multiple and degenerative in character, its etiology coming under the head of constitutional causes. It rarely affects the cranial nerves. Its invasion is sudden and attended by the usual symptoms of fever, with a temperature of 101°–105° F. A marked increase in the pulse-rate out of proportion to the febrile movement, is a peculiar symptom sometimes observed. It probably depends upon some implication of the vagus.

In the early stages of irritation the sensory nerves suffer most, so that hyperæsthesia and rapidly developing sharp, lancinating, tearing pains, which follow rather closely the nerve distribution, are the prominent early symptoms, while muscular spasms are often, indeed usually, absent, and seldom, if ever, are more than fibrillary twitchings. As the disease progresses, however, to the stage of abolition of nervous irritability, the motor nerves are the ones to suffer. The sensory disturbances decrease, but do not disappear, and gradually or rapidly developing paralysis, with loss of all the reflexes, give evidence of the implication of the motor trunks. As in other acute disease, the course and extent of the disease are very variable. In one case paresis or paralysis will be present almost from the first; in another it will be delayed some days, or even weeks. The disease is rarely arrested before the development of distinct paralysis, while some cases run an exceedingly acute course, and end in death from paralysis of the respiratory muscles. Others start in acutely, and then become chronic. In all severe cases electrical irritability of the affected muscles is largely or completely lost, and, many times, the reaction of degeneration and atrophy of the muscles will be developed. Occasionally multiple neuritis is chronic from the start. Recovery is always slow, and proportioned to the extent of parenchymatous change in the nerve-fibres. Paralysis in multiple neuritis usually begins in the lower extremities, and does not, as a rule, affect the bladder and rectum. The symptoms of traumatic neuritis are largely local, and include similar sensory and motor disturbances. The constitutional manifestations are usually those of a simple inflammation.

Differential Diagnosis.—Multiple neuritis is most liable to be mistaken for poliomyelitis. The diagnosis rests upon the acute invasion, the evidences of sensory disturbances rapidly changing to those of decrease and loss of motor function, with muscular atrophy, loss of reflexes, and the reaction of degeneration.

Prognosis.—This is exceedingly uncertain and rather unfavorable. Many severe cases, however, go on to a partial recovery, and, when the disease is arrested early, this may be complete.

Treatment.—In the early stage, during the intensity of the sensory manifestations, morphia must be employed for their relief. Hot packs and other local sedatives are often of assistance. During the stage of advancing paralysis, the treatment is purely tonic and symptomatic. When regeneration of the affected nerves begins, galvanism is of value in connection with other tonics, both to hasten the reparative processes and to improve the muscular condition. Salol and the salicylates have seemed of service in some cases.

LOCALIZED SPASMS AND PARALYSES.

(Scrivener's Palsy, etc.)

Writer's cramp is one of the more common varieties of anomalous muscular movements, or of those diseases which Duchenne calls "functional impotences." Of essentially the same nature are piano-players' cramp and the inability of tailors, dairymen, bricklayers, or telegraphers to execute movements to which they have long been accustomed.

Morbid Anatomy.—It has been thought that degenerative changes occur in the spinal cord, but according to Dr. Reynolds these diseases are due to a perverted nutrition of these parts.

Duchenne believes that the primary change is in the nerve centres, and gives as a strong argument in favor of this view the fact that the malady very quickly affects the left hand when this is used to supply the right in one who has writer's cramp. Dr. Poore, however, suggests that the lesion in typical cases is at the periphery, either in the muscles themselves, or in their terminal motor nerves. He attributes the disease to over-use and over-fatigue, not to central changes; and states that muscles which are *trained to work in order together* no longer do so when even *one* of them fails.

Etiology.—Writer's cramp, like other similar conditions, as from violin or piano-playing, telegraphing, milking, etc., is for the most part induced by long-continued use of the affected muscles.

Symptoms.—It will be sufficient to describe writer's cramp as a type of all this class of spasms, since they differ only in the muscles involved. It generally commences with a sense of weight or stiffness in the affected muscles, together with discomfort and indefinite pain, which is perhaps relieved by strong contractions or stretching the muscles. This uneasiness slowly increases, and there is added a tendency to spasmodic movements which renders the handwriting irregular and covered with unnecessary lines.

The pain and spasm at first follow only prolonged use of the pen, but soon are induced very readily, until at length all attempts at writing are abandoned. In the earlier stages the patient is able to control the spasms somewhat and to relieve them by holding his pen in an unusual way or by some other device. For a time also after more delicate manipulations are impossible, he can still perform coarser operations, but the disease almost invariably extends so long as the muscles are kept in use, and may result in severe spasm whenever any attempt is made to use the hand in writing. Occasionally it assumes a paralytic form and the patient is unable to hold a pen at all, or pain may be a prominent symptom, radiating up the arm as a severe neuralgia.

The disease may affect any or all the muscles of the hand or arm, and it rarely extends to the shoulder and trunk. More commonly the extensors and flexors of the thumb or index finger are affected, but though there is generally a distinct loss of power the muscles act perfectly in any motions save those which induced the disease.

In the other forms of local spasm the general history will be the same, and the deformities and disturbances will depend entirely upon the action of the muscles implicated. It is more likely to occur in those who merely copy than those who write an equal amount, but think at the same time—as authors and journalists.

Differential Diagnosis.—The history of the case is all-important, and will generally be sufficient for a diagnosis.

Lead palsy, with which scrivener's palsy may be confounded, is generally preceded by several attacks of lead colic; there is the peculiar blue line at the margin of the teeth, the skin assumes an earthy hue, and the "drop-wrist" exists, conditions which do not occur in writer's cramp.

Paralysis agitans, disseminated sclerosis, and the trembling due to *old age* or to *chronic alcoholismus* will be readily differentiated by the history.

Prognosis.—The prognosis is favorable. The shorter the duration of the condition, and the greater the opportunity the patient has to give the part rest, the better the outlook. Writer's cramp is much more easily relieved in the weak and nervous than in the strong and robust.

Treatment.—Absolute rest is essential, and will sometimes alone be sufficient for a cure. The mild galvanic current, blistering along the nerve-trunks, should they be tender, and rhythmical exercise of the affected muscles short of fatigue, is often of marked service. Morphine hypodermically may relieve, but does not effect a cure. The mind and body must have rest as well as the muscles. Hypodermic injections of atropia, strychnia, and Fowler's solution have been used with success. Massage to the part is recommended by Beard. In my experience the only course which has been followed by markedly beneficial results has been absolute rest of the affected muscles, with sea bathing and the internal use of iron.

CHRONIC LEAD POISONING.

(*Lead Palsy.*)

This is a morbid condition produced by the introduction of the salts of lead into the system, either through the mucous surfaces or the skin.

Morbid Anatomy.—After the salts of lead have been received into the system, they become deposited in various tissues or are discharged by the emunctories. They have been found in all the tissues of the body. They are eliminated mainly by the kidneys. In the paralysis caused by lead poisoning the muscles and nerves are early affected; later the nerve-centres become implicated. It is probable that the lead deposited in the affected tissues impairs their function and leads to their degeneration when the paralysis has existed for a long time.

Etiology.—The sources of lead poisoning are numerous: painters and workers in lead are those most frequently affected. Drinking-water, wines, and ales frequently become impregnated with it, and then become a source of infection. The application of lead powder as a cosmetic to the face and neck has caused lead-poisoning. Some persons are much more susceptible to its poisonous influences than others; I have known a few doses of lead taken as a medicine to give rise to pronounced symptoms of lead poisoning.

Symptoms.—The general health of those who are the subjects of chronic lead poisoning is always more or less impaired. Their skin becomes sallow, dry and harsh, they suffer from dyspeptic symptoms, loss of appetite, and constipation. A blue line forms along the edge of the gums immediately adjoining the teeth, which is regarded by some as diagnostic of lead poisoning—it is often present in those working in lead who are free from other symptoms.

The most important and characteristic symptoms are *intestinal colic*, and affections of the nervous system. Lead colic has been considered in the list of Intestinal Diseases. The most frequent of the nervous affections is drop-wrist from paralysis of the extensors of the fore-arm. It generally comes on gradually after one or two attacks of colic. Sometimes its advent is sudden. In painters the right hand is first affected, but after a time both hands are involved. The signs of lead palsy are loss of power over the extensor muscles of the fore-arm; first the patient is unable to abduct the thumb, then to extend the finger, then to extend the hand on the fore-arm, and the hand drops when the arm is held in a prone position. The paralysis is generally limited to the muscles supplied by the radial nerve.

The paralyzed muscles waste rapidly and lose to a greater or less degree their electro-contractility; there is no loss of sensation in the paralyzed limb, and not infrequently it is the seat of severe pains and tenderness. In some instances other muscles besides those of the fore-arm are affected, as the deltoid and triceps, and the palsy may involve the muscles of the lower extremity, especially the extensors of the foot and leg. In rare instances all the voluntary muscles are involved. Gouty subjects are peculiarly susceptible to lead poisoning, and in such cases the cirrhotic kidney almost always exists, giving rise to albuminuria and the other phenomena of the cirrhotic form of Bright's disease.

This condition is often accompanied by amaurosis and other grave nervous symptoms.

Differential Diagnosis.—The diagnosis of lead colic has been considered under the head of Intestinal Colic. Lead palsy may be distinguished from other forms of palsy, by the history of the case, by the absence of cerebrospinal disturbance, and by the blue line on the gums. When the muscles of a paralyzed limb respond to the influence of the electric current lead poisoning may be excluded.

Prognosis.—Chronic lead poisoning is rarely a direct cause of death, although it may exist for years, the longer its duration the less prospect there is of complete recovery. Extreme wasting of the paralyzed muscles with loss of electric contractility renders the prognosis unfavorable. In some instances the muscular power may be regained when the excitability does not return.

In most cases the general health is not seriously impaired, and the recovery from the paralysis, if not complete, is partial. The fatal cases are those which have been a long time exposed to the poisonous influence of lead, and who have been intemperate.

Treatment.—The first thing to be accomplished is to remove the patient from all sources of lead poisoning. Extreme personal cleanliness is important for those who cannot avoid such exposure. The habitual use of lemonade made with sulphuric acid is regarded, to some extent, as protective; it acts by converting the carbonate and other salts of lead in the stomach into the insoluble sulphate. Various methods have been proposed for removing the lead from the system, the most effective of which is baths containing some soluble sulphide.[1]

Iodide of potassium is recommended on the ground that the iodide makes, with the insoluble salts of lead deposited in the tissue, a new soluble salt, which can be eliminated by the kidneys. Its administration should begin with fifteen grains a day, and be gradually increased to thirty grains a day; it may, in anæmic subjects, be combined with chloride of iron. The bowels should always be kept freely open.

The only effectual remedy for restoring the paralyzed muscle is electricity in the form of Faradization. Its application should not be continued more than ten or fifteen minutes three times a day for two or three months. Severe shocks should be carefully avoided, although a current of high tension causes no movement in the paralyzed muscles. It is important that each paralyzed muscle should be treated separately.

CHRONIC MERCURIALISM.

(*Mercurial Tremor.*)

Chronic mercurial poisoning may result from the long-continued introduction of mercury into the system, either through the stomach, respiratory organs, or skin.

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[1] Dr. Pereira recommends baths medicated by dissolving sulphide of potassium in the proportion of two ounces in fifteen gallons of water.

Morbid Anatomy.—No characteristic lesions have been discovered in those who have died of chronic mercurialism, except the deposit of mercury in the tissues, especially the brain, liver and kidneys.

Etiology.—Workers in mercury, as gilders, looking-glass manufacturers, and those engaged in quicksilver mining, are those who chiefly suffer from chronic mercurialism, although it may result from its long-continued medicinal use. Those who are exposed to its fumes are especially liable to its poisonous effects.

Symptoms.—The manifestations of chronic mercurialization are mainly confined to the nervous and muscular system, and may be designated as *mercurial tremors.* Its first indication is a tremulousness of the hands and arms, accompanied by numbness and tingling, with pain in the joints. These symptoms may continue for years without interfering materially with the general health of the individual; but sooner or later the entire muscular system becomes invaded, and speech, deglutition, and respiration are more or less interfered with. Choreic movements occur, the patient is unable to walk or stand without assistance, and the face is contorted by muscular spasms; while the patient is in the recumbent posture and makes no muscular efforts, the muscular spasms cease, but as soon as he attempts to stand or move, the choreic movements commence.

In an advanced stage of the disease the convulsive movements do not entirely cease when the patient is in the recumbent posture. After the tremors have continued for a long time and have been severe, the patient loses appetite, becomes sallow and emaciated, and cerebral symptoms develop, the most constant of which are headache, vertigo, delirium, and epileptic convulsions.

Differential Diagnosis.—Mercurial tremor may be confounded with *multiple sclerosis*, *paralysis agitans*, and *chorea.* But a history of exposure to mercurial poisoning, and the fact that the nervous symptoms were preceded by ptyalism, ulcerated gums, mercurial fetor of the breath, nausea, colicky pains and diarrhœa, are generally sufficient to establish a diagnosis. It is also to be remembered that in paralysis agitans the muscles of the head and neck are not involved in the convulsive movements, and that the position of the patient does not influence the spasms.

Prognosis.—Mercurial tremor does not often directly cause death, but if exposures to the causes of mercurial poisoning are continued, death may result from exhaustion, intestinal or cerebral complications, or from intercurrent disease.

Treatment.—As soon as any of the symptoms of mercurial poisoning are present, the individuals must immediately be removed from all chance of exposure to the poison. If this cannot be effected, and they are compelled to continue occupations where they are exposed to the fumes of mercury, they must wear a protection over their face, and exercise the greatest personal cleanliness.

Drugs are of little service, the treatment is altogether prophylactic.

PARALYSIS AGITANS.

Shaking palsy, or the *trembles*, is a disease of advanced life characterized by motor weakness and tremors of the voluntary muscles, especially of the limbs, occurring independently of muscular exertion, which are finally followed by paralytic symptoms.

Morbid Anatomy.—As yet no constant changes have been discovered. Some authorities consider it of spinal, others of cerebral origin. Among the former are Charcot, Lebert, Marshall Hall, and Rosenthal. Among the latter are Oppolzer and Skoda. Senile changes in the brain and cord are found in a certain number of cases. There may be sclerotic patches in the pons, the medulla oblongata, the optic thalamus, and hippocampus major, and Charcot has found increase of the epithelium of the central canal in the cord with pigmentation of the cells in the posterior columns of Clark. Diseased arteries and slight sanguineous exudations have also been noticed.

Etiology.—Rarely occurring before forty, the liability to it is increased every year thereafter. It is more common in men than women, and occurs chiefly in the lower classes. Violent emotions, as grief, fear, anger or distress of mind, degeneration of the heart and *vessels*, and great bodily fatigue and exposure, are among its exciting causes. There are no indications that the disease is hereditary.

Symptoms.—In nearly all cases paralysis agitans is insidious in its approach, and begins in one foot, hand, or possibly a single finger or the thumb, as a slight oscillating motion, which is quite rhythmical and characteristic. For a time this trembling may be intermittent, but appears without any apparent cause and unexpectedly. In this early stage it can possibly be arrested by an effort of the will, a condition in marked contrast with the very decided increase in the tremor which late in the disease follows every effort to control the muscles. As the disease advances similar oscillating movements affect the muscles of the forearm, arms and shoulder, and the entire limb is in a continuous tremble. The remaining limb of the affected side usually becomes involved before the disease crosses the median line. Pain, weariness and stiffness in the affected muscles precede, in some cases, the development of the characteristic tremors; or the disease may attain its full development through a series of increasingly frequent sudden attacks of tremor lasting only a few days each.

At the height of the disease nearly all the limbs are involved in an incessant motion which is liable to severe exacerbation upon muscular exertion or during mental disturbance, and which ceases only during sleep or anæsthesia.

Later on, rigidity, painful cramps, and contractions affect not only the muscles of the limbs but also those of the trunk, neck and face, giving the patient quite a characteristic appearance. The countenance assumes a fixed, staring look of distress, the head is drawn forward and the trunk flexed; the lower limbs and arms, which are drawn away from the side, are rigid, and all the joints are flexed, often causing marked deformity of the hands. Although the rigidity of the limbs does not prevent walking, the patient's gait is characteristic. As he rises, or when he stands, there is great unsteadiness and difficulty in maintaining equilibrium, which, as he starts to walk, causes him to *run* forward to avoid falling. This disturbance of equilibrium is not associated with vertigo.

The muscles of respiration and deglutition are not involved, but the voice is often tremulous and speech is slow, hesitating and laborious, so that words are distinctly broken up into syllables. Although muscular movements are attended with extreme fatigue, the force of the contractions is but slightly diminished until late in the disease, when, from increase in the rigidity, the patient takes to his bed, and the muscles suffer in their nutrition or become distinctly fatty.

As the end approaches, the memory and intelligence fail in connection with the generally defective nutrition. Although the disease may last for twenty or thirty years, death most commonly results from some intercurrent disease.

Differential Diagnosis.—In *disseminated sclerosis* tremors occur only when the muscles are in use ; the disease begins in the *lower* limbs, affects younger persons, and paralysis occurs early. The patient has no tendency to run forward, and does not present the peculiar physiognomy of shaking palsy. *Senile, alcoholic, lead* and *mercurial* trembling are readily diagnosticated by the previous history and concomitant symptoms.

Prognosis.—Paralysis agitans is a very chronic disease, and the outlook is *never* favorable. It may last twenty-five years. After a few years the muscles waste, the patient is confined to his bed, there is physical and mental exhaustion, bed-sores form, and death results from asthenia or complications. The more common complications are acute lobar pneumonia and pleurisy. Paralysis agitans has been recovered from in the early stages, but Eulenberg says that there is reason to doubt the diagnosis in such cases.[1]

Treatment.—No definite results have been attained by the use of any remedy. All the nerve stimulants, tonics, and sedatives have been employed, of which Charcot considers hyoscyamus the only useful one, and the effects of this are temporary. The constant current has seemed to have some value as a distinctly curative agent. Beyond this, general tonic treatment is the most that can be attempted, and this should never be omitted.

FACIAL PARALYSIS.

(*Bell's Paralysis.*)

Bell's paralysis is a paralysis of the muscles of the face due to any lesion implicating the nucleus or fibres of the seventh pair of nerves.

Etiology.—It may be caused within the skull by blood extravasations, tumors, and inflammatory products, which give rise to pressure. Fracture and morbid growths may cause pressure on the nerve, in its passage through the cranial bones, sufficient to produce the paralysis. It may occur in connection with disease of the internal or middle ear and from local neuritis. Outside the skull, blows, wounds, swellings of the parotid gland or other tumors may cause it. *It is most frequently the result of a draught of cold air on the side of the face, especially while sleeping.*

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[1] Handfield Jones thinks that there are two forms: one entirely incurable, occurring in *old* persons and depending upon organic changes in the central nervous system ; the other, in *younger* persons, curable, and probably not dependent upon organic changes.

Symptoms.—Its onset is usually gradual; but when fully developed its symptoms are striking and characteristic. All the muscles supplied by the seventh nerve on one side of the face are paralyzed. The forehead is smooth and motionless on the paralyzed side, the corner of the mouth is drawn to the opposite side, and the paralyzed side closes less perfectly than the other. The patient cannot close the eye on the affected side. As soon as the face is moved the paralysis is unmistakable. Whistling and drinking are impossible. Certain letters, as P and B, cannot be pronounced; food collects between the cheek and teeth on the palsied side. The tears run down over the cheek, and, if the chorda tympani is involved, the sense of taste is perverted or destroyed on one-half of the anterior portion of the tongue. At the same time the salivary secretion is diminished. The uvula is usually deflected; the ala of the nose becomes flaccid, and the nostril on the affected side is narrowed and loses its rotundity. Imperfect closure of the eye exposes the organ to all sorts of injuries, hence disease of the cornea and conjunctiva is very common. During the first two or three days the muscles show increased irritability to the electrical current; but they gradually lose their Faradic, while they retain their galvanic irritability.

Differential Diagnosis.—When otorrhœa, disturbances of hearing, obliquity of the uvula, diminished salivary secretion, and loss of taste occur, the origin of the paralysis is within the aquæductus Fallopii. When the taste is normal and the uvula straight the cause is usually peripheral, *e. g.*, cold. In these cases also electro-muscular contractility is rapidly lost. The origin may be supposed to be central when *other* nerves are involved. Bell's palsy is usually associated with paralysis of the sixth nerve.

Prognosis.—In organic disease of the brain or with lesions of bone the prognosis is unfavorable. When arising from cold, slight injuries, or syphilis the prognosis is favorable. Complete recovery is usually reached in two or three months. There is no rule by which one can estimate its duration. The more the electro-muscular contractility is diminished the less the chances of complete recovery.

Treatment.—When due to cold, apply a few leeches to the mastoid process, followed by hot fomentations; subsequently blisters behind the ear and other counter-irritants may be resorted to, and the alternate Faradic and voltaic currents are to be relied upon. Massage and shampooing may be tried. Gowers recommends inunctions of oleate of morphia. When due to syphilis, anti-syphilitics are indicated. Niemeyer recommends mercurial ointment.

ECLAMPSIA AND INFANTILE CONVULSIONS.

Epileptiform convulsions are of frequent occurrence in connection with distinct lesions of the nervous centres as well as with various reflex disturbances. Indeed, they may be symptomatic of any cerebral disease, as compression from fracture, hemorrhage and tumors, or thrombosis, embolism and inflammatory processes. In those conditions where they can be ascribed to distinct pathological changes they are termed eclampsia. Clinically such convulsions, as well as those from uræmia and other poisons, are often identical in appearance with epileptic fits, and are to be diagnosticated by the concomitant symptoms.

When such convulsions occur in children, as they often do from reflex irritation during teething, in gastro-intestinal disorders, in the early stages of blood poisoning, and, indeed, in any condition which in the adult produces a chill, they are then called infantile convulsions. Although frequently indistinguishable from epilepsy, the convulsions of eclampsia, and more particularly infantile convulsions, may present only a portion of the true epileptic fit or even be very slight. They are, moreover, less regular in their occurrence, and, until the cause is removed, tend to increase in frequency and intensity. They are less sudden in their development also, and, although much more fatal, owing to their frequent dependence upon an irremediable cause, generally cease permanently when the cause is removed. Infantile convulsions present the widest range of intensity, but are always a cause of anxiety, especially when the respiratory muscles are involved, as is frequently the case.

The indications for *treatment* are, of course, found in the cause. In syphilis, uræmia, anæmia, gastro-enteritis, etc., the treatment will in most cases be under way before the occurrence of convulsions. When, however, they are the initiatory symptoms, all possible causes must be carefully sought. The discovery of a cause will indicate the treatment. Infantile convulsions often arise from slight causes that frequently escape detection. In such cases the general health must be improved, the alimentary canal freed from a possible hidden cause, and the clothing carefully inspected and made loose and unirritating. The usual anti-spasmodics, belladonna, the bromides, etc., may then properly be used. Hot baths, counter-irritants to the back and neck, or cold to the head, are often of service. Chloroform is the most appropriate agent for controlling the spasms temporarily.

TETANUS.

Tetanus, or *lock-jaw*, is a tonic spasm with paroxysmal exacerbations of the voluntary muscles; those of the lower jaw, neck, and pharynx are usually first affected. Acute and chronic varieties are recognized; the latter is called *tetanus mitis*.

Morbid Anatomy.—There are no constant lesions in tetanus, and those which are commonly present are quite as possibly secondary as primary.

In traumatic tetanus the nerves supplying the affected parts are sometimes inflamed, but even this condition is not invariably present. In the cord there is often more or less hyperæmia, with slight effusion, and perhaps extravasation. This is frequently attended by some œdematous softening and interstitial exudation of finely granular or structureless matter, especially in the gray substance, in the fissures, and on the surface of the cord. Tetanus is usually traumatic and may follow the most trivial injury, as a splinter in the finger, but is more apt to develop after compound or complex fractures, lacerated, crushed, and punctured wounds, and wounds complicated by the presence of foreign bodies. It may occur after abortion or normal delivery, and *trismus nascentium* is ascribed to the wound at the navel.

Climatic conditions have a distinctly exciting influence in the production of tetanus. It is much more common in hot than in temperate climates, and rapid changes of temperature, cold and wet, are especially favorable to its development. It is said that fright, anxiety, or depression markedly predisposes to its occurrence, as in armies it prevails most extensively among the defeated.

Clinically, tetanus can be excited by strychnine, ergotin, brucine, picrotoxin, and caffein. Occasionally tetanus arises from unknown influences, when no wound or abrasion is present, and when the only apparent assignable cause is exposure to wet and cold. More recent investigations have seemed to show that tetanus may be a specific disease of bacterial origin.

Tetanus may occur at any age and in either sex, but is most frequent in adult males.

Symptoms.—Tetanus generally comes on in from six to twelve days after the injury, but may be delayed three or four weeks or appear within a few hours. In the largest number of cases it begins with stiffness of the muscles of the neck and jaw. This quickly extends to the muscles of mastication and facial expression; the patient's jaw becomes locked and the head fixed, and the face wears a peculiar frown. The tonic stiffness is aggravated by every attempt to use the muscles. Deglutition is difficult, and later becomes almost impossible. By degrees the other muscles are involved, the trunk is stiff and more or less curved, the abdomen tense and hard, and the limbs extended and rigid. When the diaphragm is moved, a sharp, sudden pain shoots through the body from the ensiform cartilage, which is considered diagnostic. It is accompanied by intense dyspnœa. This general rigidity of the muscles is continuous and progressive, but is marked by paroxysmal attacks in which all the symptoms are immensely exaggerated. They are excited by any muscular action, by jars and other slight causes, or may occur spontaneously. During a spasm all the muscles become powerfully contracted. The limbs are extended, the back arched, and the face assumes the *risus sardonicus.* The head is retracted, and the patient may rest only on his head and heels. The respiratory muscles suffer also, and respiration may be entirely arrested, and the face become cyanotic. As the paroxysm passes away it is only a remission; the muscles are still hard and stiff, the jaw closed, and the respiration rapid and shallow. The intense cramping pain of the paroxysm gives place to a heavy ache and soreness.

Notwithstanding the severity of the disease, consciousness and intelligence are rarely impaired, and the temperature and pulse-rate are only elevated on account of the muscular action. Just before death, however, in many instances, there is a rapid and enormous rise in the temperature, which may reach 112° or 114° F. The urine is scanty, the bowels are constipated, and the body is bathed in a profuse sweat. Reflex irritability is increased to a high degree throughout.

Differential Diagnosis.—The absence of headache, delirium, and coma, and a normal temperature in the intervals between the attacks, will suffice to distinguish tetanus from any *cerebral* or *cerebro-spinal* inflammation.

Hysteria, hystero-epilepsy, and sometimes *epilepsy* may simulate it, but the development of the disease quickly affords a diagnosis.

Strychnia poisoning is to be differentiated by the history of the case and the examination of voided matter. In strychnia poisoning, consciousness is lost, and the muscles of the jaw, head, and neck are last and least affected.

Prognosis.—Tetanus usually terminates fatally before the tenth day; but if the twelfth day be passed and the temperature does not pass 102° F., and the respiratory muscles are not involved; or if the disease has occurred at a remote period from the reception of the wound, the outlook is quite hopeful. When the patient is young, when strabismus occurs, or the wound is very recent, and when rigidity appears early the case is nearly always fatal.

Treatment.—So far as is known, no treatment has any controlling effect upon tetanus. Innumerable remedies have been tried, with equally bad results. A highly nutritious diet, with alcoholic stimulants, is, perhaps, the best treatment. Alimentation must be carried on by a stomach-tube or by the rectum. Recently, curare, nitrite of amyl, and hydrate of chloral (in forty-grain doses) seem to be the favorite drugs. Locally, ice and cold effusions to the spine prove beneficial, although hot applications are more grateful to the patient.

The utmost care should be taken to avoid all irritation and to keep the patient in the most absolute quiet.

NEURALGIA.

The term *neuralgia* is applied in a very general way to *pain*, which is either of idiopathic origin or constitutes the principal and at times the only symptom of some obscure lesion. Neuralgia is a symptom indicative of direct injury to, or altered nutrition of, a sensory nerve, which in the former case is more or less persistent, but in the latter is usually paroxysmal.

Morbid Anatomy.—It may be functional or organic; but in the majority of instances no changes can be found after death.[1] When neuralgia is a symptom of *acute neuritis* or *peri-neuritis*, the nerve trunk is hyperæmic and swollen or degenerated and atrophied; when a symptom of *chronic neuritis*, the nerve has undergone sclerotic processes, and compression with degeneration of the nerve-substance follows.

Neuritis may be descending or ascending. When it attacks nerves at various points it is called disseminated or migrating neuritis. When neuralgia is a symptom of pressure from *tumors*, either in brain, cord or at any point along the nerve trunks, the pain will be confined to the single nerve. Gummata, aneurisms, and osteomata are the tumors which usually induce such compression.

Etiology.—Neuralgia is often an hereditary disease in those of a neuropathic tendency. Any disease causing general, or local, permanent or transient *anæmia*, is a marked *predisposing* cause.

Among *exciting* causes are cold (especially *damp* cold), lead, mercurial and other states of chronic blood poisoning, and traumatism. Disease of the genito-urinary tract, especially in women, often excites reflex or sympathetic neuralgia in remote nerve-trunks. Reflex neuralgia is also induced by decayed teeth, dyspepsia, worms, constipation, etc. Neuralgia may follow or accompany herpes zoster, and occurs *very* frequently in convalescence from relapsing fever.

It is rare before puberty, but just *at* this epoch there is a marked predisposition to it. Those between twenty and fifty years of age suffer most frequently. Women are more liable than men; but males suffer from sciatic neuralgia much more frequently than females.[2]

The theory that neuralgia often depends on dilatation of the venous plexuses which surround a nerve at its exit from a bony canal, is supported by the fact that the first branch of the trigeminus suffers far oftener than either the second or third, or both combined.[3]

Symptoms.—Before the actual pain begins in a nerve, there may be numbness, slight cutaneous hyperæsthesia, or some peculiar skin sensation which is well-known by the neuralgic individual. The pain is at first intermitting, later it is continuous with slight remissions. The character of the pain varies: it may be dull, boring, stabbing, tearing or darting, and is confined very distinctly to the course and distribution of the affected nerve. Indeed, many patients trace exactly the course of some nerve when pointing out the locality of the pain. Sudden movements, as turning and coughing, often increase the pain.

Increase of pain on pressure is an important point; the exacerbation is greatest during a paroxysm, and greater in proportion to the intensity of the original pain. Certain points are markedly sensitive: these are at the exit of nerves from bony canals, or foramina, the spot where they pass through a muscular aponeurosis, at their bifurcation, and where terminal branches become superficial. These pain-points are better marked the longer the patient has suffered from neuralgic attacks. In connection with the pain, there is generally associated with it some vaso-motor disturbance, as extreme pallor or vivid redness and reflex movements and twitchings of the muscles. Should the nerves of a *gland* be attacked, secretion will probably be *increased*. After cessation of the pain the part often feels sore and bruised, and there is a general sensation of exhaustion and weariness.

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[1] It is claimed that the acid products of metamorphosis of nerve-tissue acting upon the nervous system must be neutralized by the blood, before pain ceases. Also, that nutritive lesions of the central sensory tract within the confines of the gray matter are the essential lesions. Peripheral pain is supposed to originate in the cord.

[2] Henle states that the left side is predisposed to intercostal neuralgia on account of the arrangement of the venous circulation.

[3] *Allg. Wien. Med. Zeit.*, 1876, pp. 24, 26.

Actual temporary paralysis, muscular spasm, herpetic eruptions, and anæsthesia of the skin may complicate or follow an attack of neuralgia, and later the muscles supplied by the affected nerves may be atrophied and become abnormally weak. During a prolonged paroxysm the pain may extend from one nerve to another of a different origin.[1] In a few rare cases mental effort or excitement will exacerbate, or even excite, a paroxysm of neuralgia.

If neuralgia be caused by *neuritis* the pain is more continuous, and the nerve *may* be felt as a hard cord beneath the skin, which latter is red and œdematous. With neuritis of a mixed nerve, twitching and contractions occur with the pain. In neuralgia of functional origin, the pain is more likely to shift and to involve corresponding tracts on the other side of the body or head.

One of the most common forms of neuralgia is that of the *tri-facial nerve*, usually attended with painful spasm, called *tic douloureux*. One or two, rarely all the divisions, may be involved. The first branch is its usual seat, when it is termed *brow-ague;* the third is rarely attacked. When the *ophthalmic division* is affected the neuralgia is called hemi-crania or *migraine*.

Clavis hystericus is a variety of *tic* in which there is a sensation as of a nail being driven into the skull. It is usually met with in anæmic females. The hair on one side of the head or one eyebrow may turn white, or pigmentation may occur along the course of the pain, and the tongue on the side of the pain may exhibit epithelial overgrowth in long standing *tic douloureux*.

Acute glaucoma and recurrent iritis are said to result from trophic changes due to neuralgia.[2]

Pain on pressure is usually best marked (1) at the exit of the frontal branch, (2) the exit of the inferior maxillary branch, (3) over the temporal and parietal bones, or (4) along the supra-orbital ridges (supra-orbital neuralgia).

Sciatica is a neuralgic affection of the sensory nerves of the sciatic plexus.

It may be caused by the pressure of tumors and inflammatory exudation within the pelvis, or by caries or carcinomatous vertebræ at the point where the nerves pass through the intervertebral foramena. Irritation of the peripheral branches of the sciatic, due to pressure along the line of the nerve, from tumors, etc., may cause sciatica, but in the majority of instances the origin is rheumatic and the direct result of taking cold. Chronic malarial infection may be the cause of sciatica. It is most frequently met with in males between the ages of twenty and sixty.

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[1] Epileptiform neuralgia is that variety of tic douloureux where the seizures are very abrupt and accompanied by spasm of the facial muscles.

[2] Anstie states that near the painful parts the periosteum and the fibrous tissue are thickened.

It is usually preceded by tingling or stiffness in the buttock, back of the thigh, knee and leg. The pain may be continuous or intermittent, and its most frequent seats are the posterior and outer part of the thigh (particularly near the tuberosity of the ischium), the outer side of the ankle, and the dorsum of the foot. It usually comes on gradually, the pain becoming more intense at night. The patient usually lies with his legs flexed. In walking he moves the affected leg slowly, as any sudden motion greatly aggravates the pain. The pain is most markedly increased by pressure over the posterior iliac spine, at the fold of the buttock and the head of the fibula. Cramps in the muscles of the leg are common. The limb may be atrophied and the patient pass into a semi-paralytic condition, which is very apt to be chronic. It is a very obstinate affection, lasting usually from six weeks to two months, though it may last for years. Relapses are not uncommon.

Intercostal neuralgia is an affection of *any* of the dorsal nerves; the anterior branches of two or three of the nerves upon the left side are those usually affected. It occurs in women as a rule. Intermittent pain is felt in the region of the sixth, seventh, eighth and ninth intercostal nerves, tearing or stabbing in character, increased by coughing or sneezing, and perhaps accompanied by a dry cough.

There are three diagnostic points of tenderness: (1) at the exit of the nerves from the spine, (2) at the side of the chest, where they become subcutaneous, and (3) near the sternum or median line at the terminal branches. Cardiac palpitation, dyspnœa, nausea and vomiting are frequent symptoms of this so-called *false pleurisy*. Herpes zoster, intolerable itching, and attacks of angina pectoris often complicate it.

Cervico-occipital neuralgia is usually attended by pain along the course of the occipitalis major,[1] and often resembles that form of muscular rheumatism called torticollis, or wry-neck. (See art. Rheumatism.) A branch of the *brachial plexus* may be involved; the ulnar, however, is more frequently affected than any other.

Coccyodynia is common in women, and is due to neuralgia of the coccygeal plexus.

Headache.—Headache, or cephalalgia, is properly a form of neuralgia, as it can only be referred to the sensory nerves supplying the meninges and scalp, and like other neuralgias is of both organic and functional origin. It is a frequent symptom of cerebral disease, either inflammatory or such as produces compression of the cranial contents, and is especially severe in the acute forms of meningitis and some cerebral tumors. It results from disturbance of the cerebral circulation, which causes either compression of the cranial nerves or anæmia, and consequent disturbance of nutrition. Its primary cause, however, is more frequently in other organs, as the or genito-urinary tract, in which cases the headache is the result disturbances, frequently of the circulation, from vaso-motor irritati a similar nature is the headache resulting from the strain of th muscle, consequent upon defects of refraction. Again, headach quently a symptom of blood poisons, as in rheumatism, gout an diseases.

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[1] Gray's *Anatomy*, pp. 636-637.

In these diseases, as probably also in headache with high tempera condition is presumably one of direct irritation of nerve centres, o fective nutrition. Headache assumes a great variety of forms. It limited to one half the head, to the forehead, vertex, occiput, tem gion, or any point on the cranium, or it may be diffuse and exten eye, face and neck. In character and severity it may assume an characteristics of neuralgia. Headache is a symptom of exceedin cult interpretation. In a general way, however, it may be stated th ache of gastric or hepatic origin is commonly frontal and thro character and associated with cerebral congestion. It may be bil unilateral. Headache at the vertex is quite constantly symptomati bral disturbances of local origin, or due to reflex irritation startin pelvic organs, especially the genital tract of the female. Pain in t ital region is mostly an accompaniment of disorders of circulation, motor spasm and anæmia in particular. The pain of cerebral com or tumor, although often diffuse, is generally localized, persistent, intense.

All forms of cephalalgia may be attended by hyperæsthesia, esp the optic and auditory nerves, with subjective sensations of l sound, by vertigo, nausea, drowsiness or wakefulness, and possibl um. Visceral neuralgias have been considered in the list of Visc eases.

Differential Diagnosis.—Neuralgia may be mistaken for *myalgi litic periostitis,* and for *cerebral abscess.*

Myalgia is distinguished by its non-paroxysmal character, by being increased by *motion,* and by the fact that the attachment muscles are the points chiefly involved.

Syphilitic periostitis is to be distinguished from neuralgia by ence or absence of other symptoms of constitutional syphilis.

Cerebral abscess often occurs secondarily to caries of the internal after otitis in childhood; neuralgia rarely appears before puberty bral abscess frequently follows a blow or injury; neuralgia comp seldom. In the former there are no true *points douloureux;* present early in severe neuralgia. In cerebral abscess the pain completely intermit; intermissions of pain, complete, and of con length, occur in neuralgia. The pain is at first severe in cerebral in neuralgia it is slight at first and gradually exacerbates. Pain bral abscess is often limited in situation, seems deep-seated, thou it has no relation to the site of the abscess; in neuralgia pain is su and follows the distribution of recognizable nerve branches belo the trigeminus or great occipital. In cerebral abscess there are no well localized vaso-motor or secretory complications, while lachrymation or congestion of the conjunctiva usually occurs in neuralgia. Cerebral abscess is rare in old age, and then generally traumatic; neuralgia is most common at that period.

Prognosis.—Life is rarely compromised by neuralgia, but when it is persistent the general health may be seriously affected. When occurring in early life and with no hereditary predisposition the prognosis is the most favorable.

Treatment.—Neuralgia has been well said to be *the cry of a nerve for better blood.* Should anæmia be evidenced, a generous diet, cod-liver oil, the hypophosphites, or small doses of phosphorus and the appetizers, along with quinine, iron and strychnine should be ordered. Neuralgia due to *syphilis* demands iodide of potash; to rheumatism, the anti-rheumatics; to gout, colchicum; and to malaria, quinine, but in many non-malarial cases also, especially in *tic,* quinine is the most effectual remedy. A patient with neuralgia should be removed from all exposure to cold and irritations of all kinds.

Locally, blisters, the continuous current, chloroform, opium, belladonna and veratria liniments, and cold, or very hot water may be applied, and these sometimes afford permanent, nearly always temporary relief. Aconite enjoys the highest reputation at the present day among local remedies. Firing, sinapisms and actual cautery are frequently beneficial. Sometimes prolonged residence in a warm, dry climate is the only means of effecting a permanent cure.

For *immediate relief of pain,* morphine is the most effectual. Neuralgic attacks and headache that are accompanied by *flushing* of the face are often relieved by ergot. But when the face is very *pale,* nitrite of amyl is to be preferred. Gelsemium is sometimes especially effectual in the treatment of trigeminal neuralgia. This and croton chloral are largely employed. In severe chronic neuralgias a portion of the nerve may be excised (neurectomy), or the nerve may be simply cut (neurotomy). More recently antipyrin has been employed in all forms of neuralgia. The results are extremely satisfactory in many cases. It is not always successful, however; and, in any case, it requires increasing doses after a short time. It can be regarded only as a temporary expedient, and its favorable effects should never lead to neglect of radical measures for the removal of the cause.

Nerve-stretching may be practised upon any trunk which can be surgically reached. The sciatic is the nerve which has been stretched with most success. In *headache,* cold to the head and heat to the feet, or at times the persistent application of heat to the head for several hours, will afford relief. Guarana, caffeine, and similar remedies are often very useful in *sick headache.* In these cases a purge or an emetic will also frequently bring relief.

In all severe cases of sciatica, in addition to the treatment of neuralgia in general, *absolute rest* is essential to its successful management. If it is caused by gout, rheumatism, or syphilis, treatment appropriate to these conditions must be employed. If there be a chronic malarial taint, quinine and arsenic must be given in full doses. The hypodermic injection of morphine gives the most speedy relief. The point of the needle should be introduced deep into the tissues over the exit of the nerve. In many instances its daily use for some time will cure sciatica, even of long standing. The continuous voltaic current is often palliative and sometimes curative. The systematic treatment with baths at the Hot Springs of Arkansas and Virginia, I have found especially efficacious in sciatica that has resisted all other remedial measures. The application of the hot iron and blisters along the course of the nerve have, in some instances, acted remedially.

MEGRIM.

(*Sick Headache.*)

Sick headache, or hemi-crania, is a form of neuralgia attended by marked gastric and nervous disturbances.

Morbid Anatomy.—Megrim is probably due to disordered cerebral circulation, the exciting cause of which is vasomotor disturbance. Changes similar to those of epilepsy are generally considered to be the pathological condition ; that is, vasomotor irritation with arterial spasm and consequent anæmia of the cerebral ganglia, followed by relaxation and congestion. The neuralgic element is possibly due to compression of nerve trunks. It is hardly supposable that irritating elements in the blood could have such a selective action upon a single nerve. This condition, however, still demands an ultimate cause, which is probably nervous (cerebro-spinal) exhaustion, following prolonged irritation, as indicated in its etiology.

Etiology.—Megrim is often hereditary, or, more exactly, the nervous weakness and instability which predispose to the affection are hereditary. Whether inherited or acquired, it commonly develops before thirty, and subsides in later life. Digestive disturbances are frequent exciting causes, but a much larger proportion of cases are due to nervous irritation and exhaustion. It is an almost unfailing symptom of chronic uterine irritation or sexual excesses, and is frequently due directly to mental labor, worry, or excitement. In neurasthenic patients, it is often excited by over-exertion, or the lack of it, by too much or too little sleep, and by irritation of the nerves of special sense—flickering light or loud noises—and in some cases the slight disturbance of co-ordination attendant upon the use of the stereoscope or opera-glass is sufficient to excite an attack.

Symptoms.—As the term indicates, hemi-crania is almost invariably confined to one side of the head, and is generally distinctly localized in the frontal, temporal, or occipital region, and even when it attacks all three places, or becomes diffuse, the pain is still most intense and persistent at a small circumscribed point in each region. In such cases there is often a sensation of an intra-cranial cord joining the painful points.

Frequently, and especially in cases due to ocular strain, the eye becomes the seat of pain and is tender and hyperæsthetic. Early in the attack the face may be pale and the cardiac action slow and weak. Very soon, however, the head becomes hot and the pulse slow, and with each heavy heart-beat the carotids pulsate strongly and the pain is greatly increased. Generally within a few hours nausea supervenes, and may be attended by distinct recurring chills and paleness of the surface. The patient is greatly depressed and is wretchedly sick. If the pain is not too severe he may fall asleep, to wake in the morning with only a soreness about the scalp and stiffness of the muscles of the neck remaining. More frequently the nausea increases until relieved by an attack of vomiting. A few hours of sleep then restores the patient to his usual condition.

Quite characteristic premonitory symptoms are present in many cases. The most common are disorders of vision in the form of retinal anæsthesia or irritation. The anæsthetic spot may be located in any part of the retina, but generally affects the macula lutea. Retinal irritation causes the patient to see variously colored lights and scintillations. The disturbance in vision may commence with a wavy glimmering at the outside of the field of vision or by the appearance of a black spot close to its centre. Similar disturbances of the other nerves of sense, either irritative or paralytic, may be present, but they are less common than the visual disturbances.

Hemi-crania may last from a few hours to two or three days, but in most cases is relieved within twenty-four hours. It is very apt to recur at regular intervals, and become more intractable with each attack.

Treatment.—By way of prophylaxis, the patient should avoid all known causes of the attack, and pursue a tonic course of living. At the beginning of the attack full doses of alcoholic or other stimulants may prevent its development. Later, the bromides, quinine, strychnia, belladonna, cannabis indica, caffeine, guarana, and chloral at times afford relief. When nausea is present, however, an emetic, followed by a few hours' sleep, brings about the most speedy cure. Morphia hypodermically is the best and surest means for the relief of pain. Antipyrin, as in other forms of neuralgia, will often afford speedy relief.

During the interval between the attacks the treatment should be such as will as far as possible render inoperative its cause. No two cases will require the same hygienic or therapeutic measures. The main thing is to overcome the acquired or hereditary neurotic tendencies of the patient by diet, exercise in the open air, and cheerful surroundings. Drugs accomplish very little for this class of sufferers except to give temporary relief.

FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

I shall consider under this head :

I. *Epilepsy.*
II. *Hysteria.*
III. *Hystero-Epilepsy.*
IV. *Catalepsy.*
V. *Neurasthenia.*
VI. *Chorea.*
VII. *Sunstroke.*
VIII. *Spinal Irritation.*
IX. *Vertigo.*
X. *Sea-sickness.*

EPILEPSY.

Epilepsy is a chronic functional disease of the nervous centres marked by sudden signs of temporary loss of consciousness or some other mental disturbance, accompanied by tonic or clonic convulsions.

In its typical and fully developed form the disease has received the name *epilepsia gravior,* or *le haut mal,* and when mild and incomplete is called *epilepsia mitior, le petit mal,* or *epileptic vertigo.*

Morbid Anatomy.—Different portions of the nervous system have been regarded as the seat of lesions which may cause epileptic seizures. Some have located these lesions in the convolutions of the hemispheres, the ganglia at the base, or the pons and medulla oblongata. Others have claimed that all the nervous centres are involved. Death has occurred in some cases during an epileptic seizure in which no change was discovered at the autopsy except cerebral hyperæmia. Many pathologists claim that the vaso-motor centre in epileptics is so easily excited that slight impressions result in arterial spasm producing anæmia of the brain. Brown-Séquard states that the true seat of epilepsy is in nerve cells having the power of producing muscular contractions, and that these cells are located chiefly at the base of the brain. Experiments on animals show that epileptiform convulsions may be produced by irritation of the skin after the removal of the brain and cerebellum.

The pathology of epilepsy is still obscure, and no uniform, constantly recurring histological changes have as yet been discovered. Russell Reynolds sums up its pathology as follows :

I. The seat of primary derangement is the medulla oblongata, upper portion of the spinal cord, and vasomotor system of nerves.

II. This derangement consists in an increased and perverted readiness of action in these organs;[1] the result of such action being the induction of spasm in the contractile fibres of the vessels supplying the brain, and in those of the muscles of the face, pharynx, larynx, respiratory apparatus, and limbs generally. By contraction of the vessels, the brain is deprived of blood, and consciousness is arrested; the face is, or may become, pale by being deprived of blood; from contraction of the muscles mentioned there is arrest of respiration, the chest walls are fixed, and the other pheuomena of the first stage of the attack are brought about.

III. The arrest of breathing leads to the special convulsions of asphyxia, which are in direct proportion to the completeness and continuance of the asphyxia.

IV. The subsequent phenomena are those of poisoned blood, *i. e.*, of blood poisoned by the retention of carbonic acid, and altered by the absence of a due amount of oxygen.

V. The primary nutrition change which is the starting-point of epilepsy may exist alone, and epilepsy be an idiopathic disease.

VI. This change may be transmitted hereditarily.

VII. It may be induced by conditions acting upon the nervous centres directly, such as mechanical injuries, overwork, isolation, emotional disturbances, excessive venery, etc.

VIII. The nutrition change of epilepsy may be a part of some general metamorphosis, such as that present in the several cachexiæ, rheumatism, gout, syphilis, scrofula, and the like; and further, it may often be associated with change in the cortical substance of the cerebral hemispheres.

IX. It may be induced by unknown circumstances determining a relative excess of change in the medulla during the general excess and perversion of organic change occurring at the periods of puberty, pregnancy, and dentition.

X. It may be due to diseased action extending from contiguous portions of the nervous centres or their appendages.

XI. The so-called epileptic aura is a condition of sensation or of motion dependent upon some change in the central nervous system, and, like the paroxysm, is a peripheral expression of the disease; not its cause. Paralysis of the cerebral blood-vessels, and resultant hyperæmia of the medulla, is a constant change in a severe epileptic seizure.

Etiology.—Thirty per cent. of epileptics give a history of an *inherited tendency*, either to epilepsy or some neurosis; and children of consanguineous marriages are often epileptics. It most frequently develops between the ages of ten and twenty. The next most frequent period is between the second and the tenth year. In a small number of cases it exists at, or develops immediately after, birth.[1] Sex appears to have no influence, except in hereditary epilepsy, which develops earlier among girls than boys.[2] That puberty is an exciting cause of epilepsy is a fact accepted by the majority of authorities. Irritation of some portion of the nervous system is its frequent exciting cause, such as injury to peripheral nerves, the skull o meninges, and diseases of the brain substance.[3] Sunstroke has induced it. Epileptiform seizures are not infrequent symptoms of disease of different portions of the nervous system. Among its nervous causes, excesses in venery and onanism have undoubtedly been over-estimated.

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[1] Gowers thinks that loss of inhibitory function of the nerve cell is far more likely than increased irritability. For a complete and exhaustive summary concerning pathology and pathogenesis, *vide* Hughlings-Jackson, *Medical Times and Gazette*, 1879, vol. i., p. [illegible]

Among its psychical causes are great anxiety, grief, mental overwork, and long-continued depressing emotions. Sympathetic epilepsy is claimed to arise in children from dentition and intestinal irritation. It may aris from irritation of the genitals, anomalies of menstruation, and phimosis Blood changes are also enumerated as among its causes.[4]

Symptoms.—The phenomena of epileptic seizures differ so widely that i is impossible to give a description which will answer for all cases. *Epilepsia gravior* may or may not be preceded by premonitory symptoms. I present, these warnings may precede the seizure for a day or only for tw or three minutes. The epileptic aura of Galen, the sense of a mist or vapor rising from the feet, occurs only in rare instances. Under the head o prodromata are included changes in disposition, moroseness and irritability cold feet, spasm of certain muscles, epistaxis, headache, vertigo, a marke increase or decrease in the sexual appetite, optical illusions, hallucinations involuntary discharge of urine and fæces, great somnolence or insomnia darkening of the skin, changes in the appetite, cardiac palpitation, cardialgia, vomiting, abundant flow of tears, and excessive secretion of saliva Sometimes the attack is preceded by a definite sensation referred to th head, stomach, or limbs. Drawing the head toward one shoulder is sometimes a warning of an epileptic seizure. In the majority of cases prodromata are absent, and the onset of the fit in a typical attack is sudden an attended by complete loss of consciousness. Uttering a loud, sharp cry the epileptic falls heavily, or sinks to the ground. The face is extremel pale immediately before and at the time of the seizure, and there may b tonic spasm of the muscles of the eye and face. The pupil is invariabl dilated at the onset. Tonic spasm of all the muscles immediately occurs the eyes are fixed and staring, and the muscles of the face, trunk, and extremities are rigid. Opisthotonos or emprosthotonos may occur, or th body may be bent sidewise. The face soon becomes dark, the veins turgid, and though the carotid pulsates strongly the radial pulse is weak Respiration is impeded and asphyxia rapidly develops, until after a fe seconds—rarely over a minute—*clonic convulsions* succeed the tonic spasm which, though general, are usually best marked upon one side. Sensation is usually wholly lost, and only in rare cases can reflex action be excited. The unconsciousness still continues. The muscles contracting and relaxing in quick succession induce the most violent contortions. The tongue is thrust between the teeth, which, closing upon it, cause deep indentations or lacerations of its edges. The teeth are sometimes broken; bones may be fractured or dislocated, and muscles torn from their attachments. The patient froths at the mouth, and, from the injuries to the tongue, the saliva is often bloody. The body is bathed in a profuse (sometimes very fetid) sweat, and frequently the contents of the bladder, bowels and vesiculæ seminales are forcibly ejected. All secretions are abnormally increased. The breathing is forcible, irregular, and rapid, and the auxiliary muscles are called into play; the face is turgid and distorted, the eyes protrude, the pupils are alternately dilated and contracted, inspiration is accompanied by loud gurgling noises, the pulse becomes full and labored, and when the cyanosis reaches its maximum the paroxysm, which seldom lasts longer than one or two minutes, begins to abate.

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[1] Reynolds and Echeverria state that hereditary epilepsy does not develop later than the twentiet year.

[2] Brown-Séquard states that after the twenty-fifth year women are attacked oftener than men.

[3] Westphal (*Berlin. Klin. Wochen.*) has shown that in guinea-pigs blows on the head may immediatel give rise to epileptiform attacks; and that on pinching the skin of the epileptogenic zone several week after, convulsions will occur.

[4] Gowers states that rickets causes it, through defective nutrition of the nervous system.

The fit may terminate suddenly or gradually. If it subsides gradually the spasms become less violent and frequent, the respiration quieter and more regular, and the patient passes into a comatose state. Consciousness gradually returns, and the patient appears as if waked out of a deep sleep. He recovers rapidly or remains confused, delirious, or maniacal for hours. A day may elapse before complete recovery is reached. The patient has no recollection of the attack. The degree and duration of stupor after an attack have no relation to the duration of the convulsive period. A slight seizure may be followed by great mental disturbance, and vice versâ. Marked dicrotism of the pulse often occurs as the patient is recovering consciousness; and for twenty-four hours the ophthalmoscope shows hyperæmia of the fundus oculi. The urine after the attack is increased in quantity and contains an excess of urea and phosphates.

Brown-Séquard gives the accompanying table of the causes and effects of an epileptic attack:

CAUSE.	EFFECT.
I. Excitation of certain parts of the excito-motor organs of the nervous centre.	I. Contraction of blood-vessels of the brain and face; tonic spasm of the muscles of the eye and face.
II. Contraction of the facial blood-vessels.	II. Facial paleness.
III. Contraction of the blood-vessels of the cerebral lobes.	III. Loss of consciousness, congestion of the base of the brain and the spinal cord.
IV. Extension of the excitation in the excito-motory organs of the nervous centre.	IV. Tonic contraction of the laryngeal, cervical and some respiratory muscles (laryngismus and trachelismus).

CAUSE.	EFFECT.
V. Tonic contraction of some respiratory and vocal muscles.	V. Epileptic cry.
VI. Further extension of the excitation in the excito-motory organs.	VI. Tonic contraction reaching most muscles of trunk and limbs.
VII. Loss of consciousness alone, or with tonic spasm in trunk and limbs.	VII. Fall or precipitation, forward or backward, to the ground.
VIII. Laryngismus, trachelismus and rigid spasm of some respiratory muscles.	VIII. Insufficient breathing; obstacle to entrance of blood into the chest and to its issue from the cranio-spinal cavity.
IX. Insufficient breathing, rapid consumption of oxygen, and detention of venous blood in the encephalon.	IX. Increasing asphyxia.
X. Asphyxia and perhaps pressure by accumulated venous blood in the base of the brain.	X. Clonic convulsions everywhere: contractions of the bowels, the bladder, the womb, increase of secretions, efforts to inspire.
XI. Exhaustion of the nervous power generally, and of the reflex excitability, especially return of regular respiratory movements.	XI. Cessation of the fit, coma, or fatigue, headache and sleep.

Le petit mal, or epilepsia mitior, is a momentary loss of consciousness; the patient while about his usual avocations suddenly stops, or drops whatever he may hold, has a fixed gaze for a second or two, and upon coming out of such a faint or blank proceeds as if nothing had happened. Sometimes these blanks may be accompanied by vertigo, and then the patient will stagger slightly. In rare cases he proceeds mechanically with whatever is occupying him during the paroxysm. He often pales for a few minutes and then grows red in the face. The pupils are somewhat dilated. The mind may be distinctly confused for a long period after such an attack. Sometimes momentary spasmodic contractions occur in the muscles of the face, tongue, throat, eyes and neck. The head is turned slightly to one side and the face is pale. Clonic spasms never occur. There may be slight cyanosis when the diaphragm and respiratory muscles are involved. Sometimes certain fingers, or part of one extremity, suffer transient spasm.

The variations from the typical phenomena of an epileptic seizure are so numerous that it is impossible to give them in detail; I shall only refer to those which are of common occurrence.

Sudden tonic spasm of the facial and thoracic muscles may be followed by a clonic convulsion without any loss of consciousness. An attack may be marked by such motor activity that the patient runs or walks rapidly during a period of complete unconsciousness. Sometimes maniacal excitement takes the place of the fit.[1] In this delirium an epileptic may be harmless and wanders around in a dazed condition; or be exceedingly dangerous to those about him. Kleptomania and dipsomania are said to be exhibitions of epileptic delirium. Brown-Séquard describes nocturnal attacks of epilepsy that not infrequently occur without the knowledge of the person so affected. In such cases the individual on waking is tired and exhausted; he has pains in the limbs, back and head, his mind is confused and his memory enfeebled; he is disinclined to exert himself, and remains during the day in a confused state. His tongue shows the indents of the teeth, and the pillow may be blood-stained. More rarely it is found that an involuntary discharge of urine has occurred. Such attacks, although frequent and violent, may remain altogether unknown and unsuspected by the patient or his friends.

Between the paroxysms the condition of epileptics varies greatly. In the majority there is no impairment of mental or physical condition; not infrequently, however, there is depression of nervous vitality and mental activity. Of all the abnormalities met with, sub-normal temperature is the most common.[2] Of the mental faculties, memory is most often impaired. Women show mental disturbances more frequently than men. The *earlier* epilepsy commences the *less* liable are mental changes to occur; and the mental deterioration is in *inverse ratio* to that of muscular disturbance. The most remarkable mental phenomena are those which constitute the so-called epileptic mania. Epileptics are frequently gloomy, capricious and irritable, all the finer psychical functions are dull, acquisition of new ideas is difficult, and hypochondria, melancholia and imbecility may occur as late exhibitions of the disease. Motor disturbances, such as tremors and clonic or tonic spasms, are not infrequent between the paroxysms. Epilepsia major is more common than epilepsia mitior, and hereditary tendencies seem to predispose more to the former than to the latter.

As regards frequency of attack there is the widest range: the first fit may also be the last; they may occur once a year, or two or three times in the twenty-four hours. In women it sometimes seems to be connected with the menstrual epoch. Eighty per cent. of all epileptics are attacked oftener than once a month; sometimes paroxysms occur on days that are multiples of seven. Often three or four fits occur in a day, and then ensues a period of immunity. When the seizures follow one another so closely as to leave no rest,[3] we have the status epilepticus, in which the temperature may rise to 108° F., or higher as death approaches. If the patient recovers, bed-sores are liable to be formed. Pneumonia and pulmonary œdema are apt to occur in this condition. Seizures of *petit mal* are usually very frequent. All the different forms may occur in the same individual.

Differential Diagnosis.—An epileptic seizure may be confounded with

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[1] *Delirium epilepticum.*

[2] Brown-Séquard, in *Quain's Dictionary*, states that the health is very poor, an opinion antagonistic to all other authorities.

[3] In Delasiaure's case there were twenty-five hundred attacks in one month in a boy of fifteen.—*Traité de l'Epilepsie*, Paris, 1854.

cerebral apoplexy and *hysteria*. It is often difficult to distinguish *le petit mal* from an attack of *syncope*.

Convulsions from *uræmia, opium poisoning*, or *alcoholismus* are attended by coma as the chief event, and are, each of them, accompanied by such peculiar signs, or urinary conditions, and give such a definite previous history, that they will not long be mistaken for an epileptic seizure.

In the convulsions of children caused by *dentition, falls*, and *gastric disturbances* there is not complete loss of consciousness; the fit is of shorter duration than an epileptic paroxysm, is longer in coming on, and is not followed by stupor. The discovery of a cause of the seizure is an argument *against* epilepsy.

Convulsions from *organic brain-disease, tumors, chronic softening, meningitis*, and *sclerotic processes* are distinguished by the attendant interparoxysmal symptoms, viz.: pain, mental aberration of various kinds, paresis or paralysis, and disorders of special senses. In other words, a convulsion is a part only, and not the chief part, of the symptoms; whereas a paroxysm is the prime event in epilepsy. Moreover, the previous history, the slowness of invasion, and the absence of subsequent stupor in organic brain disease will confirm the diagnosis.

Hysterical convulsions are always preceded by hysterical symptoms; volitional power is diminished, the fits come on gradually, the pupils are not dilated, there is no frothing at the mouth, loss of consciousness is not complete, tonic and clonic spasms *alternate*, stupor does not follow, and the subsequent hysterical mania has its own peculiarities. The attack is always followed by a profuse flow of pale, limpid urine.

Syncope differs from *le petit mal* in that the loss of consciousness is not sudden, is always preceded by a weak, faint, sickening sensation, recovery is slow, and the patient recollects the details of the syncope. Loss of consciousness is usually longer in syncope than in *epilepsia mitior*.

Malingerers overact their part, the conjunctivæ retain their sensibility, and the size of the pupils and the color of the face are both normal.

Prognosis.—Epilepsy rarely directly causes death. But its long duration and the suddenness of its onset make it a dreaded disease. About two to five per cent. undergo spontaneous cure.[1] The curability of the disease diminishes with its duration. Inherited epilepsy is rarely recovered from. Epilepsy beginning before the twentieth and after the fiftieth year furnishes the best prognosis. Reynolds states that the more obscure the origin the worse the outlook. Alcoholismus always renders the prognosis worse.

Treatment.—The two things to be accomplished in the treatment of epilepsy are, if possible, to remove the cause or render it inoperative; and to diminish the number, length and severity of the paroxysms.

When *auræ* exist it may be possible to abort the fit by tying a handkerchief around a limb, pinching or rubbing the surface, irritating it by means of cold or galvanism, and pricking it with needles. When muscular contraction precedes a fit, *forcibly* exciting the contracting muscles or a blow on them will sometimes prevent the convulsions. When disturbances of respiration precede a paroxysm, inhalation of ether, chloroform, or amyl nitrite may abort it. An emetic, purge, a hypodermic of morphia and atropia, ice to the nape of the neck, hot water to the extremities, valerian, belladonna, a large dose of chloral hydrate, breathing very fast, running, reading very rapidly and loudly have all been found in some instances to abort epileptic paroxysms. Reynolds advocates the administration of diffusible stimulants. When an epileptic fit is once established there is little to be done but to prevent the patient from injuring himself. The chest and neck should be freed from close-fitting garments, and if possible a piece of rubber or cloth should be inserted between the teeth.

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[1] Nothnagel.

The measures employed for the cure of epilepsy are innumerable. Trephining over cranial depressions, operations for phimosis, excisions of cicatrices, removal of neuromata, opening of abscesses, ligating the carotids, application of caustics to the throat, and tracheotomy have all been undertaken for its cure. Since epilepsy is a neurosis, different drugs must not only be employed with different individuals, but the doses must be varied in different cases. The bromides have the most extensive reputation, and at the present time are more used than any other remedy. They should be given in large doses and continuously for a long period, and only discontinued temporarily when the symptoms of bromism appear. Sixty grains of bromide of potassium a day in divided doses is the usual amount to commence with; it may be gradually increased until one hundred grains a day is administered. It is best to commence with the bromide of potash, the bromide of ammonium, iodide and bicarbonate of potash in a strong, bitter infusion—I prefer hops. With the bromides the oxide of zinc, strychnine, arsenic or atropia may be given.[1] Oxide of zinc (one and one-half grains a day at first, increasing to five grains *per diem*), especially with valerian root, or belladonna, or hyoscyamus, is regarded as next in efficacy to the bromides. Atropine and ammoniated sulphate of copper are regarded by Brown-Séquard as forming a most powerful compound in idiopathic epilepsy. The same authority ranks next in order the *cotyledon umbilicus*, silver nitrate and bromide of zinc. Whenever there is a weak pulse, the sesquicarbonate of ammonia must be substituted for the bromide of the same salt in the combination treatment.[2]

In mild epilepsy, or *le petit mal*, large doses of bromide of ammonium should be administered until a condition of bromism is reached. Cod-liver oil is especially useful in this form of epilepsy. Iron is only to be used—and then as the citrate—in the anæmic or cholorotic. Manganese is often serviceable here. External applications such as setons, issues, inunctions, croton oil, blisters, or the actual cautery to the nuchal region, have been extensively used without satisfactory results. Galvanization of the sympathetic is strongly recommended by some.[1] Epileptics should lead a life free from mental excitement or physical excess.[2]

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[1] Echeverria recommends conium, and Clonston the Indian hemp.

[2] Belladonna is recommended by Troussean is one-fifth grain of the extract daily for the first month to be gradually increased until from one to two grains are taken daily.

HYSTERIA.

Hysteria is a functional disorder of the nervous centres, affecting primarily the psychical faculties, especially the will, reason, imagination and the emotions; and secondarily both the motor and sensory tracts, in which the protean manifestations at different times indicate abolition, exaltation, and perversion of functional activity of the nervous centres.

Morbid Anatomy.—Hysteria has no pathological changes or morbid anatomy. The special functional disturbance is generally considered to be an exalted irritability of sensory centres and peripheral expansion, which results in an acquired, or is associated with a congenital, neurasthenia, most marked in the higher centres, but extending to those controlling automatic movements, and characterized by partial or complete suspension of inhibitory influence.[3] It is quite possible that in many cases the centric neurasthenia may be the primary condition and the cause of the exalted irritability.[4]

Etiology.—Hysteria affects females principally; usually making its appearance between the ages of puberty and thirty years. Over one-fourth of the cases occur between the ages of twenty and thirty; a little less than one-fourth between the ages of fifteen and twenty; and about one-sixth between the ages of ten and fifteen. It is rarely developed after the menopause, although it frequently occurs just at the climacteric. It is most liable to occur in women of a neuropathic tendency and in members of families in which epilepsy, chorea, catalepsy, and insanity have occurred. Anything which affects the emotions powerfully, such as fright, anger, jealousy, grief, and disappointment, predisposes to its development, and secret nursing of imagined wrong or anxiety is especially liable to induce it. Sexual abuse, masturbation, onanism and premature cessation of ovulation are at times exciting causes of hysteria. Its relation to uterine and ovarian disease is direct and well established,[5] but is by no means constant, as many patients with severe ovarian disturbances remain entirely exempt from hysterical phenomena. Hysteria is undoubtedly oftener met with in the single than in the married, and is intensified by the menstrual epoch. Occupation and position in life have much to do with its production. Women who lead a life of continual excitement are more prone to hysteria than any other class. Among savage nations and hard-working women it is unknown or rare. It is said that since the blacks have been freed and their education and condition bettered, hysteria, previously unknown, has appeared among them.

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[1] Nothnagel recommends methodical hydrotherapeia for three or four months, especially in cases that are not inveterate.

[2] Reynolds advocates quinine, but Brown-Séquard considers it highly injurious, stating that malarial disease in epileptics is better treated by arsenic. Recently Lepine has had success from bleeding and depletion. Kunze reports radical cures from subcutaneous injection of curare; Vallender from apomorphia. Gowers, in *Gulstonian Lectures*, says borax deserves a trial when bromide fails. Very recently picrotoxine and cocculus indicus have been tried and found to produce—especially the former—most beneficial effects.

[3] Jolly and Buzzard.

[4] Rosenthal states that the vaso-motor system is also involved, and that spasm of the cerebral arteries and consequent anæmia are often present in hysterical paroxysms.

[5] Charcot claims that hysterical fits can be produced by firm pressure over the ovaries.

It not infrequently becomes epidemic, and is apparently contagious.

Symptoms.—The symptoms of hysteria are manifest through all the nervous phenomena, and may be grouped as psychical, motor, sensory and sympathetic.

I. In many cases mental and moral disturbances appear only during the attack, and the patient has full control of the mental powers in the intervals. More frequently, and when the condition has become chronic, the patients are constantly irritable and excessively emotional. As a rule their judgment, energy, and cóncentration are enfeebled, and although their memory is not affected the will-power is greatly impaired. During their hysterical paroxysms they always want an audience; they crave attention and sympathy, and will at all times deceive and practise most dishonest measures to obtain them. Their emotions pass beyond their control, tears and laughter being apparently always at their command. Hallucinations and various kinds of fancies and delusions are common.

After a violent fit of hysteria, patients often become dangerously unmanageable, mischievous, and highly abusive or blasphemous. The coma that follows an attack is like a deep sleep, and may last for hours or days. More or less analgesia is present, but complete unconsciousness never occurs. Probably a so-called "trance" is but prolonged hysterical coma. Ecstasy and somnambulism, temporary catalepsy and trance, are all reckoned by some among chronic hysterical psychoses.[1]

II. The motor symptoms of hysteria are very varied. *Globus hystericus* is the most common; the patient imagines that a lump rises from the epigastric region into the throat and remains there causing a sensation of choking. Spasm of the respiratory muscles produces peculiar, harsh, rasping, expiratory sounds, and the inspirations are prolonged, rapid, and whooping in character, accompanied by yawning, hiccough, laughing, crying, and sneezing. There is a loud, barking, brassy cough (the hysterical cough), but no expectoration. The patients claim that all voluntary movements are impossible; they cannot rise or move from their beds—yet they gesticulate wildly and perform irrational movements in excess. The facial muscles are in constant action. Reflex action is so exaggerated that the slightest irritation produces spasms. Clonic spasms of muscles of the face and cervical region and of the muscles of the thigh are common.[2] Tonic muscular spasms in the limbs are frequent, often lasting for months; they may suddenly disappear, but these contractions resist the influence of chloroform and persist during sleep. Abdominal phantom tumors are thus produced, but long-continued Faradization will reduce them. When the tonic spasms affect, as they may, portions of the alimentary canal,

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[1] Griesinger and Briquet.

[2] Rhythmical contraction of the thigh muscles induces an apparent pulsation which may be mistaken for that of aneurism.

vomiting, griping pains, borborygmi, eructations, diarrhœa or constip
tion, and dysphagia occur. Retention of urine and great distention of th
bladder may happen. In rare instances the secretion of urine is almo
entirely suppressed.[1] In hysterical hemiplegia the face and tongue are n
involved. While walking, hysterical patients look about, whereas a *tru*
paralytic keeps his eye on his feet. The paraplegia may be complete an
the patients unable to walk, but their limbs are perfectly well nourishe
and they can regain the upright position without assistance. Hysteric
is distinguished from organic aphasia by the fact that the patient is ab
to write his wishes with the greatest readiness. Hysterical aphonia com
on abruptly, and as abruptly disappears. When an hysterical patient h
a convulsive seizure the globus hystericus precedes the fall, which alwa
takes place where there is no chance of injury. The patients often ta
continuously and incoherently during their convulsive seizure, and thro
themselves into the most grotesque attitudes. Complete loss of consciou
ness rarely if ever occurs. The pupils are not dilated, and no respirator
symptoms are present sufficient to cause asphyxia.[2]

III. Derangements of sensibility form one of the most common exhib
tions of this disease. Local or general hyperæsthesia is never entirel
absent ; it is sometimes evinced by increased acuteness of the senses
Photophobia is common. The sense of touch is so exaggerated that hy
terical women will recognize individuals by the touch ; the olfactory sens
is also exceedingly acute, and patients are disturbed by the slightest nois
and can recognize friends by their step at a long distance. Muscæ vol
tantes, tinnitus aurium, pains and neuralgias in various parts are all com
mon. The pains complained of are greatly in excess of any discoverabl
cause, and cease when the attention of the patient is diverted. The pai
often simulates left intercostal neuralgia or is situated over the vertebra
spines or stomach, in the joints, mammæ, skull or the iliac regions. Pain i
the skull, as if a nail were being driven into the head, or a kettle wer
simmering on top of it, called by the ancient physicians *clavus hystericus*
is by many regarded as pathognomonic. The whole cutaneous surfac
may be hyperæsthetic, or only parts of it. Sometimes there are ob
served hysterical angina pectoris and hysterical peritonitis. All th
senses in an hysterical patient are abnormally acute. The genital organ
are often so sensitive that sexual intercourse is impossible. On the othe
hand, anæsthesia is of frequent occurrence in hysterical persons ; it may ap
pear in any part of the body and be limited to a distinct portion of a singl
nerve. The anæsthetic parts are usually pale and their temperature sub
normal. Anæsthesia may be superficial or so deep that pins can be thrus
into the deep tissues without any expression of pain. The conjunctiv
loses its sensitiveness and may be rubbed or touched without causing con
tractures of the lids. There may be coexistent loss of sensibility in the mus
cles, bones, and joints.[3]

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[1] T. Buzzard in *Quain's Diction of Med.*

[2] Hughlings-Jackson advances the hypothesis that inhibitory control of the spinal cord over reflex actio
is temporarily suspended, the *cerebellar* influence having full play.

[3] Jolly.

In some instances the pharynx and epiglottis may be tickled or pinched, or irritating vapors inhaled without producing the customary results. Large fæcal accumulation in the rectum is presumably due to similar anæsthesia of its mucous membranes. There may be hemiopia in one or both eyes, accompanied by loss of smell, taste and hearing. Sensations as if a limb or part were greatly enlarged or attenuated, as if the feet were being buoyed up or loaded with lead, or as if pins and needles were being thrust into the waist are of common occurrence.[1]

IV. Of the circulatory changes, cardiac palpitation is perhaps the most common. Feeble heart action, with a small and hard, or a full and soft pulse, is frequently noticed during hysterical fits. The abdominal aorta, and sometimes other arteries, pulsate so strongly as to suggest aneurism. According as there is stimulation or paralysis of the vaso-motor nerves there will be a cold, pale surface or hyperæmia, redness, and consequent profuse sweating. Coldness of the extremities is one of the most common evidences of vaso-motor change. The dilatation of the vessels may become so great that hemorrhages will occur in the skin, internal organs, genitals, and stomach. It is often difficult to diagnosticate hysterical hæmatemesis from that due to *ulcer*. A single observation is rarely sufficient for a diagnosis.[2] The following hysterical phenomena are all undoubtedly due to vaso-motor disturbances, viz.: fever and chill, flashes of heat alternating with rigors, hyperæsthesia, enlargement and œdema of the joints,[3] an abundant flow of pale, clear urine deficient in salts, excessive salivation, abnormal dryness of the mouth, increased flow of gastric juice, an abundant secretion of milk, lasting for years,[4] and profuse uterine and vaginal secretions.[5]

Differential Diagnosis.—Hysteria may be mistaken for *epilepsy*, *multiple sclerosis* of the *brain* and *spinal cord*, *hypochondria*, *neuralgia*, and *uræmic coma*. It is distinguished from *epilepsy* by its slow onset, by incomplete coma, a normal pupil, sobbing and crying, and absence of subsequent stupor. The tongue is not bitten in hysteria. An epileptic seizure is short and the convulsions are not symmetrical.

Multiple sclerosis of the *brain* and *cord* is often accompanied by paroxysms like those of hysteria; but between the attacks the psychical symptoms and emotional disturbances are absent.

In *hypochondria* the patient is always morose; there are not those variations in temper that are so characteristic of hysteria. Hypochondria is rare before the thirtieth year, is more common in men than in women, and is seldom marked by convulsions. The two diseases may be conjoined.

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[1] Charcot notices that with hemi-anæsthesia there is usually ovarian hyperæsthesia of the opposite side.

[2] Astley Cooper and Parrot record cases where hemorrhages have occurred from the breast and conjunctivæ.

[3] Brodie.

[4] Briquet.

[5] Laségue has described, under the name of *hysteria periphérique*, a group of cases of considerable interest, in which, although the patients do not exhibit the general hysterical temperament, the slightest peripheral irritation causes obstinate muscular spasms. Such are certain cases of rheumatic torticollis and of blepharospasm from slight and passing irritation of the conjunctiva. Laségue says: "The transition from the typical hysteria to the other (functional) diseases of the nervous system is not abrupt, but by imperceptible gradations."

The comatose state following an hysterical seizure is distinguished fro *uræmia* by an examination of the urine, by the fact that dropsy is a sent; and the coma is preceded by sobbing, crying, and other hysteric phenomena.

Neuralgia, if of hysterical origin, ceases when the patient's attention diverted. In genuine neuralgia the pain follows the distribution of a nerv and there are certain recognizable painful spots; in so-called hysteric neuralgiæ the reverse is the case.

Organic paralysis is to be distinguished from hysterical paralysis by tl plumpness of the limb or part in the latter, and the electrical reactio which is normal.

Prognosis.—The prognosis in hysteria is always favorable, although r covery is rarely permanent, but exacerbations and remissions occur at i regular intervals. Some develop every phase of the disease at differe epochs. Its tendency is to cease after the menopause, but it may contin to old age. Briquet states that when it commences in youth it is mo persistent than when it occurs later in life. If associated with uteri diseases and displacements, the prognosis is better than when it is pure psychical.[1] When it is constitutional, hereditary, or an evidence of tl neuropathic tendency, even temporary recovery is rare.

The *hysterical contractures,* when prolonged, often cause permanent d formities.

Treatment.—Moral treatment is far more efficacious than medicine Discipline, exercise in the open air, healthy occupations, early hours, an if possible, a change of residence, all exercise a marked influence o hysterical subjects. Bromide of sodium or potassium, valerian, asafœtid belladonna, hyoscyamus, and hydrate of chloral are all at times of servi in controlling the more active manifestations of hysteria. When a cau can be reached it should, if possible, be immediately removed; and uterin diseases and displacements must receive their appropriate treatment. Iro should be given when anæmia exists. Many authorities state that ha their cases have been cured by the use of opium, and all agree that hy terical patients tolerate it in large doses. The attacks may generally b shortened by dashing cold water over the patient, and sometimes by pre sure over the ovaries. Subcutaneous injections of morphine, or inhalatio of ether or chloroform until complete insensibility is reached, are sometim advisable when the seizure is very violent.[2] In tympanitis and coli enemata of asafœtida are useful. Hysterical vomiting, often very ob stinate, is best treated by the blandest possible diet. In paralyses o hysterical origin electricity and the internal use of strychnine are som times of service. Hysterical pains are most efficiently relieved by hyp dermic injections of morphine. Aphonia may be treated by the ele tric current. Sea-baths or a course of hydrotherapy are often highl advantageous to hysterical subjects. Phosphorus and strychnine are regarded by some as specifics, and may be given in small doses. Children who are peculiar and have a tendency to hysteria, should be subjected to a firm, gentle discipline during their childhood and period of development. The manner of the physician, his conversation in the presence of the patient, the behavior of the friends and family both during and between the paroxysms, all have a great influence upon the case.

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[1] Wunderlich and Rullier describe cases of acute fatal hysteria with high temperature, great dysphagi and frequent epileptiform convulsions.

[2] Reynolds, quoting Dr. Hare, states that forcibly preventing the patient from breathing for a certa time, by holding the nose and mouth, is followed by a long breath and a relaxation of the spasm.

HYSTERO-EPILEPSY.

This is a very grave form of hysteria, attended by epileptiform convulsions and marked by the occurrence of peculiar anæsthesia, paralysis, and *muscular contraction*. It has no especial *morbid anatomy*.

Etiology.—Epilepsy may be the primary disease, and some strong psychical disease superinduce hysteria; or epilepsy may slowly develop after long-continued hysteria. The etiology is the same as that of hysteria; but puberty, the menopause, and extreme fright[1] are among its most frequent causes.

Symptoms.—An hysterical aura, usually abdominal, precedes the convulsion, which at first is identical with an epileptic seizure. Following the clonic convulsions is a short period of muscular relaxation, during which the patient appears comatose, but which is soon followed by contortions of the most violent character. The motions may intentionally indicate any or all of the vilest passions or fears, or there may be simply irrational twistings. Opisthotonos usually occurs after the attack, the patient usually suffering from hysterical excitement, laughing or crying immoderately, and has hallucinations and delusions resembling those of delirium tremens. Contractures, either paraplegic or hemiplegic, subsequently occur in one or more limbs, which may be persistent, and yield only to deep chloroform narcosis. It is to be remembered that these hysterical contractures may occur without any other symptoms of hysteria ever having existed, or may follow burns.[2] After a long duration they sometimes relax from a great moral shock. During such a fit the temperature may rise to 105° F.[3] Ovarian hyperæsthesia almost invariably precedes these attacks.[4] Anæsthesia and analgesia are common, but usually affect only one half of the body.

The special senses may all be affected, and color-blindness is not uncommon.

Differential Diagnosis.—The diagnosis of hystero-epilepsy in a well-marked case is easily made. The salient points of hysteria and epilepsy are combined, and the picture of a patient in the fit is one that will not be confounded with any other condition.

Prognosis.—The prognosis is the same as in hysteria, and far more favorable than in epilepsy.

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[1] The tragedies of the Commune in Paris during the Franco-Prussian war are said to have produced many severe attacks in hysterical females.

[2] *Progrès Médical*, Feb. and March, 1883.

[3] Charcot.

[4] Charcot states that ovarialgia is an important part of the seizure.

Treatment.—The treatment will require a combination of the remedie proposed for hysteria and epilepsy in the proportion that each enters as a element of the disease. Metallo-therapeutics have been extensively employed in the treatment of this affection. A few discs of metal are bound at intervals around an anæsthetic limb ; in ten to twenty minutes sensation returns to the skin around the discs, and then to the whole limb, but, unfortunately, in the mean time corresponding parts on the other limb gradually lose their sensibility, and the results are not permanent. The slightest electrical currents produce the same results. Contractures of years duration often can be cured or transferred in like manner. Metals, magnets, bits of wood—all have produced the same effect. Different metal act on different subjects. Gold, silver, iron, tin and copper have all been used. Long-continued blistering and Faradization have removed contractures of long standing.

CATALEPSY.

Catalepsy is a functional disease of the nervous system, closely allied t hysteria and epilepsy. It is characterized by loss of consciousness, sensation and volition, accompanied by a peculiar muscular rigidity in which the limbs remain for some time in whatever position they are placed There are no appreciable pathological changes, but the muscular rigidity is generally considered to be of centric origin.

Etiology.—Catalepsy may occur at any age, but is rarely met with excep in females about the age of puberty, and is usually associated with hysterical phenomena. It may precede melancholia and epilepsy. Traumatism, strong emotions, fright, shock, and, in many instances, religiou excitement may induce an attack. Hereditary influence is frequently marked, and it occurs in families where insanity, mania, epilepsy, etc., hav occurred.[1]

Symptoms.—Catalepsy occurs in paroxysms which are either regular o irregular. Headache, vertigo, hiccough, etc., may precede the attack Consciousness is suddenly lost, and the limbs—remaining in the position occupied at the onset—are as rigid as if petrified, soon relax a little, however, and can be moved, but will remain in whatever position they ar placed. They resist passive movement as if made of wax, hence the nam *flexibilitas cerea.* The rigidity slowly yields to the force of gravity. Sensibility and reflex movement may be totally or partially lost; rarely i there paroxysmal hyperæsthesia. The respiration and heart movement are weak ; the face is expressionless, and often has a death-like appearance The skin is cold, and the temperature is commonly lowered perhaps 2° o 3° below normal. Substances placed in the back of the mouth are slowly swallowed. In a few cases there is only partial loss of consciousness, th patient being able to appreciate strong sensorial or emotional impressions When the attack is of short duration it vanishes as quickly as it appeared and an impression upon the patient remains like that following a confuse dream. When the attack lasts for many hours or days several paroxysm go to make up the whole attack. Between the attacks there are no symptoms as a rule.[1] The attacks may occur at regular intervals; the slightest mental disturbance or excitement may bring on a paroxysm.

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[1] Eulenburg inclines to the view that malarial infection may cause it.

Differential Diagnosis.—True catalepsy cannot be mistaken; but it may be, and often has been, successfully simulated.

Prognosis.—The prognosis is favorable, except in those cases where there is a marked nervous tendency in the family. The prognosis is best where there are no symptoms between the attacks.

Treatment.—Treatment should be directed more especially to the accompanying hysterical diathesis, but we may endeavor to rouse the patient by the use of ammonia, snuff, or the Faradic current. An emetic will generally cut short an attack.[2] The wet pack and the cold douche have been used. Between the attacks iron, quinine and antispasmodics—valerian especially—are indicated.

NEURASTHENIA.

Neurasthenia spinalis is a functional weakness of the spinal cord; or, as Rosenthal calls it, a depressed form of spinal irritation.[3] It is commonly known as *nervous debility*. Rolando, Luys and others have advanced views concerning the cerebellum that may lead to this organ being regarded as the seat of the disorder. Some authors claim that it is an anæmic condition of the spinal cord, but its morbid anatomy is not as yet determined.

Etiology.—Men are far more liable to this condition than women. It often develops at puberty, but is common in adult and middle life. It is most frequent in those of a neuropathic tendency. Sexual excesses, masturbation and onanism are said to induce it. Excessive mental labor, late hours, long-continued emotional disturbances of any kind, insomnia, insufficient or improper food, and excessive use of tobacco or alcohol may excite it in those who are predisposed to neuroses. Rosenthal claims that the prolonged action of these causes in the young produces irritability of the medullary and vaso-motor centres, and thus the vascular equilibrium of the cord is lost.

Symptoms.—These patients are weak, easily fatigued and prostrated by slight muscular exertion. They are languid and despondent. There is aching in the limbs, the sleep is broken, or there is actual insomnia, and they complain that they are always tired and the subjects of nervous debility. They suffer constantly from dorsal and lumbar pains and nocturnal emissions, and the passage of a urethral sound produces excessive pain and sometimes convulsions. The sexual powers are enfeebled. During excitement or after the use of alcoholic stimulants, neurasthenic patients are able to perform a large amount of mental labor, but afterwards they are greatly prostrated. The emotions are easily excited, and they often imagine that they are the subjects of some grave organic disease. There is a tendency to melancholia and hypochondriasis. Neurasthenia is not at first accompanied by anæmia, but later the insomnia and anorexia induce it. The tongue is coated. Flatulence, dyspepsia, and dilatation of the stomach are usually present.

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[1] Eulenburg states that cataleptic children are often remarkably bright.—*Ziemssen's Encyc.*

[2] Gowers advocates the subcutaneous injection of apomorphia—one-twentieth to one-twelfth grain.

[3] Erb claims that it is not a manifestation of hypochondria, and that, although often combined with it it is to be regarded as distinctly of spinal origin.

Often the patients have a healthful appearance, which leads one to suspect that they are feigning disease.

Differential Diagnosis.—Neurasthenia may be mistaken for *incipient ataxia, incipient myelitis,* or *commencing vertebral caries.*

In *ataxia* the lancinating pains, disorders of sensation, the iron band sensation, the ocular symptoms and the increased galvanic excitability will enable one to reach a diagnosis. The paralysis which occurs in *myelitis* distinguishes it from neurasthenia.

In *spinal caries* the pain on motion and the angular curvature, in connection with the traumatic history of the case, will establish the diagnosis.

Prognosis.—The prognosis in neurasthenia is always good. It may continue for months, or relapses occur; but complete recovery may always finally be reached under proper treatment.

Treatment.—The most important indication in this condition is to secure absolute rest. Change of scene, nutritious diet, outdoor life, and especially sound sleep at night tend to produce a cure. Sea bathing is highly recommended, and a light wine or beer with meals is frequently of service. The functions of the skin should be carefully attended to. Iron, strychnine and some form of the hypophosphites are indicated.

CHOREA.

(*St. Vitus's Dance.*)

Chorea is a disease of the nervous system marked by clonic muscular contractions without order or rhythm, which tend to subside spontaneously after a few weeks' duration.

Morbid Anatomy.—Chorea has usually been regarded as a purely functional disease, but recent investigations, although leaving the pathology still somewhat obscure, seem to indicate that active hyperæmia of the brain and cord is always present, if not the exciting morbid condition, and is due to vaso-motor disturbance, which may be associated with the rheumatic diathesis or result from various mental and reflex irritations.

The occasional occurrence in chorea of capillary emboli and thrombi, with consequent minute points of softening in the gray matter of the brain, corpora striata, optic thalami and cord, together with the fact that in nearly all fatal cases endocarditis, with valvular vegetations, is present, has given rise to the supposition that these conditions represent the pathology. The commonly unilateral nature of the disease, its cessation during sleep, the absence of large emboli with consequent paralysis, and finally the fact that it is only in a small proportion of cases that the capillaries are found obstructed, are serious and fatal objections to this theory.

Whatever the morbid condition, it probably affects more especially the corpus striatum, thalamus, or a single hemisphere primarily, but in severe cases, when the muscles of deglutition and phonation are affected, extends to the medulla. An ataxic gait occasionally indicates disturbance of the cord.

Etiology.—Chorea is most frequently met with between the ages of six and sixteen, *i.e.*, from second dentition until puberty, in children whose parents have suffered from hysteria, epilepsy, and other forms of functional nervous disease; from two-thirds to three-fourths of all cases occur in girls. Feebleness of constitution, and the injurious system of forcing the education of children, as well as the conditions which tend to the premature development of the sexual instincts, predispose to chorea. Anæmia, chlorosis, onanism, and anomalies of menstruation are also predisposing causes.

Acute articular rheumatism and its cardiac complications bear such an intimate relationship to chorea that many authorities regard them as one and the same affection under different forms. The rheumatic diathesis and the resulting cardiac disease must certainly be accepted as among the most important causes of chorea. The more directly exciting causes are fright, shock, and extreme mental labor or any form of severe nervous disturbance.

Symptoms.—The onset is seldom well marked, although cases are recorded where, after a fall or shock, not more than four hours have elapsed before distinct choreiform movements occurred. It may be said that in these cases recovery is also rapid. As a rule, the child's disposition becomes irritable or moody, and although choreic subjects are very excitable, there is decided mental weakening, indicated by loss of memory and interest in things that have before interested them. The sleep is disturbed and hallucinations are common, and actual mania may be a precursor of chorea.

The first direct indications of the disease are a restlessness of movement and clumsy handling of the limbs. There will usually for a while be intervals when these children act naturally for a short time, but, if observed carefully, and especially when they are conscious of being watched, they are seen to drag a foot, fidget with their fingers, twitch the shoulders, or jerk the head in a peculiar manner. As they gradually lose control of their movements they stumble in walking, spill their food or drink, and frequently drop articles they may be holding. The choreic movements are usually unilateral, at first confined to one hand, leg, or side of the face, and in a small per cent. of cases the manifestations remain limited to one side, but more commonly extend to the other side within a few days.[1]

In the fully developed disease the symptoms vary in degree rather than kind. In the mildest cases a child simply seems awkward, breaking dishes, stumbling about the room, hurting himself with knife and fork, or, in the case of older children, never being able to correctly perform tasks where slight dexterity is required. Irregular action of the muscles of speech may occur, and words may be uttered against the will of the patient. Spontaneous pain is often complained of in the affected side.

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[1] Broadbent considers the parallelism between hemichorea and hemiplegia so perfect as to suggest at once that the two affections represent different conditions of the same nerve-centres, and that it is made more complete by the very discrepancies as they may at first sight appear.

In the worst form of the disease every feature and limb may be hideously contorted, the teeth ground together or snapped off, and bones may be broken. A patient will turn somersaults without rest, rush around in a circle, colliding with nearly everything in the room; or, if in bed, may be suddenly contorted and thrown therefrom with violence enough to produce a fracture or dislocation. Between these two extremes are cases of every degree, but in all the convulsions are made up of irregular, sudden, impulsive movements, which are entirely involuntary and aggravated by every attempt at voluntary movements. After the muscles involved have been in incessant action and violent contraction for hours, muscular exhaustion does *not* occur. In most instances, however, the muscles enjoy complete repose during sleep. When this is not the case rapid and intense anæmi occurs. As in these cases movement is almost continuous, and the patient can neither eat nor be comfortably fed, death from exhaustion may result.

Chorea of the laryngeal muscles is marked by a monotonous voice havin a deep pitch, and deglutition is often greatly interfered with. The pupil are commonly dilated and the special senses may be slightly blunted, bu the cutaneous sensibility is rarely affected. The bowels are constipated a a rule, although sometimes the fæces are involuntarily discharged.[1] Afte chorea has lasted for a long time the heart's action is disturbed; anæmia i marked, and the mental condition of the child approaches that of the idiot In girls who are old enough the menstrual functions will usually be deranged. The skin is harsh and dry, and in this class of patients hysteri often develops as a sequel.

Chorea is almost invariably accompanied by some paresis and often b complete paralysis during or preceding the development of the convulsions.

Differential Diagnosis.—*Disseminated sclerosis* of the nerve centres accompanied by tremor and jactitation that may be mistaken for chore especially as it is a disease occurring in children. But ankle-clonus, pares of both lower extremities, and the occurrence of tremor on voluntary excitation of the muscles, will decide the case. *Hysteria* and *epilepsy* ar readily distinguished from it, as is also the *tremor of old age.*

Prognosis.—Chorea is a chronic disease of varying duration, but in mos cases lasting for two or three months. Relapses frequently occur during puberty; but may occur after intervals of twenty or thirty year Complete recovery is the rule; the patient fully recovers his intelligen and muscular strength. Among children the mortality is about five pe cent., and death is usually preceded by delirium, and is due usually t asthenia or some complication. Hemiplegia, aphasia, hemianæsthesia anæmia, heart disease, rheumatism, and erysipelas or abscesses originatin in wounds which the sufferer inflicts on himself, may be reckoned amon the complications. Abortion or premature delivery may occur when mother is choreic. Very rarely does *permanent* mental derangement follow.

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[1] Bence-Jones states that the amount of urea excreted is increased.

Treatment.—Should an exciting cause be discovered (constipation or intestinal worms), it must be immediately removed. In all cases mental and bodily rest, a generous but bland diet, and pleasant, and, if possible, rural surroundings should be ordered. Children with chorea should not go to school. Many authorities advocate a generous wine in connection with iron. Sleep should be secured by the use of hydrate of chloral if necessary; Harley advocates conium. The most useful drugs are arsenic, zinc, the bromides, and hydrate of chloral. Arsenic has given me better results than any other drug; it must be given in proportionately increasing doses until its specific physiological effect is produced. Copper, the silver salts, and strychnia are much used by the French.[1] Weir Mitchell has successfully used salicylate of soda, probably in rheumatic cases. In extreme cases chloroform and other anæsthetics may be needed. The hypodermic injection of curare, friction-electricity, and galvanism are recommended. Baths, wet packs, or a thorough rubbing often act beneficially, and the ether spray along the spine seems to induce sleep and diminish violence of the choreic movements[2]

SUNSTROKE.

(*Insolation.*)

Insolation is that complex of symptoms occurring in persons exposed to extreme heat under unfavorable circumstances.

Morbid Anatomy.—The heart is usually firmly contracted, but it may be flaccid. The left heart is empty, while the right side and the venous tracts are filled with dark, often fluid blood. The blood is seldom coagulated; its corpuscles are crenated and do not tend to form *rouleaux*, and contain less oxygen than normal. The lungs are intensely congested, œdematous, and sometimes exhibit spots of hemorrhage. The spleen is swollen and soft, and, with the kidney and liver, exhibits cloudy swelling or parenchymatous degeneration. The meninges are intensely hyperæmic, and there may be evidences of incipient meningitis. The ventricles of the brain contain more or less serum, and the brain substance itself is congested or hemorrhage has occurred into it. The cord is sometimes abnormally soft. In severe cases the body is covered with ecchymoses, and sub-serous hemorrhages are common. In the neck the sympathetic ganglia, the vagi, and the connective-tissue are surrounded by, or infiltrated with blood. Rigor mortis comes on very rapidly.[3]

Insolation generally results from exposure to heat, in persons who are exhausted by either mental or physical labor.

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[1] *Gaz. Médical*, Paris, Oct., 1846. *Jour. de l'Anat. et de Phys.*, 1874; also Trousseau.

[2] In the Children's Hospital at Paris much reliance is placed on gymnastic exercise, performed with pleasant surroundings and music.

[3] Very recently Arndt states that in his autopsies there was anæmia of the brain and its membranes; and that observers must have been misled by the blood escaping from large congested vessels, and running over an anæmic brain. By him all the viscera are described as pale and œdematous. *Virchow's Archiv.*, vol. 64, pp. 15–39. See also Koster in *Berlin. Klin. Wochen.*, No. 34, 1875. Also for July 17th, 1876.

practices and habits which cause nervous strain and result in nervou haustion, may also produce spinal irritation. Chronic alcoholism an opium habit may also induce it. All severe diseases where there is a longed drain [1] on the system will be followed by spinal irritation. A ropathic tendency or hysteria often accompanies or causes spinal i tion.

Symptoms.—The one constant and special symptom of spinal irritati tenderness, which may be excited either by pressure or motion. It extend along the entire spine, or be localized over a single vertebra. tenderness, which varies greatly in degree, becomes marked on th plication of heat, cold, electricity, and other irritants. The spinous ess is the place where pressure causes greatest pain, and if seve may excite convulsions and paraplegic or cataleptic symptoms. T hyperæsthesia is very marked, but anæsthesia is rare, and myalgia ma exist with pains in internal organs. In about half the cases it is taneous; its character is very variable, but its seat is generally at point of exit of the nerves from the spinal column.

Motor disturbances are common. Weariness, heaviness, and pa paraplegia of the lower limbs follow the slightest exertion. Contra occurs in some muscles, especially those of the forearm, and twitch spasms, and choreic movements may be present. Cardiac palpitati very common; and nausea and vomiting, nervous cough, embarr phonation, deglutition, and breathing, or attacks of fainting are no common.

Patients with spinal irritation are depressed, melancholy, and irri and subject to insomnia, headache, dizziness, and disturbances o special senses. Vaso-motor changes are marked. The extremities are sometimes blue, and the face alternately pales and flushes.

When the point of tenderness is in the cervical region the pains ar ferred to the head, pharynx, and chest, and are associated with psy disturbances. When it is lower there are respiratory and cardiac symp and if in the dorsal region it is accompanied by pain in the stomach, dyspepsia, nausea, and vomiting. This last is the most frequent se the disease. Lumbar spinal irritation is less common and is indicat neuralgic pains and weakness in the lower limbs, myalgia in the ab nal and lumbar regions, spasm of the vesical and anal sphincters, ut and ovarian pains, and disorders of menstruation.

In many cases there is dysuria and vesical spasm, increased des urinate, and the discharge of a large amount of pale, limpid urine occur. Sometimes the liver, kidneys, or bowels are the seat of funct derangements. The disease may progress slowly or it may be of shor ration, and rapidly become severe or as rapidly improve.

The symptoms are always variable and inconstant, and the pains shift from one part to another. No true paralysis of limbs or of the sp ters ever occurs. Sometimes spinal irritation will suddenly pass into rasthenia.

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[1] Hammond calls spinal irritation *anæmia of the posterior columns of the cord.*

Differential Diagnosis.—Spinal irritation may be mistaken for *spinal congestion, meningitis, myelitis, tumors,* and *tetany.*

In *spinal congestion there is no tenderness.* Paralytic symptoms are frequently present, and gastric and cardiac derangements are never prominent. In spinal congestion the symptoms are aggravated by the supine position, in spinal irritation the reverse is the case. It is claimed that the subcutaneous injection of one-thirtieth grain of strychnine will aggravate the symptoms in spinal congestion, while in spinal irritation its administration affords relief. Spinal irritation is of much longer duration than congestion.

Spinal meningitis is accompanied by pyrexia, and the pain in the spine is increased by motion, so that the patient assumes, and remains in a fixed position. The pain is violent and diffused in meningitis, and muscular spasms occur in the back and neck, which are never present in spinal irritation.

The presence of the iron-band sensation about the waist, paralyses, vesical irritation, and relaxation of the sphincters, and anæsthesia, especially in the early part of the disease, are almost diagnostic of *myelitis,* and are never met with in spinal irritation.

Spinal irritation is differentiated from *spinal tumors* by the fact that in the latter the symptoms are localized, permanent, and unaccompanied by visceral derangements, which in irritation assume such a variety of forms.

The rare disease called *tetany* by Trousseau is differentiated from spinal irritation by the *muscular contractions,* which are accompanied by trembling, anæsthesia, and a feeling of intense fatigue.

Prognosis.—The prognosis is favorable, although after apparent recovery the disease is apt to return. Very frequently it resists treatment, especially if gout, rheumatism, scrofula or syphilis exist.

Treatment.—The remedies which are employed in the treatment of anæmia are always indicated in spinal irritation. Alcoholic stimulants are usually of service and in many cases must be given freely, combined with a meat diet and exposure to sunlight and fresh air. Injections of morphia or atropia combined with strychnine should be given over the site of tenderness, the dose at first being small and gradually increased to the point of relieving pain. Aconite and veratria may be applied locally in the form of an ointment. The galvanic current and the Faradic current in some cases will give immediate relief. The daily application of the ice-poultice or the actual cautery is highly recommended. Absolute rest in the country with a highly nutritious diet often does more for this class of patients than any other treatment.

VERTIGO.

Vertigo has been well defined as the consciousness of disordered equilibration. It may vary from an uncomfortable sensation to one in which the patient is unable to maintain his equilibrium. It may be momentary or of long duration.

Morbid Anatomy.—Lesions are only found in labyrinthine or apoplect form vertigo, called Menière's disease; all other varieties are purely fun tional. In aural vertigo there may be found hemorrhage, congestion, inflammation of the labyrinth; or there may be evidences of otitis medi obstruction of the Eustachian tube, or the presence of foreign bodies whic press upon the tympanic membrane.

Etiology.—Vertigo has been divided into *ocular, aural, stomachic, ne vous, epileptic,* and *gouty.*

I. Paralysis of a single muscle may cause ocular vertigo.

II. Menière's disease may be caused by disease of the semicircul canals and cochlea, tympanic catarrh, or spasm of the tensor tympan paralysis of the stapedius, or by syringing the ears, especially when tl tympanic membrane is perforated. Wax and foreign bodies in the meat externus may also induce it.[1]

III. Gastric vertigo is the most common, and is an almost invariab attendant on dyspepsia. Hepatic disorders, perhaps cholæmia, or chole teræmia may induce it.

IV. Nervous vertigo is induced by physical or nervous excesses, ar Ramskill ranks vertigo from overwork as next to gastric in frequenc Those who are ill-fed and overworked are predisposed to it. It is al caused by excessive use of tea, coffee, tobacco, and alcohol. Vertigo commonly present in megrim or sick or nervous headache.

V. Epileptic vertigo precedes an epileptic seizure, and usually does n occur without a well-marked paroxysm. Vertigo is also a common sym tom in many diseases of the nervous system, such as cerebral tumors, cer bral apoplexy, sclerosis, tabes, and cerebellar disease.

VI. Gouty vertigo is due to the blood-changes which characterize tl gouty diathesis. The vertigo of the aged is a result of disordered cerebr circulation, produced by the senile condition of the heart and vessel Chronic malarial infection frequently induces "cachæmic" vertigo.

Symptoms.—The sensation may be that of objects moving around the p tient, or of the patient moving around objects which remain stationar There may be a feeling of confusion or instability, or the movements ma be uncertain and unsteady. More or less suddenly a giddy sensation com on, objects become indistinct, the patient staggers, and perhaps falls, ur less he grasps something to steady himself. There is no loss of consciou ness. Nausea and vomiting are not infrequent, and there is ringing in tl ears, fluttering in the heart, and external sounds are greatly magnified.

The first symptoms in ocular vertigo will be running together of tl letters on the page, headache, nausea, and pains in the eyes. . In *Menière disease* slight or serious tinnitus aurium accompanies the vertigo. Su denly it becomes greatly exaggerated, and the patient feels as if he were i motion, or actually moves in a direction *opposite to the side on which tl ear is affected.* The motion may be forwards or backwards, to one side, about a vertical axis. These patients may be thrown to the ground, so ir tense are the movements. The eyes sometimes oscillate. Consciousness is rarely lost.

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[1] Knapp believes that there is always either a hemorrhage or serous or purulent exudation into semi-circular canals.—*Archiv. Ophth. and Otol.*, vol. ii., No. 1.

After the attack of vertigo passes off deafness remains. The vertigo and vomiting may continue for some time, and are increased by the upright position. One attack follows another, until a persistent vertiginous state is reached. When permanent deafness occurs, the vertigo ceases.

Gastric vertigo is accompanied by dyspeptic symptoms, nausea, pyrosis, heartburn, flatulence, diarrhœa, or constipation with pain and fulness in the hypogastrium. It is often so severe and sudden in its onset that the patient thinks he is soon to have a stroke of paralysis. The mental state is often deplorable, and true melancholia may ensue.

Nervous vertigo is apt to occur after excessive mental effort: the patient while standing experiences a dizzy, sick sensation, which is rarely severe; objects seem to whirl for a moment, and there is a slight tendency to fall. This form of vertigo not infrequently precedes softening of the brain in those who are overworked and badly nourished. Irritability, restlessness and insomnia often accompany it. And though gastric disturbances may be present, their relief is not followed by a relief of the vertigo. Sick headaches are frequently accompanied by nausea, vomiting and this form of vertigo.

In *epileptic vertigo* the vertiginous sensation either *replaces* the fit or accompanies it. After a paroxysm of gouty arthritis an attack of vertigo is not uncommon.

Differential Diagnosis.—The vertigo of Menière's disease may be distinguished from that of *epilepsy, apoplexy, gastric derangements* and the other causes of vertigo, by the co-existence of tinnitus aurium, deafness, combined with syncope, nausea and vomiting. The movements are in a uniform direction and tingling and numbness are absent.[1]

An otoscopic examination should be made in all cases of continued vertigo, and the tuning-fork and watch test should be employed.

Prognosis.—Vertigo in the adult, unaccompanied by visceral diseases, is not dangerous. In Menière's disease, when the labyrinthine affection is due to some remediable defect, the disease will subside on removal of the cause—such as cerumen, tympanic catarrh, etc. When the lesion is primarily of the labyrinth, a certain degree of deafness and tinnitus always remains, and recurrence of the attack is to be anticipated. The longer an attack has existed the better the prognosis.

Treatment.—Gastric vertigo demands the treatment already given under the head of dyspepsia. When disorders of vision are the cause of vertigo, rest for the eyes and properly adjusted glasses will remove it. In Menière's disease the patient should be placed in the recumbent posture and a full dose of bromide of potassium or ammonium given, followed by quinine in full doses. Charcot states that this plan is attended by the best results.[2]

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[1] Woakes states that aching of the upper extremities and discoloration of the hands may occur from irradiation of the irritation from the inferior cervical ganglion to the brachial plexus.

[2] Gowers and MacKenzie recommend gelsemium, salicylate of soda, counter-irritants, or even the actual cautery applied to the mastoid region.

In nervous vertigo, iron, quinine, strychnine, and the removal of
cause are sufficient. In the overworked and under-fed, wine, hypoph
phites and a nutritious diet are indicated.

The vertigo of old age is benefited by the bichloride of mercury and
tincture of iron ; a highly nutritious diet and small doses of Burgu
wine are also of service in such cases.

SEASICKNESS.

The term seasickness is applied to a peculiar form of functional distu
ance of the nervous system characterized by severe depression and persist
nausea and vomiting.

Morbid Anatomy.—The only organic changes which have been fou
which are probably secondary, are slight hyperæmia of the gastric muc
membrane, due to the prolonged efforts at vomiting and the presence
abnormal quantities of gastric juice, and cerebral anæmia with congest
of the spinal centres. The primary irritation may properly be conside
as a form of shock arising from the unusual combination of nervous i
pressions calling for unaccustomed action on the part of the nerve centr
In other words, the nerve centres are embarrassed, and the resulting nerv
irritation manifests itself through vaso-motor disturbances in precisely
same manner as is seen when persons blush under embarrassing circu
stances or pale when startled. In the present instance this disturban
though general, is most marked in gastro-intestinal and cardiac deran
ments. Paleness of the surface from vaso-motor irritation is probably as
ciated with anæmia of the brain and congestion of the spinal centres.[1]
a consequence there is irritation of those centres, manifested by severe g
tric irritability, with nausea and vomiting of centric origin.

Etiology.—As indicated in the name, seasickness is most commonly
result of the motion of a ship. It may, however, be the result of any u
usual motions to which the person is unaccustomed, and especially such
raise the body rapidly or suddenly allow it to fall, as the motion of a swi
or an elevator. Waltzing, riding backwards, turning a somersault, or
sudden jerk of a railroad train as it starts or stops or goes rapidly arou
curves in the track, may each produce a precisely similar condition. Th
are not usually followed by the full development of the disease, solely
cause they are not repeated or continued sufficiently long.

Moreover, it is not always necessary that the patient himself should
moved. Frequently, simply watching oscillating objects is sufficient
produce a mild form of sickness. Personal idiosyncrasy is a very imp
tant factor in predisposing to seasickness.[1] Some persons never suffer, ev

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[1] Dr. Clapham reports an autopsy made four hours after death upon a man accidentally killed w
vomiting, in which there was intense congestion of the spinal cord and distention of the vessels, clo
resembling the condition found in an epileptic who had died during an epileptic seizure.—*London La*
1864.

[2] In some instances naval officers of many years' experience have been led to leave their profes
from their inability to accustom themselves to the sea.

under the most trying circumstances, while others are unable to endure the slightest motion on the water or elsewhere. This peculiar susceptibility varies also in the same person, and an individual who has resisted through several sea voyages may finally succumb during a sail on some small inland lake.

Habit and experience are generally suffiicent to do away entirely with the susceptibility to the disease, but occasionally an individual suffers, it may be with increasing severity, whenever he is on rough water.

Symptoms.—Seasickness usually presents the two stages of (1) depression and exhaustion, and (2) reaction.

It begins with a sense of weight and epigastric oppression, often described as a feeling of coldness, which at first may be distinctly intermittent, occurring only during the rapid rise and fall of the vessel. It may, however, be continuous from the start, and even at first be a distinct nausea. In any event it speedily becomes so, and is accompanied by vertigo and headache. Nausea is quickly followed by vomiting, which partakes of the nature of both gastric and cerebral vomiting. Nausea is always most intense, and at the same time the vomiting is often sudden and projectile, as from a central cause. As the vomiting continues the ejected matter is composed of intensely acid gastro-biliary secretions. Constipation is the rule, and all the secretions except the saliva are decreased. The appetite is entirely lost, and there is a marked repugnance to food, and especially to all forms of fat. In many cases the simple smell or thought of food is sufficient to excite a paroxysm of vomiting. In this stage the mental depression is very characteristic, the patients almost exulting in the thought of shipwreck as affording relief from their sufferings. In the majority of cases this condition continues from three to five days, provided the voyage is of that length, during which time the nausea, vomiting and mental depression continue with varying intensity, and is then followed by reaction and a more or less rapid disappearance of the vomiting, with return to the normal condition.

In such cases, owing to the enforced abstinence, there is for a time a ravenous appetite and a feeling of special well being.

In other cases, however, the stage of depression continues until the patient is again on terra firma, lasting it may be for weeks, or it may in a few days pass into a stage of partial collapse. The patient is sleepy and apathetic, the surface is cold, and he suffers from neuralgic pains or general numbness. Finally, a partial coma may supervene and the case assume a very grave aspect.

Convalescence is generally rapid, and the patient passes from a state of the greatest depression to one of comfort and entire recovery within a few hours; but when the case has been prolonged, convalescence may be delayed and be attended by rise in temperature and other febrile conditions.

Other forms of seasickness present differences of degree rather than of kind.

Diagnosis.—Owing to the peculiar circumstances under which it is developed, seasickness can rarely be mistaken for any other condition. It may simulate an attack of *gastro-enteritis* in the early stages. In seasickness

constipation is the rule, and the intense nausea, the persistent violent von
iting, and the loathing of food are much more marked. Gastro-enteritis
most common in children, while they seldom suffer severely from seasicknes

Prognosis.—It is very rarely fatal, but occasionally a condition of collap
develops which, if not assiduously treated, may pass into coma and deat
A general irritability of the gastro-intestinal mucous membrane often r
mains for some time after a prolonged sickness.

Treatment.—The remedies proposed and tried for seasickness are inn
merable, but as most of them are only palliative or worse than useless it
unnecessary to enumerate them. Two general plans of treatment have bee
adopted, based upon the accepted pathology of vaso-motor disturbance an
spinal congestion, (1) the sedative, and (2) the stimulant. Among th
remedies of the first class counter-irritation to the spine, or ice bags, th
bromides and nitrite of amyl have proved the most useful. The applic
tion of ice to the spine was advocated by Dr. Chapman as being the be
means for controlling spinal congestion. It is of decided value, but is un
comfortable and hardly available for a large number of cases. The br
mides are often used successfully, but their use must be begun some tim
before the voyage and continued in large doses until the patient is fully a
customed to the motion of the sea. Amyl nitrite, both from the rapidit
with which it acts and the certainty of its results, seems to be the most d
sirable and efficacious remedy yet proposed. It should be given in fu
doses upon the first appearance of epigastric distress, and repeated as ne
essary.[1]

Under the class of stimulant remedies the various forms of alcohol an
the diffusible stimulants are most used, but the results, though good in som
cases, are generally far from satisfactory.

In some cases of slight disturbance, any device which controls the mov
ments of the diaphragm may be sufficient to prevent the development of von
iting. Among the most successful of these is a prolonged even inspiratio
as the vessel rises, followed by a similar expiration during descent.
must be confessed, however, that in many instances all remedies are u
availing, and only time and experience can effect a cure.

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[1] Dr. Clapham (*Lancet*, vol. ii., 1875, p. 276) reports 121 successful cases out of a total of 124 in which am
nitrite was used.

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